MANAGEMENT

OF

ACUTE TRAUMATIC INTRACRANIAL HAEMATOMA

A study of computed tomography (CT) scan, clinical features and intracranial pressure monitoring

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DEDICATED TO

Kristina Nyareba 13.6.1985
(my mother-in-law)

Petro Maswe Ng'ombe, 15.11.1985
(my father-in-law)

and

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my father

all of whom died during the preparation of this thesis

AMEN

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ABBREVIATIONS

A	Alive, in relation to outcome
BC	Basal Cisterns
CSF	Cerebrospinal Fluid
CT Scan	Computer Axial Tomography Scan
CVD	Contralateral Ventricle Dilatation
D	Dead, in relation to outcome
df	Degree of freedom, in statistical analysis
EDH	Extradural Haematomma
GCS	Glasgow Coma Scale or Glasgow Coma Score
GR	Good Recovery, in relation to outcome
ICH	Intracerebral Haematoma
ICP	Intradural Pressure
ICPM	Intracranial Pressure Monitoring
IDH	Intradural Haematoma
INS	Institute of Neurological Sciences, Glasgow
L	Lost, in relation to outcome
MD	Moderate Disability, in relation to outcome
mm	millimetre
mmHg	millimetres of Mercury
p	Probability, in relation to statistics
SD	Severe Disability, in relation to outcome
SDH	Subdural Haematoma
V	Persistent Vegetative State, in relation to outcome
V3	Third Ventricle
X^2	Chi square, in statistical analysis

SUMMARY

This thesis is based on a study of a consecutive series of acute head injured patients admitted to the Institute of Neurological Sciences in Glasgow over a three year period. Each patient had a CT scan performed which showed an acute traumatic intracranial haematoma. Literature is reviewed to highlight the earlier problems of diagnosis and controversies about different management policies now that diagnosis has been made easier by CT scan.

The objectives of this study were: to analyse the features and management of a consecutive series of head injured patients found by CT scan to have an acute traumatic intracranial haematoma and eventually required an operation; to analyse the clinical, CT scan and intracranial pressure features in these patients and to determine the influence of each of these on the treatment of a patient; to evaluate the efficacy of intracranial pressure monitoring in the management of clinically 'silent' acute traumatic intradural haematoma; and to determine the results of different initial decisions about operative and non-operative management.

The initial list of patients admitted to the Institute of Neurological Sciences in Glasgow during the period of January 1978 to 31st December 1980 was obtained from the Glasgow Head Injury Data Bank. Information relating to patients who had been operated upon could be retrieved from that data bank. A study on the patients not operated was conducted in order to obtain information comparable to those who had been operated upon. The clinical features

studied were the patients' age, level of consciousness as determined by the Glasgow Coma Score, pupillary response to light and the outcome at six months follow up as determined by the Glasgow Outcome Scale. The CT scan (radiological) features studied were the type of intracranial haematoma, the appearance of basal cisterns and third ventricle, presence of a dilated ventricle contralateral to the haematoma and the extent of midline shift. In selected patients intracranial pressure was monitored through a ventricular catheter inserted via a frontal burr hole. The intracranial pressure features studied were the duration of monitoring and the level of intracranial pressure in millimetres of mercury.

Chapter 4 provides an overview of the results of the patient population. The total population consisted of 479 patients, CT scan being available in 411 of them. Of those with CT scan details, 86% had an intradural haematoma and 16% had only extradural haematoma.

52% of the patients with an intradural haematoma had immediate surgery and another 21% were operated upon after a period of clinical observations and or ICP monitoring. Only 3% of patients were never operated upon because of poor prognosis.

The abnormal features noted on CT scan were: obliterated basal cisterns, obliterated third ventricle, presence of a dilated contralateral ventricle, and midline shift exceeding 10 millimetres. Of the patients found to have obliterated basal cisterns, 76% were in coma. In the patients in each of the coma score groups 3-5, 6-7, 8-10

and 11-15, there were 85%, 63%, 41% and 22% of patients with obliterated basal cisterns respectively. Analysis of the three other abnormal CT scan features showed a similar result. The presence of abnormal CT scan features was associated with pupillary dysfunction. When the basal cisterns were not obliterated, 87% of patients had both pupils reacting to light, 5% had only one and 6% had neither pupil reacting to light. However when the basal cisterns were found to be obliterated, only 45% of patients had both pupils reacting to light, 12% had one and 40% had neither pupil reacting to light. Of the patients found to have both pupils reacting to light only a third had obliterated basal cisterns, but this CT appearance was found in 90% of those with neither pupil reacting to light. A similar trend was observed in relation to third ventricle appearance, presence of contralateral dilated ventricle, and degree of midline shift. When the inter-relation between the different CT scan features was examined it was found that it was common to find the abnormal features occurring together. Thus, obliteration of the third ventricle and or basal cisterns, or contralateral ventricular dilatation were found in a third of patients with a 5mm shift, three quarters of those with a 10mm shift and over 90% of those with a 15mm shift.

Patients with a subdural haematoma more often underwent operation than those with an intracerebral haematoma. The majority of those who needed immediate operation had shift or other abnormal CT scan features. Of the patients not operated upon immediately, 63 (18%) had intracranial pressure monitoring as the initial line of management.

All patients with ICP >30mmHg were operated upon, two thirds of those with ICP 20-30mmHg and 6% of those with ICP <20mmHg were also operated upon. The decision to operate had been reached within 72 hours of ICP monitoring in 93% of cases. When the initial CT scan features were related to the ICP level, 50% of those with ICP <30mmHg had abnormal CT scan features. Neurological deterioration was the basis for a decision to operate in those with ICP <30mmHg but also occurred in patients with ICP >30mmHg while awaiting surgery.

The overall results confirm the prognostic significance of clinical features. Patients who had abnormal CT scan features at the time of their initial assessment had a more unfavourable outcome than those with normal CT scan features. It was also found that in patients with coma score 3-10, the presence of abnormal CT scan features had the same predictive value as the coma score but that in those in the coma score group 11-15 the presence of abnormal CT scan features had an adverse effect worse than could have been determined basing on the coma score alone.

The results of the patients managed by ICP monitoring in this series did not differ signficantly from the previous Galbraith and Teasdale series. From the findings it is suggested that the present level of ICP at which to base the decision to operate is too high and a level of >20mmHg is recommended. Furthermore when taking the initial decision about management it is suggested that the status of the CT scan features should be considered. Patients with abnormal CT scan features should <u>all</u> be operated upon immediately, regardless of how well they may appear to be.

This is because the presence of abnormal CT scan features precedes neurological deterioration. In patients who are found to have low ICP, <20mmHg, monitoring should be continued for 72 hours and thereafter a repeat CT scan should be done.

The findings are finally considered in relation to patients with spontaneous intracerebral haematoma. The problems of the future are mentioned.

CHAPTER 1

I N T R O D U C T I O N

Head injury is an established significant health, social and economic burden that is universal; it abounds all known geographical boundaries. In most parts of the world it affects the young productive adult more than any other group. Many patients die but the consequences of brain damage, primary or secondary, are of great concern to the surviving patients, their families, the therapeutic and rehabilitating teams involved and to the community at large. Brain damage sustained at impact (primary) in missile, sudden acceleration or deceleration, injuries may be so overwhelming as to be irremediable by any known present means. Secondary brain damage, occurring at some stage after initial impact, does result in preventable mortality and morbidity.

The commonest cause of secondary brain damage remediable by a neurosurgeon is a traumatic intracranial haematoma. It is not surprising therefore that the need to detect and evacuate a haematoma has dominated head injury management policies for decades. This is because it has long been recognised that earlier diagnosis and treatment is a recipe for better results.

Computer tomography (CT) scanning was rapidly recognised as a means of offering improvement in detection of haematoma, hence improved outcome. Nevertheless its introduction also raised several questions, in particular the significance of its revelations in many patients. Subsequently a series of studies aimed at providing answers have been conducted at the Institute of Neurological Sciences in Glasgow.

The Institute of Neurological Sciences (INS) at the Southern General Hospital in Glasgow, Scotland, is a regional Neurosurgical centre catering for the West of Scotland with a population of nearly three million people. Patients are first seen in primary surgical wards in the various hospitals in the region and are then secondarily referred for neurosurgical re-appraisal and further management. Radiological diagnosis is achieved by two EMI 1010 computerised axial tomographic (CT) scanners available since 1975. Until 1978 it was customary to accept transfer of head injured patients who were either in coma, were deteriorating or had not improved after a period of observation in the primary surgical ward or who had developed focal neurological signs.

The ease with which such a non-invasive investigative procedure could be done enabled more patients to be transferred for investigations, even before they had a significant deterioration in their level of responsiveness (or consciousness). It became apparent that intracranial haematomata were more frequent than had previously been realised when investigations were invasive. Since 1978 about 500 head injured patients are referred to this unit each year of whom about 100-125 undergo surgery to evacuate an acute traumatic intracranial haematoma.

There was no difficulty in deciding to evacuate a haematoma in those patients who had deteriorated or developed focal neurological signs. The predicament was what to do with patients with an intradural haematoma but who were either improving or had actually not deteriorated.

It was customary to evacuate all extradural haematomas regardless of how deceptively well the patients were neurologically. Some patients were operated upon because their intradural haematoma was considered to be too 'large' to be observed. Others who were considered to have a potentially surgically significant intradural haematoma but its size on CT scan was 'small' were clinically observed and operated upon when they deteriorated or their neurological status did not improve. The judgement that a haematoma was small or large was a subjective interpretation of the surgeon in attendance.

It is generally accepted that an intracranial haematoma causes neurological deterioration in head injured patients as a result of raised intracranial pressure. The natural logic of evacuating a haematoma is therefore to relieve the intracranial pressure. The expected corollary to this would be that if a patient had an intracranial haematoma but had no evidence of raised intracranial pressure then there MIGHT NOT be a need to evacuate the haematoma. Galbraith and Teasdale, at this Institute, studied a small number of selected patients and suggested that measurement of intracranial pressure may be a useful guide to the need for operation in patients with clinically 'silent' intradural haematoma. Following that study it was part of the practice, in the Glasgow Neurosurgical Unit, when a patient had a 'silent' traumatic intradural haematoma to measure their ICP and to determine the need for operation based on their level of intracranial pressure (ICP). At the same time some patients continued to be observed clinically because it was felt by the neurosurgeon responsible that the

CT scan and clinical signs provided sufficient information about the intracranial events.

As my clinical responsibilities increased to Registrar and then Senior Registrar whilst working at the Glasgow Unit I became intrigued by the need to clarify the relationship between various CT scan features held to relate to neurological function, the level of responsiveness (or consciousness) and outcome of patients. Furthermore, I saw the need to determine what part intracranial pressure monitoring was indeed playing in the management of 'silent' acute traumatic intradural haematoma.

The objectives of this study therefore were:

1. To analyse the features and management of a consecutive series of head injured patients found by CT scan to have an acute traumatic intracranial haematoma during the three year period 1st January 1978 to 31st December 1980.

2. To determine how often patients with intradural haematoma eventually required an operation.

3. To analyse the clinical, CT scan and intracranial pressure features in these patients and to determine the influence on their treatment.

4. To evaluate the efficacy of intracranial pressure monitoring (ICPM) in the management of clinically 'concealed' or 'silent' traumatic intradural haematoma.

5. To determine the results of different initial decisions about operative and non-operative management.

In order to achieve these objectives, I shall first examine CT scan features in order to determine how they inter-relate with clinical features (level of consciousness and pupillary response to light) and management. Then I shall analyse features relating to intracranial pressure monitoring relating them to how the patients were (level of consciousness) and their CT scan features. Finally how clinical, CT scan and intracranial pressure features relate to outcome will be determined before drawing up my conclusions.

CHAPTER 2

REVIEW OF RELATED LITERATURE

2.1 BACKGROUND

Literature will be reviewed in order to highlight what is already known in relation to the main issues in this study. Having looked at how intracranial haematomata pose a problem today the next step will be to review the assessment of patients suspected of harbouring a traumatic intracranial haematoma by clinical and radiological methods before evaluating how intracranial pressure monitoring may assist in the initial assessment and management. Naturally to follow will be to examine what is known about the interrelationship between CT scan, clinical and intracranial pressure features before looking at the historical and current aspects of management of patients with traumatiic intracranial haematoma with particular mention of how inntracranial pressure monitoring may affect future management. Finally the assessment of outcome and of prognosis of patients after head injury will be reviewed with particular emphasis on clinical, CT scan and intracranial pressure features.

2.2. INTRACRANIAL HAEMATOMA IN HEAD INJURED PATIENTS

The incidence of head injury in a community can be estimated from hospital based statistics. There are between 200 and 300 patients per 100,000 population admitted to hospital each year in the United States and the United Kingdom. But the annual rate of those attending accident and emergency departments in Scotland alone is 1775 per 100,000 population, twice as many men as women (Jennett, Murray et al 1979). Deaths from head injury in

the United Kingdom account for about 1% of all deaths but much higher in the young adult male and about twice as many in the United States. Reports from developing countries are scarce.

Most available reports on the incidence of traumatic intracranial haematomas vary greatly because of the different admission policies and partly because of the methods used for their detection. Hospital based statistics indicate that 1 to 6% of patients seen in general hospitals have a clot (Lewin 1949, Kalyanaran et al 1970, Galbraith et al 1977). But autopsy studies have shown that about half of the cases of haematoma were not diagnosed before death (James and Turner, 1951, Maloney and Whatmore 1969). Studies based on CT scan have also shown that about half of patients in coma do harbour an intracranial haematoma (French and Dublin 1977, Zimmerman et al 1978b).

It is now clear that CT scanning of patients who have minimal or no symptoms after a head injury sometimes reveals what can be described as a clinically 'silent' haematoma. The true incidence, therefore, of this complication after a head injury is far higher than is generally reported.

There have been several variations in the temporal classification of haematomas in different series (Voris 1941, Echlin et al 1956, Nora and Rosenbluth, 1957, Fager 1958, McKissock et al 1960, Lewin 1966, Hooper 1969, Tallala and Morin 1971 and Rumamurthi 1976). Using the time interval between injury and diagnosis two types are now recognised; Acute up to 14 days; Chronic, more than two weeks (Jennett and Teasdale, 1981).

2.3 ASSESSMENT OF PATIENTS SUSPECTED OF TRAUMATIC HAEMATOMA

a) CLINICAL

It has long been recognised that the most important and consistent manifestation of damage to the brain is impaired consciousness and coma. It was rightly feared that intracranial haematoma complicating a head injury might be a curable cause for the deterioration in consciousness. Alteration in consciousness had therefore been the clinical hallmark upon which clinicians relied in order to determine which patients required further active measures (Hooper 1949, James and Turner 1951, Rowbotham 1964, Carlson et al 1968, Jamieson and Yelland 1968, 1972).

In order to detect neurological deterioration it became important to establish baseline parameters of clinical assessment with which to determine whether deterioration had in fact occurred. Hence, a number of scales were devised in order to make assessment of consciousness easier (Bouzarth 1968, Rowbotham 1964, Teasdale and Jennett 1974, Subezynski 1975, Ransohoff and Fleischer 1975, Tindall et al 1975, Brinkman et al 1976 and Yen et al 1978). Originally inherent in such a system was the acceptance of allowing patients to suffer secondary brain damage before remediable measures were taken, denying patients better prospects for improved results. Before CT scan was introduced, that might have been accepted considering the significant complications associated with the methods used in the detection of traumatic haematoma. But even today, when such a detection can be done non-invasively, it is still important to record accurately the level of consciousness, brainstem reflexes and outcome in

any analysis of head injury so that the differences in result might be explained by how the patients were in the first instance.

b) CT SCAN FEATURES (RADIOLOGICAL)

Many approaches to the diagnosis of traumatic intracranial haematomas have been employed. The accidental visualisation of the ventricular system by Duckett in 1913 in a skull film in a patient with a traumatic aerocoele was later adopted by Dandy in 1918 making it possible to diagnose reliably if a patient had a space occupying lesion: ventriculography was born and is still in use today in some centres. In 1927, Moniz described arterial encephalography (angiography) by direct carotid injection of contrast media. These investigative procedures were invasive and had a risk of dangerous complications and also needed special skills to perform and to interpret. There was great caution with which these techniques were used for the diagnosis of intracranial haematoma. Only patients who had localising signs or in particular, were deteriorating neurologically were so investigated (Loman and Myerson 1936, Bowder 1943, Brodin et al 1952, Hancock 1961, Leslie et al 1962, Rowbotham 1964, Jamieson and Yelland 1968, Lake and Pitts 1971, Fell et al 1975 and Rao 1977). An alternative diagnostic technique, but no less invasive, had been the use of exploratory twist drills or burr holes (Rand et al 1968) but this technique often failed to detect significant clots especially those situated in unusual sites. For all this time the most important question had been, 'does a patient have an intracranial haematoma or not?'

The advent of computerised axial tomographyic scanning provided a reliable method to answer this question.

Computerised axial tomography (CAT or CT) scanning was developed in England in 1969 by Godfrey Hounsfield of EMI Laboratories Ltd. The technique is carried out by a narrow beam of x-rays transversing the head in a horizontal plane through an arc of 180 degrees. The emerging x-rays are receieved by a series of sensitive detectors and recorded as numbers by a computer. These numbers, now called Hounsfield Numbers, represent the different tissue densities in relative terms, the lowest (-1000) being for air, 0 being for CSF and the highest (+ 1000) being for bone. Images appear on a screen and can be copied on to an ordinary x-ray film for later viewing. The computer images are stored in disks for temporary use and can be transferred to tapes for long term storage and potential retrieval. At its inception, single images could be produced after nine hours of computer processing, a time that lasts only a few minutes today. Whole head scanning requires a series of horizontal slices of 10mm thickness using the orbitomeatal line as the plane of orientation. The images are produced on a 160 x 160 matrix, each film containing nine pictures (Fig 2 to 5). There are at present many variations in this technique with higher definitions and resolutions and even shorter scanning time.

In 1973, Ambrose reported the clinical applications of computed tomography scanning. The validity of its clinical uses became accepted following the publication of the experience with the first 650 patients (Paxton and Ambrose

1974). The value of CT scan in the diagnosis of complications in head injured patients has been confirmed by many studies (Merino de Villasante and Taveras 1976, French and Dublin 1977, Dublin et al 1977, Koo and la Rogue 1977, Zimmerman et al 1978a, Bartlett and Neil-Dwyer 1978, Robertson et al 1979, and Snoek et al 1979, Tans 1979).

The types of lesions seen on CT scans have correlated well with autopsy findings (Jacobs et al 1976). Intracranial haematoma appear as hyperdense (relative to brain) areas. Extradural haematomas have a biconvex configuration while subdural haematoma have inner concave and outer convex appearances. Intracerebral haematoma appear as hyperdense intraparenchymatous lesions (Ambrose et al 1976, Svendson 1976 and Amendola 1977).

Early experience indicated that neurosurgeons could reliably interpret the types of lesions shown on CT scans (Galbraith et al 1977. But the pitfalls and limitations of information provided, particularly by the early generation CT scans, have been cautioned (Davis et al 1976, French 1978 and Smith et al 1981). Most reports on the frequency of the different types of haematoma show that extradural haematoma are seen in 5-15% of series while subdural haematoma in 20 - 30% (Gennarelli et al 1982, Zimmerman et al 1978b and Sweet et al 1978). Traumatic intracerebral haematoma commonly occur in the frontal and temporal lobes in contrast to spontaneous intracerebral haematoma which occur in the basal ganglia or those in relation to diffuse axonal injury which occur predominantly in the corpus callosum and basal ganglia (Adams et al 1982).

The description of intraparenchymatous lesions in the frontal and temporal lobes especially, vary in different reports; focal lesions (Gennarelli et al 1982); focal intracerebral abnormality (Zimmerman et al 1978a); areas of increased density representing intracerebral haematoma/contusions (Sweet et al 1978); or simply intracerebral haematoma (Dolinskas et al 1977). The original criteria (Paxton and Ambrose, 1974) defined an intracerebral haematoma by its density. Using that criteria Dolinskas (1977) was able to describe various sizes of intracerebral haematoma from all causes and recorded an incidence of 13% in the trauma series. Other reports describing similar lesions in different terms as above noted an incidence of 19% (Gennarelli et al 1982), 21% (Zimmerman et al 1978b) and 32% (Sweet et al 1978). This contrasts with earlier pre CT scan reports in which intracerebral haematoma were said to occur in 1-2% of series (Rowbotham 1964, Romanul 1970, Grubb and Coxe 1974).

c) INTRACRANIAL PRESSURE MONITORING

In 1951, a technique of measurement of ventricular intracranial pressure was introduced by Guillame and Janny in France. It was Lundberg who, in 1960, pioneered the use of continuous long-term intraventricular pressure monitoring in patients with neurological disorders including head injury (Lundberg 1960, Lundberg et al 1965). This has since been widely adopted and is now a standard neurosurgical procedure. The technique involves the making of a twist drill or burr hole, usually in the frontal area, and inserting a catheter into the lateral ventricle.

This is then connected to a transducer, amplifier, and a continuous paper chart recorder or computer terminal, the readings having been calibrated to read in absolute units of pressure, usually in millimetres of mercury (mmHg).

There have been several ways by which intracranial pressure monitoring may be used to provide information relating to intracranial dynamics. The simplest and most direct way is the magnitude of intracranial pressure, how high or how low it is. Ventricular Volume Pressure Response (VPR), a measure of intracranial elastance, is the immediate change in ICP resulting from a change in CSF volume. This volume-pressure test has been claimed to show a better correlation with the clinical state of the patient than the magnitude of the ICP alone (Miller et al 1973, Miller and Pickard, 1974). But because it involves additions of solutions to the ventricules it has the inherent added risk of infection, an unwanted iatrogenic complication. To circumvent the hazards of CSF manipulation it has been possible to measure the magnitude of ICP pulse amplitude (CSF pulse pressure). But it is far from clear of how such pulse amplitude studies might be relied upon to provide information relating to intracranial pressure - volume relationship or indeed their clinical usefullness (Nornes et al 1977, Foltz and Lederhaus 1977). By measuring the magnitude of ICP alone, Miller and his colleagues (1977) showed that intracranial haematoma were the commonest cause of raised intracranial pressure in acute head injury patients. The same group of workers were also able to identify which patients had developed postoperative or 'delayed' intracranial haematoma by repeating investigations in patients who had a rise in ICP. Furthermore, they were able to show that

Furthermore they were able to show that neurological deterioration invariably was associated with ICP levels in excess of 40mmHg thereby providing a warning so that necessary measures to lower ICP could be instituted. In patients in whom neurological examination is rendered not posible because the patients have been paralysed to enable mechanical ventilation, measurements of ICP provides useful guide to intracranial events. In addition the magnitude of ICP provides a practical parameter with which to assess the efficacy of different treatment modalities intended to lower intracranial pressure especially in patients with a diffuse head injury without recourse to CSF manipulation (Marshall et al 1979, Lobati et al 1979, Narayan et al 1982). There is therefore a role for the measurements of the magnitude of ICP in acute head injury patients with or without a haematoma in order to assess their progress.

The concept that measurements of intracranial pressure may be of value in assisting the management of patients with an intradural haematoma, in whom there is initial doubt as to the need for surgery, was first introduced by Galbraith and Teasdale (1981). They studied a small group of selected acute head injury patients with CT scan proven intradural haematoma and who were neurologically stable and measured the magnitude of their ICP and related this with the need to evacuate the haematoma. They showed that in the patients who were eventually operated upon because they had clinically deteriorated their mean ICP at the time of that deterioration was greater than 30mmHg. They also reported that, in that series, poor outcome in patients who needed operation was twice as often as in those who did not

2.4 INTER RELATIONSHIP BETWEEN CT SCAN, CLINICAL AND ICP FEATURES

Early reporters on the value of CT scan in the management of head injured patients were particularly overwhelmed by its ability to reveal promptly, accurately and non-invasively the type of trauma related lesions that were often only seen at autopsy (Ambrose et al 1976, Svendsen 1976, Allen 1977, Bergstrom et al 1977, Forbes et al 1978, Doninskas et al 1978. Most of the patients with lateralising signs were found to have lesions on the expected side (Espersen and Peterson, 1981). Patients who were in coma due to a severe diffuse head injury were often found to have haemorrhagic lesions affecting the corpus callosum and white matter and that the sites of lesions correlated well with the observed neurological deficits such as hemiparesis (Zimmerman et al 1978a). Apart from the type of lesions demonstrated, Becker and his colleagues (1977) noted that abnormal motor responses were commoner in patients with a midline shift greater than 10 millimetres (mm). While no relationship could be found between midline shift and the level of consciousness in patients with diffuse head injury, patients who had extracerebral haemorrhage and a significantly depressed level of consciousness often had midline shifts of 12mm or more (Clifton et al 1983). In a recent study of head injured patients without an intracranial haematoma it was reported that the absence of the third ventricle was commonly seen in patients in deep coma while the absence of basal cisterns was seen in 7 of 8 patients who had pupillary reaction to light (Teasdale et al 1984).

Thus although there has been recent interest in evaluating CT scan findings, a number of the studies have been in patients without a surgically significant intracranial haematoma. There is then the need to assess these CT scan features and to find out how they inter-relate with clinical features, especially the level of consciousness and pupillary reaction to light, thus providing a useful index to management decisions.

There have also been studies that have attempted to correlate CT scan findings with the level of intracranial pressure. Thus the presence of an intracranial haematoma has been shown to be the commonest cause of raised ICP in acute head injured patients (Miller et al 1977, Kishore et al 1981), while the presence of an intraventricular clot and ventricle compression by a haematoma was claimed to be associated with raised ICP (Tabaddor et al 1982). An earlier report could not confirm that ventricular compression alone was associated with raised ICP (Auer et al 1980). Miller et al 1977, considered that midline shift of less than 5mm had no association with ICP and so did Tabaddor et al (1982) who found out that midline shift as did the size of a haematoma poorly correlate with ICP. It has also been reported that a widened contralateral temporal horn in patients with a traumatic haematoma was associated with midline shifts of at least 12mm but there was no mention of ICP in that series (Stroving 1977a). It was later suggested that the presence of a dilated contralateral temporal horn may indeed be related to raised ICP (Haar et al 1980).

Recent observations in severe diffuse head injury patients, who did not have a haematoma, showed that the absence of basal cisterns and third ventricle correlated with ICP greater than 20mmHg (Murphy et al 1983, Teasdale et al 1984). The evidence that CT scan finding can be related to ICP is thus fragmented, at times controversial, and based largely in patients without an intracranial haematoma. A reasonable suggestion is to consider these CT scan findings in the same series of patients and to find out how they inter-relate with ICP levels in patients with traumatic intracranial haematoma. If a reasonable estimate of ICP can reliably be made by examining CT scan features that might obviate the need to measure it.

2.5. MANAGEMENT OF PATIENTS WITH TRAUMATIC INTRACRANIAL HAEMATOMA

Operative and nonoperative management of patients with head injury has been practiced for over 3,000 years. The first known written records of surgical practice are contained in Egypt in the famous Edwin Smith Papyrus which includes 27 cases of head trauma. It is known that Hippocrates and his contemporaries around 1081 BC advocated for prophylactic trepannation for letting out 'bad spirits' after head injury (Adams 1949). Percivall Pott said that 'the reasons for trepanning in patients with head injury were : first, the immediate relief of present symptoms arising from pressure of extravasated fluid; second, the discharge of matter formed between the skull and dura; third, the prevention of such mischief by the last mentioned membrane'. (Walker 1951). The problem then was

'to trephine or not to trephine'. Two hundred years ago Sir John Hunter remarked that when in doubt it is better to trepan since the operation can do no harm (John Hunter, 1986). Surgical methods have thus ben available for a long time, the problem had always been that of diagnosis, in that context who needed operation.

Angiography, pneumoencephalography and ventriculography enabled the detection of intracranial mass lesions. The new pressure was to operate on head injured patients confirmed to have a mass lesion in the hope of improving the then dreadful results (Hooper 1949), McKissock et al 1966). There is little said about unoperated cases except those diagnosed at autopsy. In one pre CT scan report on the results of surgically treated subdural haematomas it was only casually stated as a sideline that 'cases where radiologically demonstrated haematoma had resolved without surgery had been excluded from analysis' (Jamieson and Yelland 1972).

The advent of CT scan has unveiled what must have been in the minds of many clinicians: That traumatic intracranial haematoma were indeed far commoner than had been known. The problem for the surgeons was to decide which lesions should be considered surgically significant. The knowledge that delay in treating patients with intracranial haematoma is a recipe for poor results added importance to the question: **'operate or observe'?** An extreme view was to advocate radical surgery in all patients in coma in order to improve results (Becker et al 1977). On the other hand a study on a series of patients with traumatic intracerebral haematoma showed convincingly by serial CT scans that if left unoperated such haematoma do

resolve thus obviating surgery (Dolinskas et al 1977). But the experience of others showed contrasting results. Wiegel et al 1978, reported on a series of 31 patients with CT scan proven traumatic intracerebral haematoma in which all four patients operated upon died while in theremaining 27 patients treated conservatively (Non-operatively) only 4 (17%) died. Their conclusion was that 'operation was indicated only if clinical deterioration had occurred as a result of increasing intracranial pressure', although none of their patients had intracranial pressure measured. But clinical deterioration before surgery is what should be avoided. This reflects the initial difficulty in deciding rationally which patients were likely to deteriorate and therefore be operated upon early thereby improving chances for a favourable recovery.

The clinicians predicament is illustrated further by the so-called <u>delayed traumatic intracerebral haematoma</u>. Historically this was first described as traumatic apoplexy by Bollinger in 1891 and later also described as delayed deterioration (Lafforgue 1904, Courville and Bloomquist 1940, Evans and Scheinker, 1946 Nelson et al 1982), while McLaurin and Helimer in 1965 described it as the 'syndrome of temporal lobe contusion.' There have been numerous reports of its occurrence in both the pre CT scan era (Naffziger and Jones 1928, Doughtly 1938, Symonds 1940, De Jong 1942, Morin and Pitts 1970, Barathan and Dennyson 1972) and in the CT scan era (Brown et al 1978, Diaz et al 1979, Gudeman et al 1979). In each of the series in which patients had that

diagnosis eventually confirmed by CT scan, the initial CT scan was 'abnormal' and the type of lesions so demonstrated were of initial uncertain significance. It has been repeatedly shown in these series that patients with such lesions often deteriorate precipitously and that follow up CT scan provided a clearer impression of the initial CT scan findings. Mortality in such event has been about 50% (Gudeman et al 1979). These reports re-emphasise the problem of how to respond to an initial CT scan finding of an intradural lesions in a patient who is clinically stable, and in whom such a lesions might not have been suspected on clinical grounds alone.

2.6. ASSESSMENT OF OUTCOME AND PROGNOSIS AFTER HEAD INJURY

It is important to have a standard system of assessment of outcome because without it the comparison of results of the different management modalities in the different series would be impossible. Of the various methods used some had descriptions with pessimism: invalid, serious, appallic etc; or optimism; re-integrated, excellent, worthwhile etc (Overgaard et al 1973, Najenson et al 1974, Pazzaglia et al 1975, Roberts 1976). The Glasgow Outcome Scale provides a limited number of exclusive categories that still entail a detailed description of the various disabilities (Jennett and Bond 1975).

Equally significant is the timing of such assessment. It is evident that patients assessed early during convalescence may continue to show improvement in mental

and physical function. Assessment of outcome at six months appears to provide a reasonable epoch during the period of convalescence (Jennett et al 1977).

A number of clinical and investigative factors have been identified in relation to the prognosis of patients after severe head injury, ie in coma for at least six hours as defined by Jennett et al 1977. The important clinical factors include age (Caruselli and Luongo 1974, Jennett et al 1977, Teasdale et al 1979c, Frowen 1979, Narayan et al 1981) and the depth of coma as determined by the Glasgow Coma Scale (Jennett et al 1977, Teasdale et al 1979a, 1979b, Marshall et al 1979, Miller et al 1981, Braakman et al 1980, Bowers et al 1981). Investigative factors that have been correlated with outcome include level of intracranial pressure, electrophysiological tests and CT scan features. Thus raised intracranial pressure greater than 40mmHg in diffuse head injured patients has been associated with poor outcome (Miller et al 1977, 1981, Lobato et al 1979, Marshall et al 1979, Narayan et al 1982). Brain stem reflexes especially caloric (oculovestibular), oculocephalic and pupillary response (Pagni 1973, Pazzaglia et al 1975, Price and Knill-Jones 1979, Miller et al 1981, Levati et al 1982) and multimodality evoked potentials; somatosensory, auditory and visual (Greenberg et al 1977, 1981, Narayan et al 1981, Lindsay et al 1981) have all shown good correlation with the observed outcome. The latter require specialised and expensive equipment and especially trained personnel not too readily available in many neurosurgical centres. It was earlier noted that the type of lesion correlates with outcome (Munro and Mattby 1941, McKissock et al 1960a, McLaurin and Tutor 1961, Khatib et al 1967, Kvarnes and Trumpy 1968, Gallagher and Browder 1968).

The value of CT scan in relation to prognosis was at first not appreciated. As a departure from the earlier emphasis on its superior diagnostic capabilities in delineating the types of intracranial lesions there have been an increasing interest in the use of CT scan to provide indices of severity of primary and secondary damage and as a guide to prognosis. CT scan features which have been identified with poor prognosis are: **brainstem haemorrhages** (Tsai et al 1978, Cooper et al 1979), **corpus callosum and white matter shearing injuries** (Zimmerman et al 1978a), **bilateral haemorrhagic lesions** (Sweet et al 1978, Kishore et al 1981); **intraventricular haemorrhage** (Roberson et al 1979, Cordobes et al 1983, **delayed intracerebral haematoma** (Gudemman et al 1979 and a type of **intracranial lesions** (Gennarelli et al 1982, Lobato et al 1983). What may seem obvious is that a normal CT scan correlates with good outcome (Holliday III et al 1982).

There has been little mention of the value of other CT scan features in relation to prognosis. Only recently has the significance of basal cistern appearance been mentioned but mostly in severe diffuse head injuries (von Dongen et al 1983, Toutant et al 1984, Murphy et al 1983, Teasdale et al 1984). The inter-relationship between various CT scan features especially midline shift, appearance of the third ventricle and the basal cisterns and the dilated contralateral ventricle need to be examined in patients presenting with a traumatic mass lesion, a haematoma. The CT scan findings outlined so far are more in relation to the severity of primary brain damage with only the delayed intracerebral haematoma being regarded as a secondary cause of brain damage.

CHAPTER 3

PATIENTS,

MATERIALS

AND

METHODS

3.1 THE STUDY

This thesis is based on a study of a consecutive series of head injured patients who were found by CT scan to have an intracranial haematoma on admission to the Institute of Neurological Sciences in Glasgow, Scotland, during the three year period 01 January 1978 to 31 December 1980. The initial list of patients admitted to the Institute was obtained from the Glasgow Head Injury Data Bank.

The database for the study consists of patients clinical features (age, level of consciousness, pupillary reaction to light and outcome at six months follow up); CT scan features (type of intracranial lesion, basal cistern and third ventricle appearance, whether the ventricle contralateral to the intracranial haematoma was dilated or not, and the degree of midline shift measured in millimeters (mm); the duration of intracranial pressure monitoring and the level of intracranial pressure in millimeters of mercury (mmHg) when this was measured. The methods and sources used to obtain this information will now be described in detail.

3.2 THE GLASGOW HEAD INJURY DATA BANK

The Head Injury Data Bank in Glasgow was started in 1968 in order to accummulate patient information in a standardised way so as to provide a basis for management of new cases and for relating different treatment protocols to outcome (Jennett and Plum 1976). The information obtained has been, and still is, transcribed into a computer

compatible format for easier storage and potential future retrieval. The data collected prospectively consists of many variables; demographic aspects, level of consciousness at admission and at regular intervals thereafter until death or discharge, timing from injury to admission, type of intracranial haematoma or other CT scan diagnosis, laboratory investigations and outcome at regular intervals of follow up. The data is collected by especially assigned trainees on a regular basis. I had the privilege of being assigned full time to that programme through 1980 and I continued to be involved thereafter up to 1984. This opportunity gave me a wide experience in data collection and storage. By working in the out-patient head injury follow up clinic the methods of assessment of outcome were learned as were the problems of tracing patients who had failed to attend for a follow up. This proved to be a very useful experience when I conducted a separate study on the patients with intradural haematoma who had not been operated upon, the results of which constitute part of this thesis.

This Data Bank consists of a number of ongoing studies, each contributing to the database. At the time of this study the main ongoing studies were: The Severe Head Injury Study; the Operated Haematoma Study; and The Scottish Head Injury Management Study (SHIMS).

3.3 ASSESSMENT OF CONSCIOUSNESS: The Glasgow Coma Scale

The Glasgow Coma Scale was designed in order to standardise the assessment of impaired consciousness and

coma (Teasdale and Jennett, 1974). It was subsequently incorporated into the bedside neurological observation chart (Teasdale et al 1975) (Fig 1) and advocated later for world wide use (Langfitt 1978). This scale has now become the basis of neurological assessment in many neurosurgical centres in Europe, Asia, Japan, Australia and the Americas. A plea for its use in African Neurological Centres has recently been made (Kohi et al 1983).

It is a practical scale that is reliable when used by a wide spectrum of medical, paramedical and nursing staff (Teasdale et al 1978). It consists of three aspects of patient behaviour which are evaluated independently of each other: the stimulus required to induce EYE OPENING, the BEST MOTOR RESPONSE and the BEST VERBAL RESPONSE (Table 1). By allocating a score to each response a rank order is obtained with the lower scores indicating an increasing degree of brain dysfunction. The sum of the scores of the three responses, The Glasgow Coma Score, provides a practical means of expressing the overall level of responsiveness and actually provides better information about prognosis than any single component alone (Teasdale et al 1976). The minimum score is 3 and the maximum score is 15. By ranking the coma scores into groups such as 3-5, 6-7,8-10 and 11-15, it is possible to provide a rank order of the depth of impaired consciousness.

According to the Glasgow Coma Scale, coma is arbitrarily defined as inability to obey commands (best motor response 5 or worse), inability to open eyes even to pain (eye opening score = 1) and inability to utter recognisable

words (best verbal response = 1). This corresponds to an aggregate coma score of 7 or less (Table 1). This scale therefore forms the basis of assessment of consciousness in the patients in this thesis.

3.4 ASSESSMENT OF OUTCOME:

a) The Glasgow Outcome Scale

The need to standardize the assessment of outcome for the patients in the Glasgow Head Injury Data Bank was the impetus which resulted in the conception of the Glasgow Outcome Scale (Jennett and Bond 1975) (Table 2). As indicated in the review of literature, without a standard system of assessment it would not be valid to compare the relative efficacy of alternative methods of management in the different series of head injured patients. The Glasgow Outcome Scale, like the Glasgow Coma Scale, is now virtually a universally accepted method of assessment of outcome of patients after brain damage. It naturally forms the basis of assessment of results in this thesis.

The scale consists of five distinct outcome categories: DEAD; PERSISTENT VEGETATIVE STATE, SEVERE DISABILITY; MODERATE DISABILITY; and GOOD RECOVERY. The details are shown in Table 2.

By obviating the need for a detailed neurological and psychological assessment, the scale allows the overall social outcome of most patients to be assessed reliably on the basis of a structured interview which concentrates on

social and personal functioning. The scale therefore provides a basis for determining the degree of disability without a detailed description of the factors contributing to that disability.

b) Timing of Assessment

Six months follow up has been recommended as an optimum time at which to assess outcome in any series of brain damaged patients. This had arisen from the finding that 90% of those patients who had improved on their 3-month outcome by one year had already achieved the higher category within six months following the injury (Jennett et al 1981). Although motor and speech improvement are known to continue for a long time during rehabilitation, serial pychological testing has shown that recovery in cognitive function mostly occurs within the first six months (Mandelberg and Brooks, 1975, Bond, 1976) and that only about 5% of disabled patients can change to a better outcome category if followed for 18 months (Jennett et al 1981). Moreover, it had also been reported that the recovery curves of patients with severe injuries who were followed for 20 years showed that almost all who made good recoveries had done so by six months (Roberts 1976).

In this thesis therefore the assessment of outcome at six months will be the basis for determining the influence on results of the factors and management policy under study.

3.5 CLINICAL DATA

There are two main groups of patients in this thesis;

those operated and those not operated upon-during their initial or subsequent management. The sources of clinical data in either group will now be outlined.

(a OPERATED PATIENTS

From the current Operated Haematoma Study (part of the Glasgow Head Injury Data Bank) it was possible to list the patients who had been operated upon during the three years period under study. Cross-reference was made with the Operation Records Books to confirm that <u>all</u> the patients operated upon for a traumatic intracranial haematoma during that period were included.

In order to retrieve information from the Data Bank suitable computer programs were designed in the Department of Neurosurgery. The technical details of these computer programs will not be described in any detail. Each programme was designed to provide specific information which could be analysed in order to try and provide answers to a given set of questions in each chapter. Having extracted the raw data required, this was systematically analysed and eventually summarised as tables and figures. Each analysis was done by myself. At each step of the analysis the population of the patients studied was checked to ensure that some patients were not excluded. A test of statistical significance was applied whenever necessary.

The information retrieved for analysis was in regard to:

i) Patient's <u>age</u>

ii) <u>Level of consciousness</u> as determined by the Glasgow Coma Score. The coma score at admission

to the Institute and the final coma score recorded immediately preceding operation were obtained. This was in order to determine if there was a change in consciousness between initial and subsequent management.

iii) Pupillary reaction to light. There are three categories for analysis in this respect at admission and immediately preceeding operation;

1) One pupil reacts to light
2) Both pupils react to light
3) Neither pupil reacts to light

Timing of Operation. By analysing the times at which a patient was admitted to the Institute and taken to theatre for surgery it was possible to deduce how long it had taken from admission to diagnosis (CT scan) and to operation. An arbitray period of six hours from admission to diagnosis and operation was considered as having been adequate for the clinicians to have made up their minds as to the urgency of surgical treatment.

From this it could therefore be decided that an operation was immediate if surgery was undertaken within six hours or delayed if that had been considered necessary after that period.

b) PATIENTS NOT OPERATED

In the Institute there had not been a previous specific study on patients not operated upon. The Data Bank could not therefore provide the same amount of information for this group as was made possible in the operated patients.

I had to conduct a new study on these patients in order to be able to provide answers to the issues set out in my objectives.

1) NON - OPERATED HAEMATOMA STUDY

Since the Scottish Head Injury Management Study (SHIMS) consists of all head injury patients admitted to the Institute it was possible to initially list all the patients who were found to have an intracranial haematoma confirmed by CT scan. This was because information regarding patients who had been found to have a haematoma had previously been transcribed prospectively from the CT scan reports, made by one of the Consultant Neuroradiologists, and entered into the SHIMS data bank. By excluding those patients who had been operated upon, a list of the patients with intracranial haematoma but not operated upon was obtained. Again cross reference was made from admission records to ensure that some patients were not inadvertently excluded.

A study protocol was designed (Appendix 1) so that the information could be compiled in a computer-compatible format and enable matching the information available in the Data Bank on the operated patients. The many more variables included in the protocol were for the benefit of the Data Bank. The sources of information for this part of the study was obtained retrospectively from the clinical notes, nursing neurological charts (Fig 1) and notes, and discharge summaries all of which were obtained from the Medical Records Department. A decision regarding outcome at six months was arrived at from the available records.

In some instances, especially where the patients had defaulted from attending the outpatient clinic, communication was established by either telephone or correspondence to the patients, next-of-kin or family physicians (Appendix A).

ii) CLINICAL FEATURES

Eventually it was possible to extract information regarding the patients age, level of consciousness (Glasgow Coma Score) pupillary reaction to light and outcome as was in the operated patients section 3.5 (a) above.

3.6 INTERPRETATION OF COMPUTER TOMOGRAPHY (CT) SCAN FEATURES

1. Source of CT Scan Pictures

As indicated earlier, the initial listing of patients with acute traumatic intracranial haematoma in the study was linked to the radiological reports previously made by a consultant neuroradiologist. Using the list compiled from the Data Bank it was possible to obtain the patient's hospital files from the Medical Records Department. Each file was then examined in order to confirm the radiological diagnosis and to obtain the serial CT scan record number thereby enabling the original CT pictures to be recovered from the film library in the Department of Neuroradiology. When the original CT scan pictures could <u>not</u> be traced attempts were made to retrieve the original computer data stored on tapes through a rather technically complicated procedure. The information was transferred from tapes to computer discs from which new copies of the original CT scan pictures could be printed on ordinary x-ray films.

This retrieval procedure was time consuming and required the help of a Consultant Neuroradiologist.

The CT scans had all been done using either of the two second generation EMI 1010 Scanners. Each picture was printed as a standard 160 x 160 matrix with each film depicting nine pictures, Fig 2 to 5. All points of reference were comparable. In some unusual emergency events only a few slices of CT scan were done before a patient was rushed to the Operating Room because of serious and progressive neurological deterioration. In those few instances it was not possible to provide an interpretation of all the features under study.

2. RADIOLOGICAL FEATURES STUDIED

A) Type of Intracranial Haematoma

The CT scan definition of an acute traumatic intracranial haematoma is a lesion that is relatively hyperdense in relation to brain, Hounsfield Density Number 40 - 70. That density window was the basis of all CT scan prints.

Each CT scan was examined in order to define the type of lesion. This was described as follows:

i) Extradural Haematoma, a hyperdense extracerebral lesion with biconvex surfaces (Fig 2).

ii) Intradural Haematoma

1. Subdural haematoma, hyperdense extracerebral lesion with outer convex and inner concave surfaces (Fig 5).

2. Intracerebral Haematoma, hyperdense intraparenchymatous lesion usually of irregular configurations (Fig 3,4, and 4a). Excludes lesions seen exclusively in basal ganglia and corpus callosum which indicate severe primary or impact brain damage.

B) Other CT Scan Features

Four other CT scan features were studied:

1). Basal Cistern Appearance

Theseare seen on the lower slices of CT scan pictures. They are defined as the cerebrospinal fluid pathways or cisterns that surround the brain stem; perimesencephalic, interpeduncular, cisterna ambiens and quadrigeminal cisterns. They are normally depicted as regions which are hypodense relative to brain.

The appearance of the basal cistern was described either as NOT OBLITERATED (Fig 2, 4a) or as being OBLITERATED (Fig 5).

2). Third Ventricle Appearance

This is the CSF pathway that can be identified on a CT scan picture as being above the fourth ventricle but below the lateral ventricles. This was described as NOT OBLITERATED when visualised (Fig 4) or as being OBLITERATED (Fig 5) when the CT slices of the expected level could not reveal its presence.

3). Contralateral Ventricle Dilatation

The lateral ventricles are easily delineated on a CT scan and are usually grossly symmetrical (Fig 4). They may be slightly compressed (Fig 3). When there was gross asymmetry on the sizes of the ventricles such that the ventricle contralateral to the intracranial haematoma was undeniably proportionately larger than the ipsilateral ventricle then that CT scan was described as showing contralateral ventricle dilatation (Fig 4a, 5). No absolute measurements of ventricle size were done as such a technique was NOT available and is still not readily available for clinical use when interpretation of CT scan findings is made often in emergency situations This phenomenon as described in this thesis therfore reflects a personal interpretation as indeed have all the other features outlined.

4) Midline Shift

I measured the displacement of the septum pellucidum from a midline position. The midline position was projected between fixed landmarks. The landmarks were either the crista gali or the most anterior hyperdense position of the falx cerebri and the occipital protuberance or the most posterior fixed part of the falx cerebri (Fig 2,5). The measurements were done using a transparent millimetre ruler and a transparent midline marker. I repeated these measurements in order to achieve consistency of readings.

On the standard 160 x 160 matrices on the second generation EMI 1010 Scanners each millimetre measured corresponds to 5mm in absolute measurements. The measurements on CT scan pictures were made to the nearest millimetre and later converted into absolute values as 0,5,10,15,20,25 and 30mm.

3.7 ANALYSIS OF INTRACRANIAL PRESSURE FEATURES

1. Intracranial Pressure Monitoring: Technique

It is possible to measure ICP by placing a sensoring device in the lateral ventricles, the subdural space or in the extradural space. By far the most reliable method is to measure the ICP changes generated within the ventricles (Mendelow et al 1981). This was the standard procedure used in this series.

After fashioning a burr hole in the frontal region contralateral to the side of the haematoma, a saline filled 8FG calibre catheter was inserted into the lateral ventricle. This was then connected to a transducer whose position was adjusted to match the level of the lateral ventricles with the patient in the supine position (the zero level) as all readings of ICP are relative to that level. The transducer was then connected to a paper-pen chart recorder so as to obtain a permanent tracing for that patient (Figs 6 a,b,c and d). The scale was calibrated in millimetres of mercury mmHg ensuring that the baseline reading was zero relative to the atmosphere. A summary of readings of the level of the mean ICP over 15 minutes were entered as a routine into the neurological observation

chart by the attending nurse in order to provide another permanent record.

It was routine to exclude, by arterial blood gas determination, hypercarbia ($PaCO_2$ >45mmHg) or hypoxia ($PaCO_2$ <65mmHg) as factors contributing to a rise in ICP. It was equally important routinely to recalibrate the scale to exclude changes in patient position (i.e. shifting the zero level) contributing to changes in intracranial pressure readings.

I was personally involved in the monitoring of patients seen from 1980.

2. ANALYSIS OF ICP TRACINGS

In the review of literature, paragraph 2.3, the different ways in which intracranial pressure features can be analysed were critically summarised. Since the aim of this study was to find out how management decisions and results can be affected by the magnitude of ICP as had been previously suggested by Galbraith and Teasdale (1981) the analysis of ICP tracings will therefore be concerned first and foremost with the level of ICP. In addition the duration of monitoring will be examined in order to determine how long it takes to arrive at a further management decision after initiating intracranial pressure monitoring.

3. SOURCES OF DATA

Examination of the patients hospital files showed which patients had intracranial pressure monitoring. This was

counter-checked with the Operations Record Books in order to ensure that all patients who had burr holes for monitoring of ICP during the period under study were included.

From the records it was possible to ascertain when monitoring had commenced and had ceased (hence duration) and the level of ICP the highest mean ICP recorded that persisted for at least 15 minutes was noted. The patients ICP tracings were individually reviewed in order to confirm the information obtained.

3.8 STATISTICAL ANALYSIS

This was done in collaboration with and supervised by statistical staff of the University Department of Neurosurgery, University of Glasgow. The Chi square test of statistical significance was the method of choice.

TABLE 1

THE GLASGOW COMA SCALE

Parameter	Response	Score
EYE OPENING, E	Spontaneously	4
	To speech	3
	To pain	2
	None	1
BEST MOTOR RESPONSE, M	Obeys commands	6
	Localises to pain	5
	Flexion (withdrawal to pain	4
	Abnormal flexion to pain (Decorticate posturing)	3
	Extension to pain (Decerebrate posturing)	2
	None	1
BEST VERBAL RESPONSE, V	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1

GLASGOW COMA SCORE, GCS, = E + M + V

Lowest GCS = 1 + 1 + 1 = 3

Highest GCS = 4 + 6 + 5 = 15

Coma GCS + 1 + 5 or less + 1 = 7 or less

TABLE 2

GLASGOW OUTCOME SCALE

OUTCOME CATEGORY	DESCRIPTION
Dead, D	
Persistent Vegetative State, V	Entirely dependent. Shows no psychologically meaningful responses. Have wake and sleep rhythms and may occasionally open eyes spontaneously. Have abnormal motor response in all four limbs usually with spasticity.
Severe Disability, SD	Conscious but dependent Depends on the help of others for some activities during every 24 hours. Includes marked physical, mental and cognitive disabilities. Often needs to be permanently institutionalised.
Moderate Disability, MD	Independent but disabled. Disabilities include memory or personality changes, hemiparesis, dysphasia, ataxia or epilepsy. Able to return to work and participate in some social activities.
Good Recovery, GR	Recovery with restoration of normal functions, normal social participation. Able to return to full time work although there may be a minor persisting sequelae.

FIGURE 1

Neurological Observation Chart

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OBSERVATION CHART

NAME

RECORD No.

DATE

TIME

COMA SCALE

Eyes open	Spontaneously To speech To pain None	Eyes closed by swelling = C
Best verbal response	Orientated Confused Inappropriate Words Incomprehensible Sounds None	Endotracheal tube or tracheostomy = T
Best motor response	Obey commands Localise pain Flexion to pain Extension to pain None	Usually record the best arm response

Pupil scale (m.m.)

1 2 3 4 5 6 7 8

Blood pressure and Pulse rate: 240 230 220 210 200 190 180 170 160 150 140 130 120 110 100 90 80 70 60 50 40 30 20 10

Respiration

Temperature °C: 40 39 38 37 36 35 34 33 32 31 30

PUPILS	right	Size Reaction
	left	Size Reaction

+ reacts
− no reaction
c. eye closed

LIMB MOVEMENT

ARMS	Normal power Mild weakness Severe weakness Spastic flexion Extension No response
LEGS	Normal power Mild weakness Severe weakness Extension No response

Record right (R) and left (L) seperately if there is a difference between the two sides.

Fig. 1

FIGURE 2

Frontoparietal extradural haematoma
Basal cisterns NOT obliterated, BC
Third ventricle obliterated
contralateral ventricle Dilated, best
shown in occipital and temporal horns
Midline shift 15mm

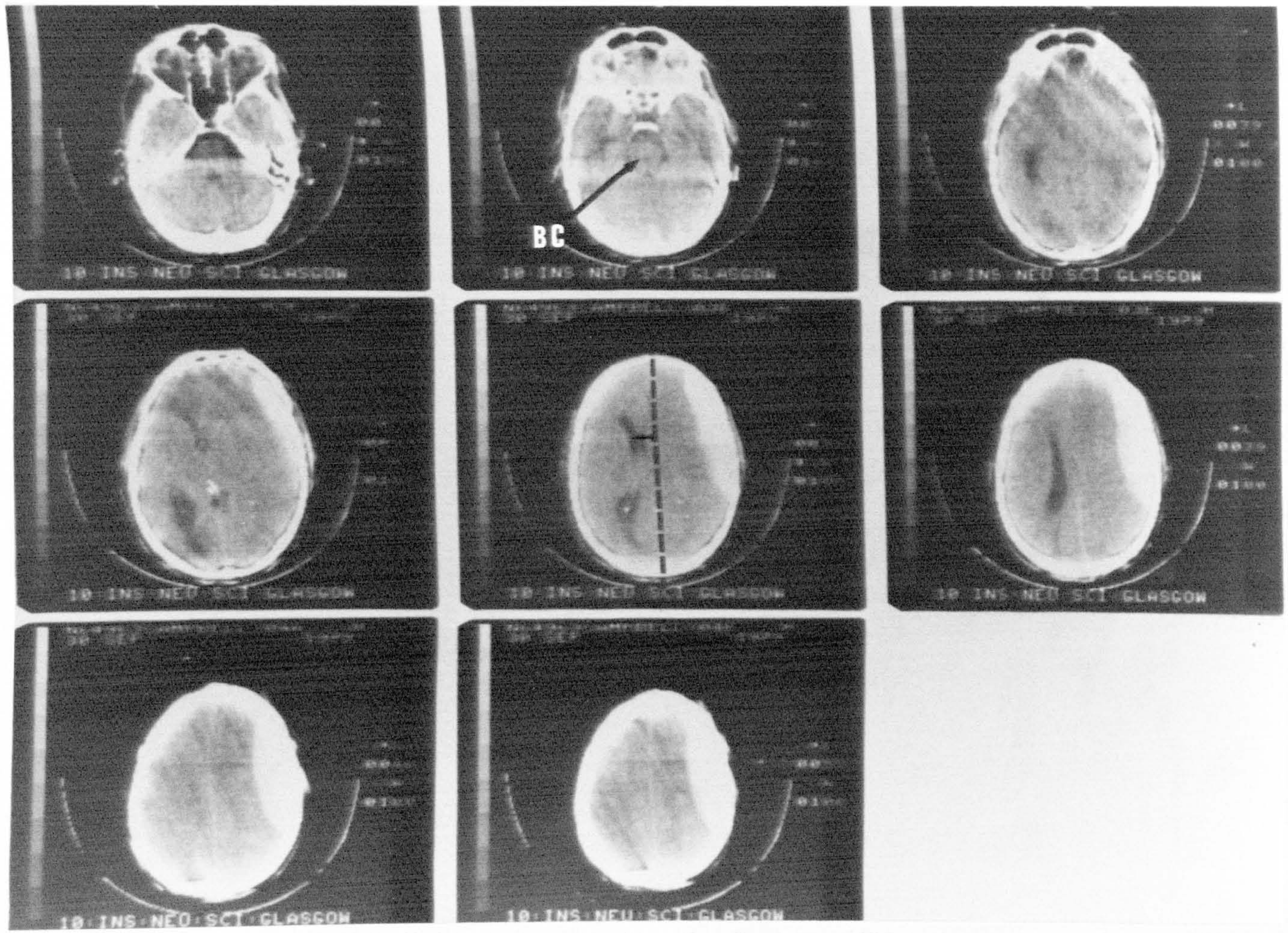


Fig. 2. Frontoparietal extradural haematoma; Basal cisterns NOT obliterated, BC; Third ventricle obliterated; contralateral ventricle dilated, best shown in occipital and temporal horns. Midline shift 15mm.

FIGURE 3

Temporal Intracranial haematoma, ICH
Basal Cisterns NOT obliterated
Third Ventricle NOT obliterated
Right Lateral Ventricles compressed
Contralateral Ventricles NOT dilated
No midline shift

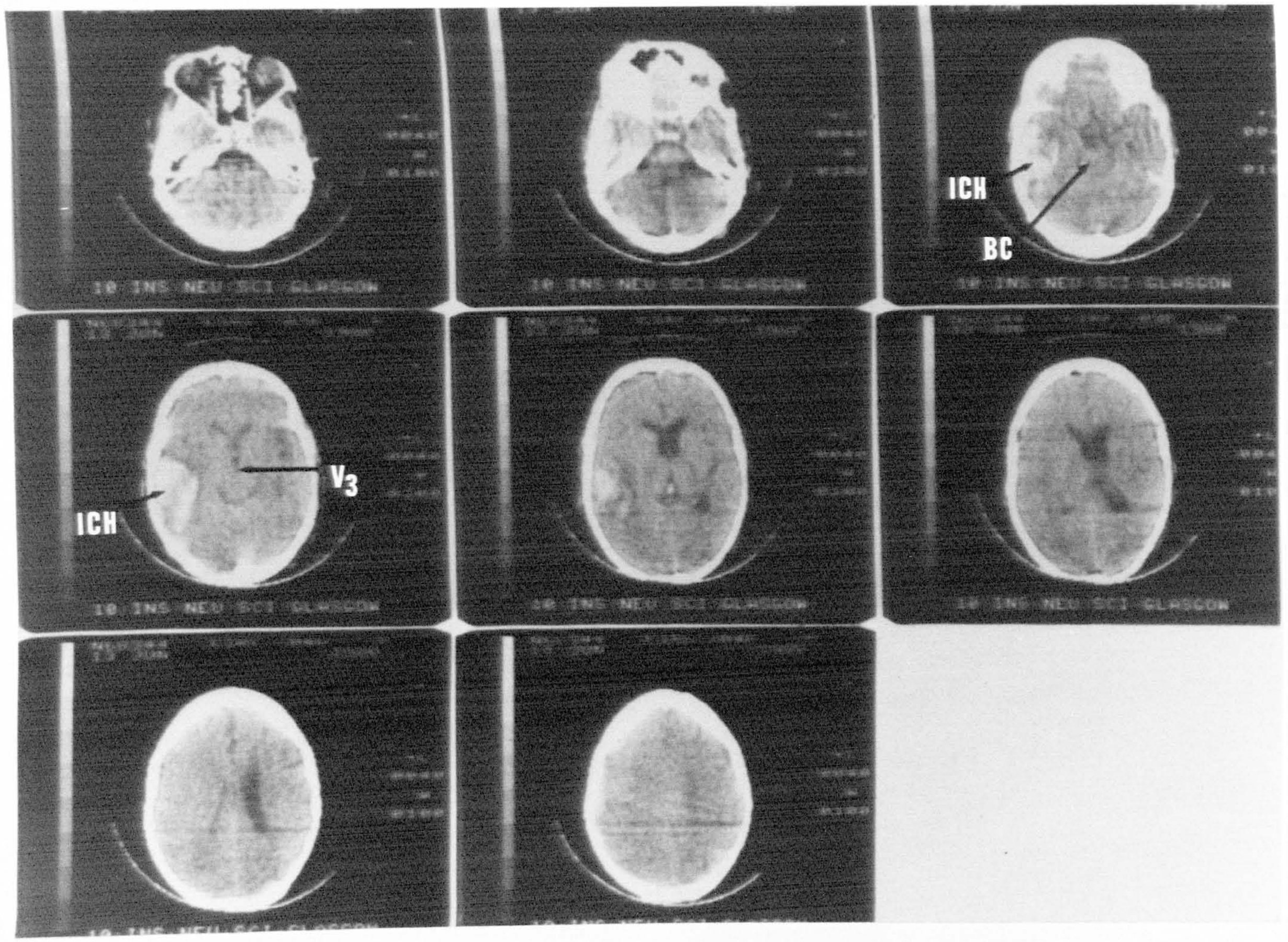


Fig. 3. Temporal intracranial haematoma, ICH; Basal cisterns NOT obliterated; Third ventricle NOT obliterated; Right lateral ventricles compressed; Contralateral ventricles NOT dilated; No midline shift.

FIGURE 4

Temporal intracerebral haematoma, ICH
Basal Cisterns NOT obliterated, BC
Third Ventricle NOT obliterated, V3
No contralateral Ventricle dilatation
(ventricles grossly symmetrical)
No midline shift

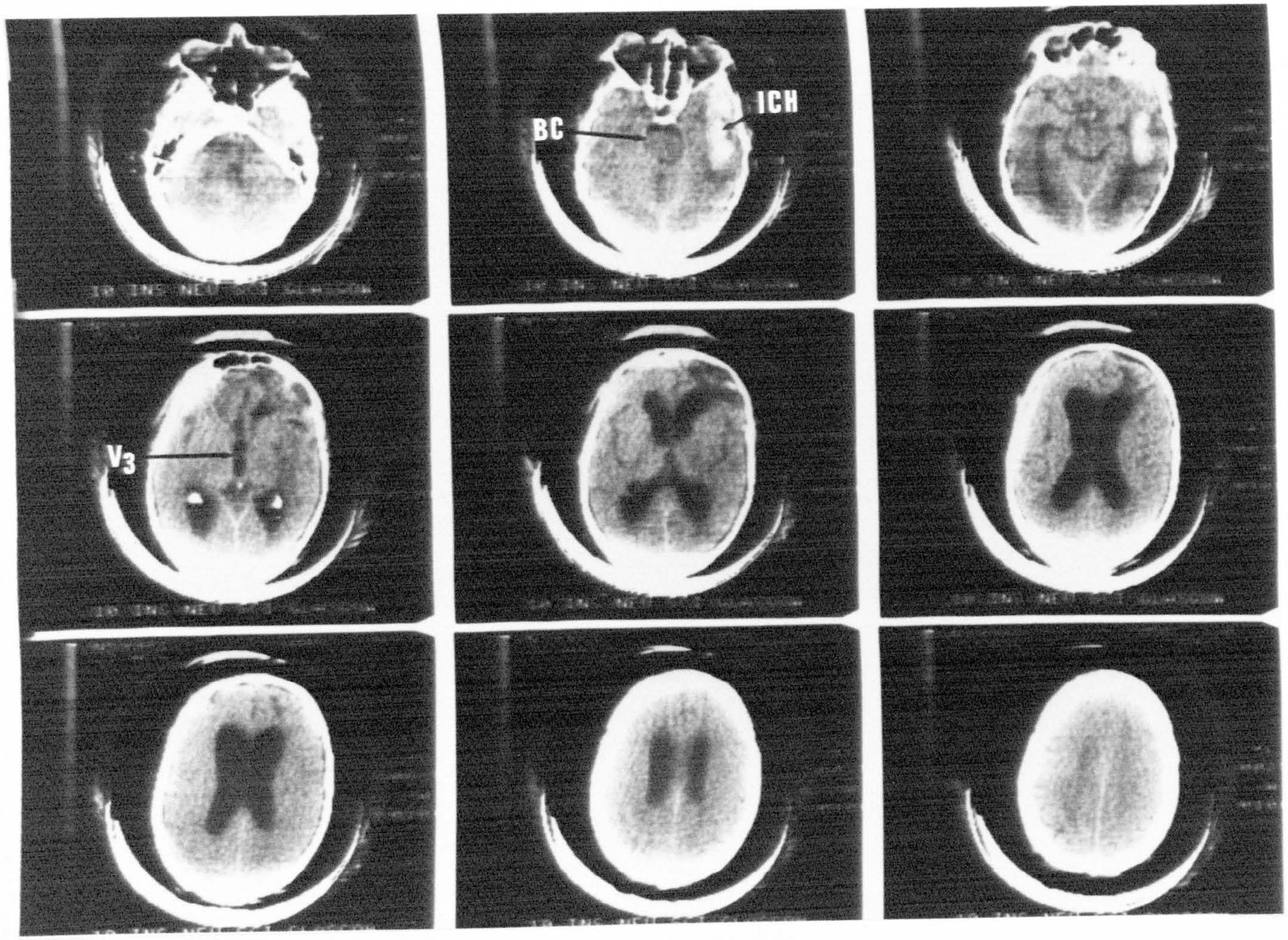


Fig. 4. Temporal intracerebral haematoma, ICH; Basal cisterns NOT obliterated, BC; Third ventricle NOT obliterated, V3; No contralateral ventricle dilatation (ventricles grossly symmetrical); No midline shift.

Figure 4a

Temporal intracerebral haematoma, ICH
Obliterated basal cisterns, BC
and third ventricl
CVD seen in temporal and occipital horns,
Midline displaced

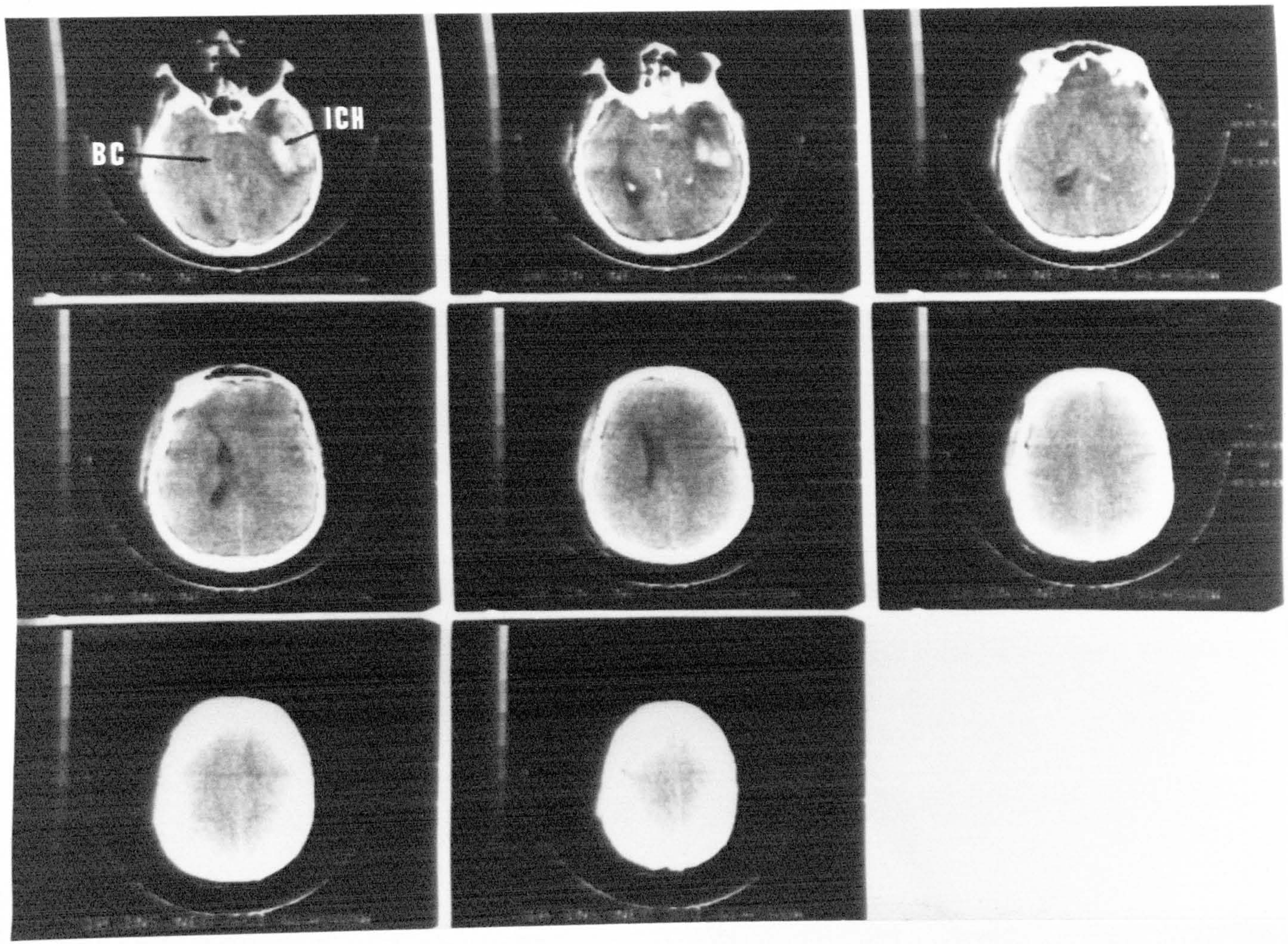


Fig. 4A. Temporal intracerebral haematoma, ICH; Obliterated basal cisterns, BC, and third ventricle CVD seen in temporal and occipital horns, midline displaced.

FIGURE 5

Fronto-temporal intracerebral haematoma, ICH
Fronto temporo-parietal subdural haematoma, SDH
Obliterated Basal Cisterns, BC
Obliterated Third Ventricle, not visible
Contralateral ventricle dilated
Midline shift 15mm

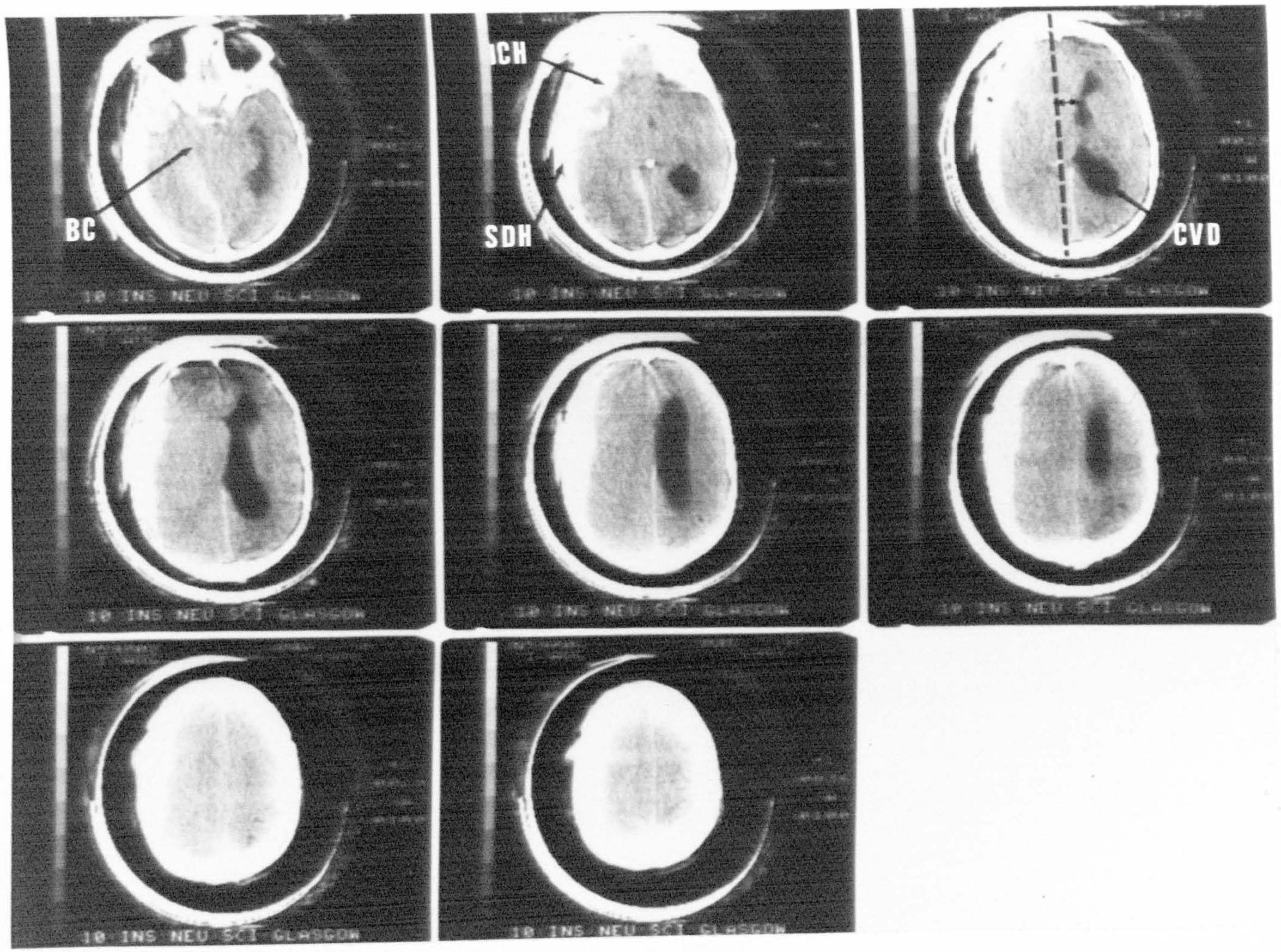


Fig. 5. Fronto-temporal intracerebral haematoma, ICH and fronto temporo-parietal subdural haematoma, SDH; Obliterated basal cisterns, BC; Obliterated third ventricle, not visible; Contralateral ventricle dilated; Midline shift 15mm.

FIGURE 6

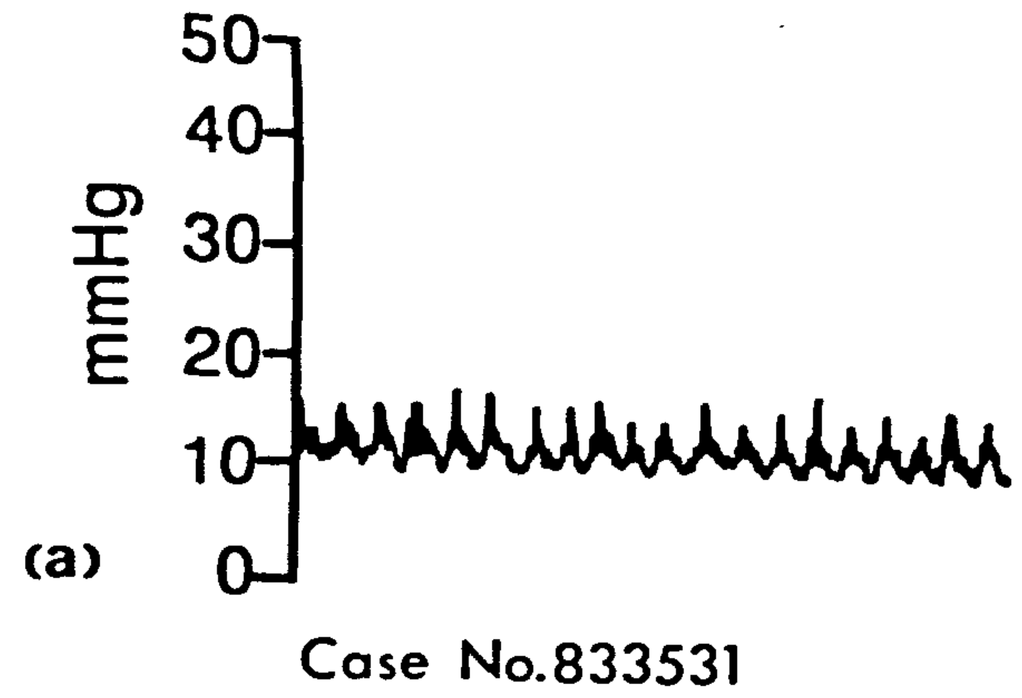
INTRACRANIAL PRESSURE TRACINGS

A. Mean ICP 10 - 20mmHg

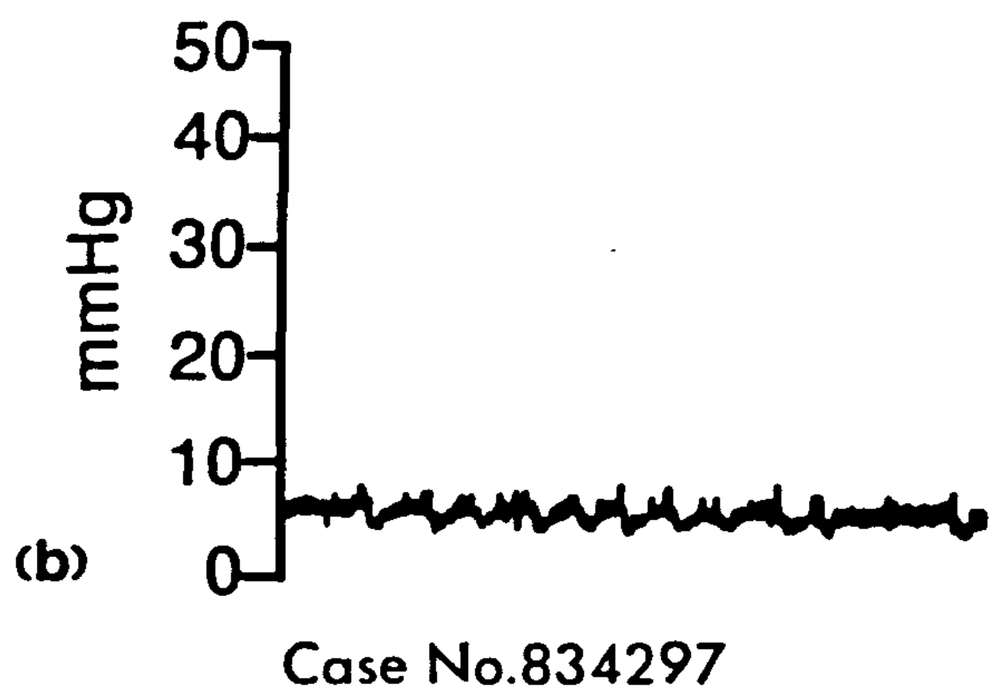
B. Mean ICP 0 - 10mmHg

C. Mean ICP 20 - 30mmHg

D. Mean ICP > 30mmHg



Case No.833531



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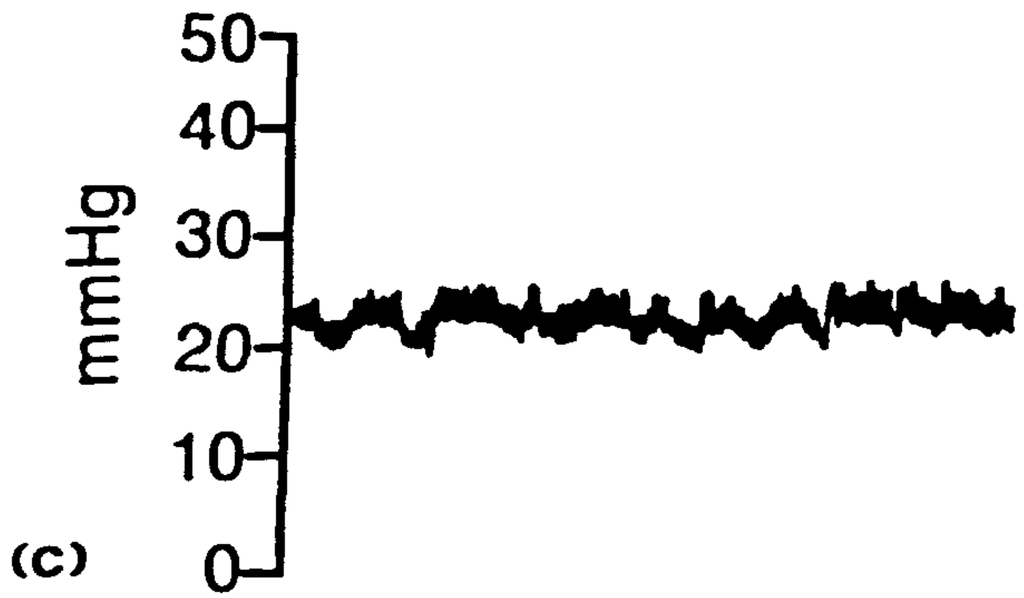

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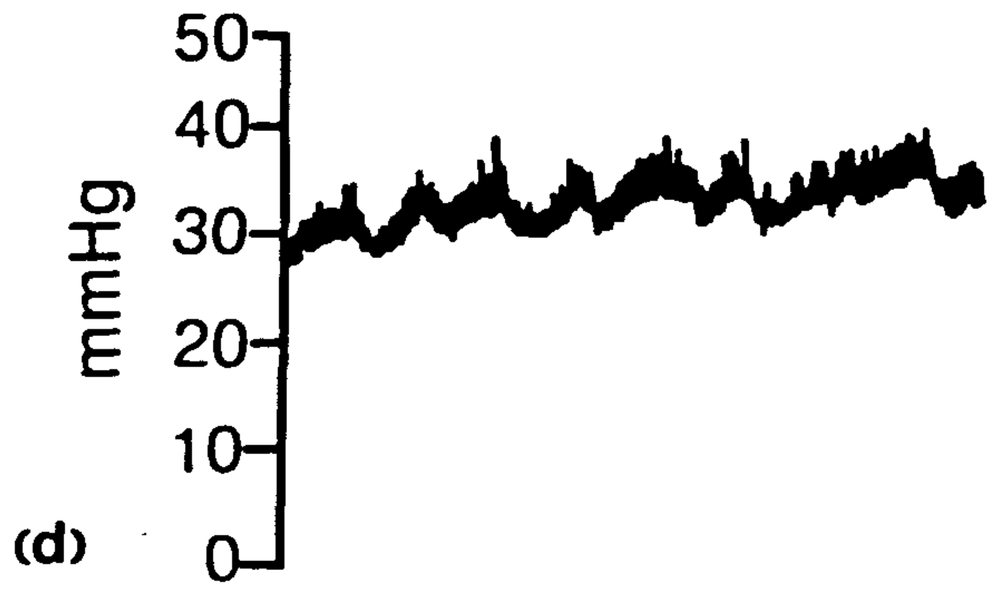


Fig. 6. a. Mean ICP > 10mmHg b. Mean ICP 0-10mmHg
c. Mean ICP 20-30mmHg d. Mean ICP > 30mmHg

CHAPTER 4

THE STUDY POPULATION

4.1 INTRODUCTION

The first part in the presentation of results will provide an overview of the patients in this study. In doing so it will be possible to characterise the features and management of these patients before proceeding to a more detailed analysis of factors already outlined in my objectives.

The emphasis will therefore be to define the patient population, the frequency and types of acute traumatic intracranial haematoma found, the type of initial and subsequent management and the overall results obtained at follow up.

4.2 RESULTS

a. PATIENT POPULATION

From the Glasgow Head Injury Data Bank 479 patients were identified as having had a CT scan diagnosis of an Acute Traumatic Intracranial Haematoma at admission to the Institute during the three year period under study (Fig 7).

There were 368 males and 111 females giving a Male:Female ratio of 3:1, confirming the male preponderence reported in most other head injury series. The age range was 1 - 84 years with a mean of 44 and median of 45 years.

b. TIME FROM INJURY TO ADMISSION TO THE INSTITUTE

As already stated in the methods, the criteria for inclusion of patients into this study was that the patients must have had a haematoma sustained within 14 days from

injury to diagnosis. Information was therefore extracted from the Data Bank and analysed in order to find out the pattern of time from injury to secondary transfer from the primary surgical ward to the Institute in order to substantiate that criteria.

The results show that of all the patients in the Data Bank, 142 (30%) were admitted within six hours, 211 (44%) within 12 hours, 274 (57%) within 24 hours and 364 (76%) within 72 hours from injury (Table 3).

c. TYPE OF INTRACRANIAL HAEMATOMA

The CT scan pictures of 411 patients (86% of the Data Bank Patients) were available for a detailed study but were not traceable in the remaining 68 patients.

Of the 411 patients whose CT scan findings were analysed, 345 (84%) had intradural haematoma while the remaining 66 (16%) had extradural haematoma (Fig 7).

d. TYPE OF INTITAL MANAGEMENT

The management of patients with an extradural haematoma was noticeably different from those with intradural haematoma.

1) Extradural Haematoma (Fig 7)

The initial decision in the management of patients with this type of haematoma had been to operate upon them early, usually soon after that diagnosis was confirmed. This was the accepted practice at this Institute.

2) Intradural Haematoma (Fig 8)

There was a variety of ways in which patients with intradural haematoma were initially managed. Broadly there are two main groups: those who had intracranial pressure monitoring and those who were managed without ICP monitoring, the clinically monitored patients.

a) Patients observed with ICP monitoring

Intracranial pressure monitoring was the first line of management in 18% of patients with intradural haematoma.

b) Patients managed without ICP monitoring

By far this constituted the largest group, 82% of patients with intradural haematoma. In these patients there were two distinct subgroups.

i) Immediate operation

Surgery was deemed of immediate need in just over half (52%) of all the patients with intradural haematoma.

The initial decision for early surgery was based on the neurological condition of the patient.

ii) Patients observed clinicially

There were 96 (28%) patients in this group. In the majority of these patients the clinicians had initially thought that there was a reasonable prospect of recovery without further management. In a small proportion (3%) further measures were considered unlikely to alter a predictably poor prognosis.

In the analysis of the different initial management groups 7 (2%) patients had not been ICP monitored but it could not be determined from the available records as to whether they had been operated upon early or had a delayed operation (Fig 8).

e. FINAL MANAGEMENT OF PATIENTS WITH INTRADURAL HAEMATOMA

Of the 345 patients with an intradural haematoma, 244 (71%) required surgery while 101 (29%) were never operated upon (Fig 7).

In the patients who were operated upon, 73% had immediate surgery while 24% had surgery after an interval. Of those who had 'interval' surgery, the indication for operation was clinical observations (usually deterioration) in half of them and due to intracranial pressure monitoring in the remaining half.

f. OVERALL OUTCOME

The management results of the patients in this series are shown in Table 4. It was earlier stated that there were 68 patients in whom CT scan pictures could not be obtained for further detailed analysis. The results have therefore been presented in order to show the outcome of all the patients in the Data Bank and also in the patients in whom CT scan pictures were available for CT scan related studies. It can be seen that the overall management results do not differ between the CT scan study patients and all the Data Bank patients.

The patients who at follow up were known to be alive but who were not available for categorisation of outcome were regarded as better than being severely disabled. This was borne from the finding that these patients had moved from their previous known addresses in order to undertake new social activities; attend school or college; get married; take up new employment; or had been 'well' enough as to emigrate. For practical purposes these patients known to be alive will in future be considered together with those with **moderate disability** and **good recovery** as having had a **favourable outcome** while those who were either **dead** or remained in a **persistent vegetative state** or were **severely disabled** will also be considered together as having had an **unfavourable outcome.** From the results then nearly 40% of all the patients in this series had an unfavourable outcome. Only 2% of patients were lost at follow up. There will be a detailed analysis of the outcome of these patients in chapter 8.

4.3 DISCUSSION

The features of the patients so far analysed in this series are similar to any large series of acute head injured patients. Of particular note were the age distribution, pattern of admissions, frequency of the different types of traumatic intracranial haematoma and the overall management outcome. This series therefore provides the material needed in order to draw conclusions so as to try and provide answers to the problems set out in the introduction to this work.

As dictated by custom at this Institute, all patients with extradural haematoma were operated upon early. I should note, however, that there have been recent claims that these haematomas occasionally can also be managed nonsurgically especially in children (Pang et al 1983). However, this study does not address itself to the non-operative management of extradural haematoma.

There is a broader spectrum of the pattern of management of patients with intradural haematoma. It can be deduced from the analysis of the initial management that there is a firm decision to operate forthwith in about half of patients with acute traumatic intradural haematoma.

The perception that the value of intracranial pressure monitoring was not clearly established as an alternative to the time honoured clinical observations is shown by the finding that only a fifth of patients had intracranial pressure monitoring compared to a third who were clinically observed. In particular there were variations depending on the clinician in attendance at the time of this study. In the event ICP monitoring led to a decision for delayed operation in as many patients as did clinical observation alone. Thus ICP monitoring tended to be used more often in patients in 'real' uncertainty. This must be borne in mind because it confirms that the two groups were not randomly selected and therefore not directly comparable.

From the analysis of the patterns of final management it can also be seen that in all 7 out of 10 patients with an intradural haematoma did need surgery. The corollary to this is that based on clinical grounds, there was need for

immediate operation in about three quarters of the patients who underwent surgery. It can therefore be inferred from this that there was initial uncertainty for the need for surgery in about a quarter of the patients who eventually actually needed it. Whether this could have been predicted from the initial clinical and investigative information is an important question to pursue.

Figure 7

THE STUDY POPULATION

The Study Population

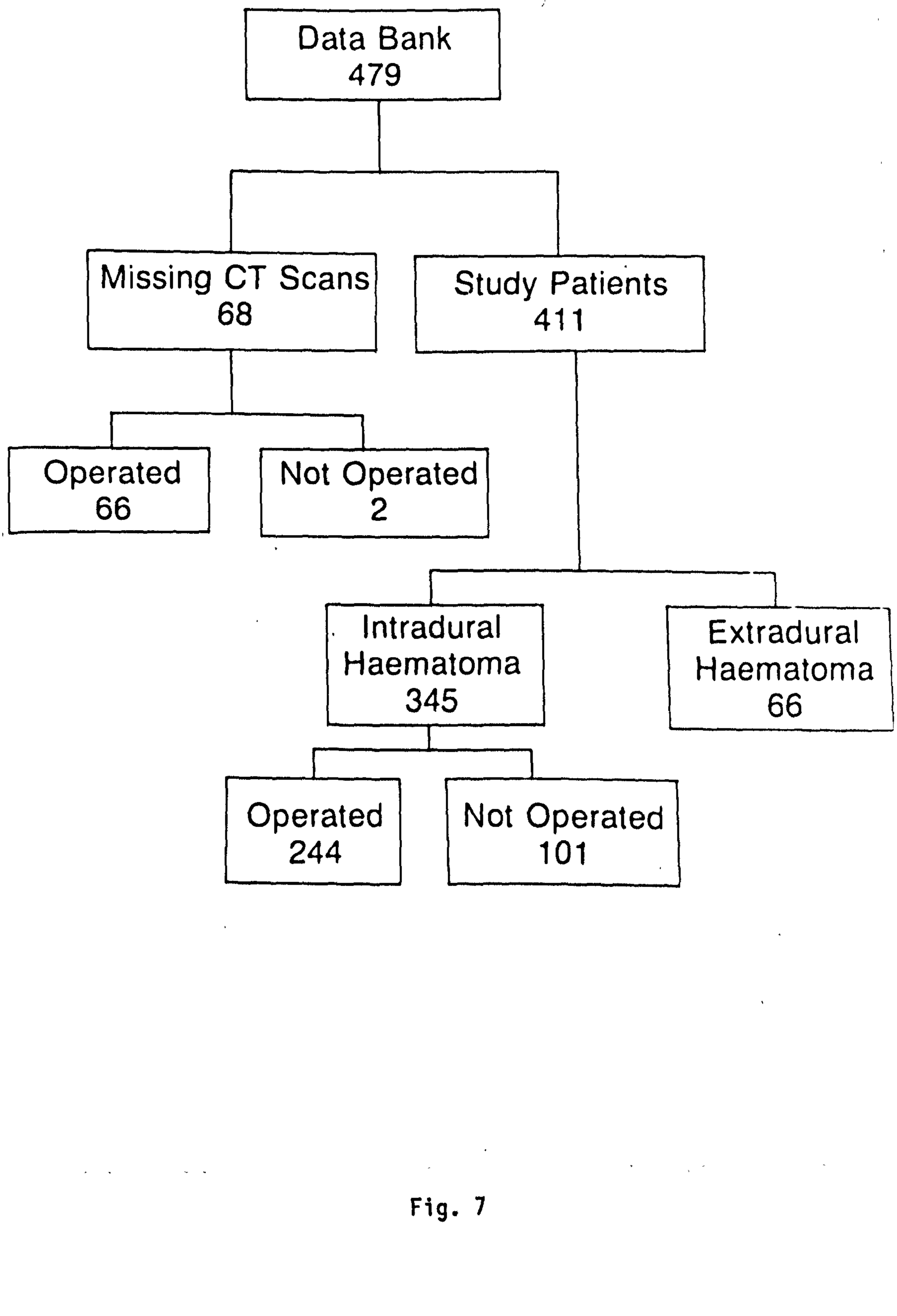


Fig. 7

Figure 8

MANAGEMENT OF INTRADURAL HAEMATOMA

Management of Intradural Haematoma

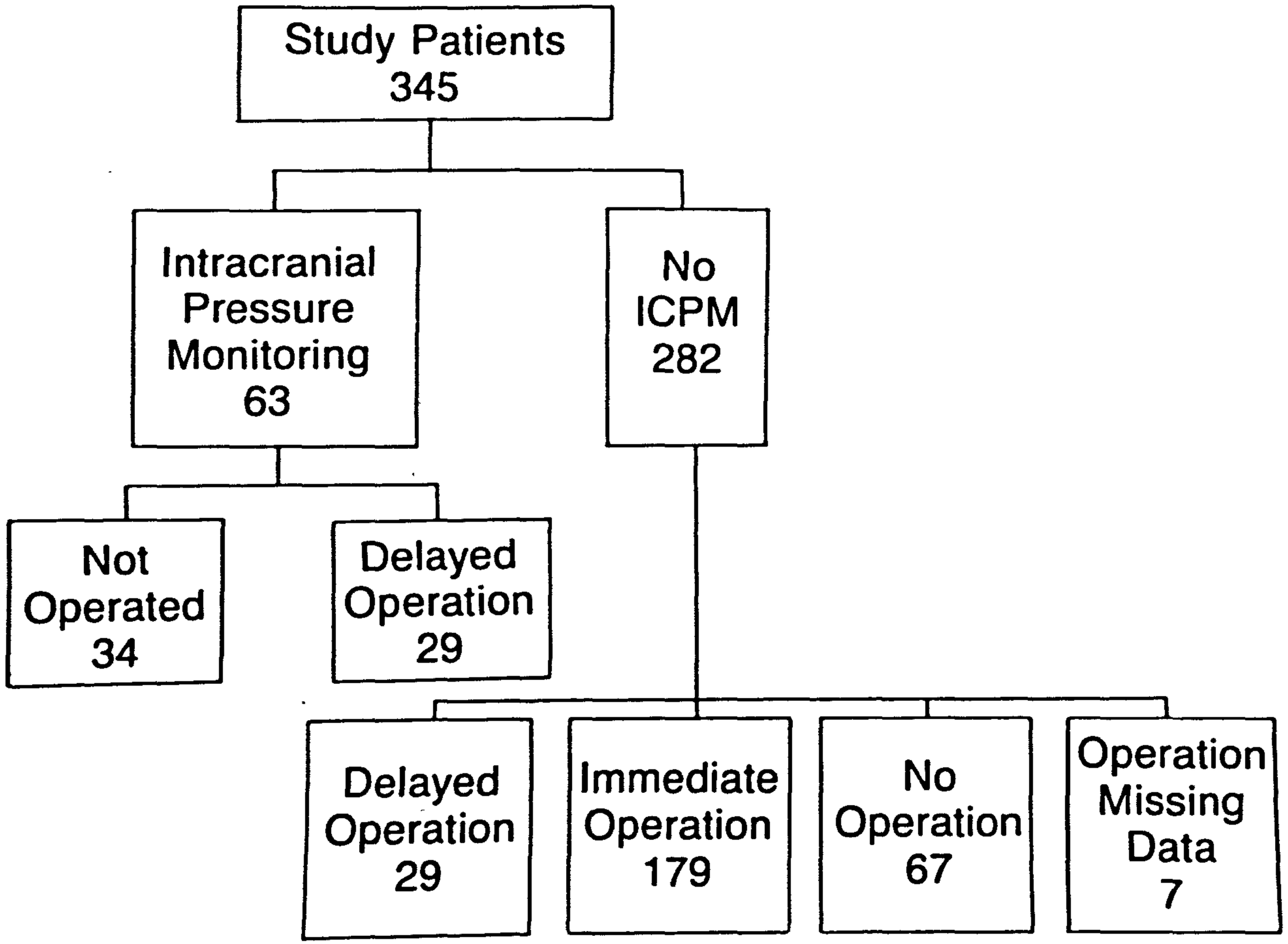


Fig. 8

TABLE 3

TIME FROM INJURY TO ADMISSION AT INS

Time (hours)	Number of Patients	%
< 1 hr	6	1%
1 - 3 hrs	59	12%
3 - 6 hrs	77	16%
6 - 12 hrs	69	15%
12 - 24 hrs	63	13%
24 - 72 hrs	90	19%
> 72 hrs	106	22%
Unknown	9	2%
TOTAL	479	100%

TABLE 4

OVERALL OUTCOME AT

SIX MONTHS FOLLOW UP

Outcome Category	All Data Bank Patients		Missing CT Scan Patients	CT Scan Study Patients	
	n	(%)	n	n	(%)
Dead	131	(27%)	16	115	(28%)
Vegetative	5	(1%)	0	5	(1%)
Severe Disability	46	(10%)	6	40	(10%)
Moderate Disability	84	(18%)	19	65	(16%)
Good Recovery	165	(34%)	18	147	(36%)
Alive	40	(8%)	9	31	(8%)
Lost	8	(2%)	0	8	(2%)
TOTAL	479	(100%)	68	411	(100%)

CHAPTER 5

RELATION BETWEEN CT SCAN FINDINGS AND CLINICAL FEATURES

5.1 INTRODUCTION

The clinical state has always been of paramount importance in the initial and subsequent decisions about management of patients suspected of acute traumatic intracranial haematoma. As impaired conscioiusness is an accepted measure of the severity of brain damage it follows that changes in the level of consciousness reflect changes in intracranial dynamics, both anatomical and pathophysiological.

The development of an intracranial haematoma does cause secondary brain damage whose severity can be gauged by the extent of the change in the level of consciousness. The mass effect of a haematoma is held to be responsible for the changes in the topographical relations and pathophysiological parameters of which the clinical sequelae is the deterioration in consciousness. It can be re-iterated that deterioration in consciousness has always been the hallmark of clinical decisions about management of head injured patients. Information assisting in determining which patients were likely to deteriorate, and therefore needing early surgical intervention, would be valuable.

The response of the pupils to light is an important clinical index of brainstem function. It has been traditional to emphasise the significance of pupillary function in the initial and subsequent monitoring of all head injured patients. This practice can be attributed to Hutchinson who, over one hundred years ago, described two cases of unilateral pupillary dilatation resulting from a

temporal extradural haematoma (Hutchinson 1867, 1878). As an emphasis to the 'new' sign Rowbotham said "a fixed dilated pupil is an infallible sign of raised intracranial pressure and it is a feature of paramount surgical significance for if the uncinate herniation is not relieved the patient is almost certain to succumb" (Rowbotham 1964). The observation that pupillary dysfunction was due to tentorial hernation has long since been confirmed by experimental (Jennett 1960), pathological and anatomical (Sunderland 1958, Plautt 1963) and radiological studies (Plautt, 1960). Azamuja et al 1956, Finney and Walker 1962, Liliequest 1960, Komaki and Handel 1974, Osborn 1977, Stroving 1977a).

Since CT scan provides better visualisation of intracranial topography in life then there is a need for better understanding about what significance could be attached to the different CT scan findings. What can we learn from CT scan about the patients present clinical state and the likely future course, especially the need for operation or not and eventual outcome? In this chapter I will examine the relation between CT scan and clinical features at the time of the initial CT scan and so explore the significance of topographical CT scan findings.

5.2 PATIENTS & METHODS

1. The CT scan features to be analysed will be:

a) Basal Cistern Appearance

b) Third Ventricle Appearance

c) Presence of Contralateral Ventrical Dilatation

d) Midline Shift

2. The clinical features to be analysed will be:

a) The level of consciousness as determined by the Glasgow Coma Score. This will be divided into rank order groups of Coma Score 3 - 5, 6 - 7, 8 - 10, and 11 - 15 in order to provide a spectrum of the measure of the level of consciousness. The Glasgow Coma Score is that value recorded immediately proceeding CT scanning.

b) The reaction of the pupils to light immediately preceeding CT scanning. This will be categorised as one, both or neither reacting to light as indicated in Chapter 3.

All patients will be included in this part of the study so as to enable evaluation of the mass effect of an intracranial haematoma regardless of type.

5.3 RESULTS

The results of the analysis of the relations between the CT scan features and clinical features will be presented in three parts. The first part will be with regard to the level of consciousness while the second part will be with regard to pupillary function or dysfunction. Finally the inter-relationship between the CT scan features will be analysed.

a. CT SCAN FEATURES AND THE LEVEL OF CONSCIOUSNESS

Each of the four CT scan features will be analysed individually.

i) Basal Cistern Appearance (Table 5)

Of the 383 patients in whom both the basal cistern appearance and coma score was known, 186 (49%) had obliterated cisterns.

There were 161 patients in coma, of whom 124 (77%) had obliterated cisterns. In contrast, only 62 (28%) of the 222 patients who were not in coma also had obliterated basal cisterns.

In the 186 patients who had obliterated basal cisterns 124 (76%) were in coma compared to only 37 (19%) of the 197 patients whose basal cisterns were not obliterated.

When the different coma groups are examined further then the proportion of patients who had obliterated cisterns in coma score groups 3-5, 6-7, 8-10, and 11-15 is 85%, 63%, 41% and 22% respectively.

These differences are statistically significant (X^2 = 105.64, df = 3, $p<0.001$).

ii) Third Ventricle Appearance (Table 6)

Both the appearance of the third ventricle and coma score was known in 386 patients of whom 205 (53%) had an obliterated third ventricle.

Of the patients in coma, 129 (80%) had an obliterated third ventricle compared to only 76 (34%) of the patients who were not in coma.

For each of the coma score groups 3-5, 6-7, 8-10, and 11-15 there were 88%, 67%, 38% and 32% of patients respectively had an obliterated third ventricle.

In the patients in whom the third ventricle was obliterated, 129 (63%) were in coma while only 33 (18%) of the patients in whom the third ventricle was NOT obliterated were also found to be in coma. The differences are statistically sifnificant (X^2 = 86.46, df = 3, p<0.001).

iii) Contralateral Ventricle Dilatation (Table 7)

The topography of the ventricles was known in 388 patients, of whom in 185 (48%) the contralateral ventricle was dilated.

In the 163 patients who were in coma 113 (69%) had contralateral ventricle dilatation but only 72 (32%) of the patients not in coma also had a dilated contralateral ventricle.

For each of the coma score groups 3-5, 6-7, 8-10 and 11-15 there were 79%, 56%, 33% and 31% of patients respectively who were found to have a dilated contralateral ventricle. Coma was observed more frequently in patients whose contralateral ventricle was dilated than in those in whom the ventricles were symmetrical 61% and 25% respectively. The association was statistically significant (X^2= 55.45, df = 3, p<0.001).

iv) Midline Shift (Table 8, Fig 9)

This could be determined in 391 patients, 164 (42%) were in coma and 227 patients were not in coma.

How frequently coma was observed for each shift was derived from Table 8 and is shown in Figure 9.

Nearly 1 in 5 (19%) of the patients without a shift were found to be in coma. This proportion increased to about 1 in 3 for a 5mm shift but at 10mm shift half of the patients were in coma and so were three quarters of those with a 15mm or more shift. This shows that increasing midline shift increases the likelihood of a patient being in coma.

b. CT SCAN FEATURES AND PUPILLARY REACTION TO LIGHT

Each of the four CT scan features will again be analysed individually. Overall two thirds of the patients had both pupils reacting to light while in the remaining third either one or neither pupil reacted to light.

i) Basal Cistern Appearance (Table 9)

About half of the patients had obliterated basal cisterns. Less than half (45%) of those with obliterated cistern had both pupils reacting to light while 12% and 40% had either one or neither pupil reacting to light respectively.

When the basal cisterns were not obliterated 87% of patients had both pupils reacting to light compared to only 5% and 6% with either one or neither pupil reacting to light respectively. This obliteration of cisterns was more after related to pupillary dysfunction; bilateral pupillary dysfunction occurred seven times more often, and unilateral pupillary dysfunction twice as often, in patients with obliterated basal cistern as in those in whom the basal cisterns were not obliterated.

Conversely, when both pupils were reacting to light 92 (one third) of the 271 patients had obliterated basal cisterns. But when only one pupil was reacting to light,

two thirds of the 38 patients had obliterated cisterns and nearly 90% (86 of 93) of patients with both pupils not reacting to light had obliterated basal cisterns.
The observed differences are statistically significant (X^2 = 85.95, df = 2, p<0.001).

b) Third Ventricle Appearance (Table 10)

Of the 183 patients in whom the third ventricle was not obliterated 90% had both pupils reacting to light but only 5% of them had one and another 5% had neither pupil reacting to light.

Of the 219 patients who had an obliterated third ventricle, 104 (47%) had both, 29 (13%) had one and 81 (37%) had neither pupil reacting to light. These results are comparable to those observed in relation to basal cistern appearance above. Similarly 38% of patients with both pupils reacting to light had an obliterated third ventricle compared to 76% and 87% with one or neither pupil reacting to light respectively had an obliterated third ventricle.

iii) Contralateral Ventricle Dilatation (Table 11)

There were 208 patients in whom the contralateral was NOT dilated of whom 79% had both pupils reacting to light and of whom 18% had either one (8%) or neither (10%) pupil reacting to light.

The contralateral ventricle was dilated in 197 patients of whom 55% had both pupils reacting to light and 11% and 35% had either one or neither pupil reacting to light respectively.

When both pupils were reacting to light 103 (38%) of the 271 patients had a dilated contralateral ventricle. In the patients with only one pupil reacting to light this phenomenon was seen in 55% of patients and in 75% of those in whom neither pupil reacted to light.

iv) Midline Shift (Table 12, Figs 10 to 12)

The presence or absence of contralateral ventricle dilatation, basal cisterns or third ventricle was interpreted as an **all** or **none** phenomenon. Midline shift, however, is a graded parameter providing a spectrum from no shift to the maximum observed shift.

The results relating the degree of midline shift in millimetres (mm) for each type of pupillary response are shown in Table 12. The frequency of each type of pupillary response for each degree of midline shift was derived from this table and shown in figures 10,11 and 12.

How often patients were found to have both pupils reacting to light decreased steadily from 81% when there was no shift to 16% when there was a 25mm shift (Fig 10). The opposite relationship was found in patients in whom neither pupil reacted to light. The frequency of non-reacting pupils increased from 12% when there was no shift to 46% for a 15mm shift and 71% for a 20mm shift. The numbers were small for greater shifts.

In the patients in whom only one pupil was reacting to light there is a striking lack of pattern comparable to those with both pupils reacting or not reacting to light (Fig 11).

c. INTER-RELATION BETWEEN CT SCAN FEATURES

Midline shift being a quantifiable continuous variable provides a parameter against which the other three CT scan features can be related. This relationship will be examined first by individually analysing the other three CT scan features and then their combination.

1) Indvidual CT scan features (Table 13)

The results of the analysis of the relationship between the individual CT scan features (obliterated basal cisterns or third ventricle and CVD and midline shift in patients with intracranial haematoma show that it is rare to find these abnormal CT scan features in patients with no midline shift. With increasing midline shift it became increasingly commoner to find abnormal CT scan features. This is demonstrated by finding that about a third of patients with a 5mm shift, three quarters of those with a 10mm shift and over 90% of those with a 15mm shift had other abnormal CT scan features.

Another observation is that obstruction of the third ventricle occurs more often at any shift than do obliteration of the basal cisterns or the presence of a dilated contralateral ventricle. This might suggest that obstruction of the third ventricle occurs earlier than either of the other two and that it is almost certain to find all the three occurring together for shifts of greater than 15mm.

ii) Combination of CT scan features (Table 14)

This analysis refers to the 391 patients in whom all the four CT scan features could be determined.

None of the patients who otherwise had other normal CT scan features had a midline shift of greater than 10mm. Although patients who had 1 or 2 other abnormal CT scan features were seen at all ranges of shift, they commonly occurred in those with a 5-10mm shift. When there was no shift there were no patients in whom all the other three CT scan features were abnormal. At least 90% of patients with midline shifts greater than 10mm also had all other CT scan features abnormal.

5.4 DISCUSSION

There is a good correlation between clinical features and the four CT scan features studied. Patients in coma were commonly found to have abnormal CT scan features, in particular, obliterated basal cisterns or third ventricle and a dilated contralateral ventricle. A decreasing level of consciousness was associated with an increasing likelihood of finding these abnormal CT scan features. With regard to the significance of the midline shift, the 10mm mark appears to be a watershed area on either side of which there is a dramatic change in the proportion of patients found to be in coma. This suggests that shifts of greater than 10mm may have the same significance as the other abnormal CT scan findings. About a quarter of the patients who were not in coma were also found to have these abnormal CT scan features. It is conceivable that these

features may represent the upper limit for which the mass effect of a haematoma can be compensated and that small subsequent changes in the mass effect are followed by larger rises in intracranial pressure and hence alteration in consciousness. The finding that decreasing consciousness was associated with increasing likelihood of seeing abnormal CT scan features gives support to the proposition that abnormal CT scan features precede changes in responsiveness. The implication of this may be that in patients found to have these abnormal CT scan features, there is a need to evacuate the haematoma so as to relieve raised ICP or prevent it from rising to dangerous levels. But the first issue would be to confirm that these abnormal CT scan features do indeed indicate raised intracranial pressure.

Abnormal pupillary function was found more commonly when abnormal CT scan features were present. Although there is a clear pattern between the presence of absence of reaction in both pupils with midline shift, that was not the case with unilateral pupillary dysfunction. There is no obvious explanation to this observation but it is possible that if it had been easier to clearly and reliably define unilateral basal cistern obliteration a better correlation might have been seen. Another possible explanation for this observation is that unilateral pupillary dysfunction may have been to be due to solitary lesions of the oculomotor nerve as a primary impact related damage rather than a consequence of a rise in intracranial pressure due to the haematoma.

Bilateral unreactive pupils were seen three times more often than unilateral unreactive pupils in patients with the other three abnormal CT scan features. However, when the basal cistern or third ventricle or lateral ventricle appearances were regarded as normal then only a few patients, about 5% each had either one or neither pupil reacting to light, that is there were no differences unlike those seen when abnormal CT scan features were present.

The occurrence of unreactive pupils in patients with supratentorial masses was first conceived by Meyers in 1920 who considered it to be due to transtentorial temporal lobe herniation. How easily this herniation may occur depends not only to the mass causing the shift but on the size and configuration of the tentorial incisura (Plautt 1963). Unfortunately measurements of the size of the tentorial incisura were not possible with the second generation CT scanner used in this work. Such measurements may have been useful in assisting to explain the observation that unilateral pupillary dysfunction did not correlate clearly with midline shift.

The analysis of the inter-relationships between CT scan features shows that it is common to find abnormal CT scan features occurring together in patients with a major midline shift, (>10mm). It would appear from these results that there could be an hierachy of the features. It would therefore seem that an intracranial supratentorial haematoma initially exerts its effect by displacing the midline.

Once the midline is displaced then the third ventricle and basal cisterns are distorted and this results in the CSF flow being impeded. When the third ventricle is obliterated an internal obstructive hydrocephalus ensues, raising the ICP and obliterating the basal cisterns. By virtue of the compression of the ipsilateral ventricle by the haematoma CSF flows from that ventricle into the contralateral ventricle hence forming a one chamber hydrocephalus. Because the development of these events takes place over hours to a few days before there are changes to compensate for the rising ICP then alteration in consciousness occurs. This contrasts the contralateral ventricle dilatation seen in patients with brain tumours (Findlay and Cummins 1981) in whom the development of headache affords earlier diagnosis before the basal cisterns obliterate or before there is pupillary dysfunction.

The contention at this stage is that the combination of abnormal CT scans does reflect clinically significant raised intracranial pressure in head injured patients with a haematoma. This contention will be examined further in Chapter 7 when intracranial pressure features will be related to CT scan features. How these findings influence management will also be examined further in Chapter 6.

TABLE 5

BASAL CISTERN APPEARANCE
AND
GLASGOW COMA SCORE

Basal Cistern Appearance	GLASGOW COMA SCORE					
	3-5	6-7	8-10	11-15	Unknown	TOTAL
Not obliterated	9	28	35	125	1	198
Obliterated	70	54	26	36	16	202
Unknown	3	3	3	2	0	11
TOTAL	82	85	64	163	17	411

(X^2 = 105.64 df = 3 P<0.001)

TABLE 6

THIRD VENTRICLE APPEARANCE
AND
GLASGOW COMA SCORE

Third Ventricle Appearance	GLASGOW COMA SCORE					
	3-5	6-7	8-10	11-15	UNKNOWN	TOTAL
Not Obliterated	8	25	38	110	2	183
Obliterated	72	57	24	52	14	219
Unknown	2	3	2	1	1	9
TOTAL	82	85	64	163	17	411

(X^2 = 86.46 df = 3 P<0.001)

TABLE 7

CONTRALATERAL VENTRICLE DILATATION (CVD)
AND
GLASGOW COMA SCORE

	GLASGOW COMA SCORE					
CVD	3-5	6-7	8-10	11-15	UNKNOWN	TOTAL
Absent	17	33	41	112	5	208
Present	65	48	21	51	12	197
Unknown	0	4	2	0	0	6
TOTAL	82	85	64	163	17	411

(X^2 = 59.45 df = 3 P<0.001)

TABLE 8

MIDLINE SHIFT
AND
GLASGOW COMA SCORE

Midline Shift mm	Glasgow Coma Score Group 3-5	6-7	8-10	11-15	Unknown	TOTAL
0mm	5	19	29	73	2	128
5mm	9	13	17	47	2	88
10mm	19	20	10	27	5	81
15mm	24	20	8	9	5	66
20mm	22	8	0	6	2	38
25mm	2	2	0	1	1	6
30mm	1	0	0	0	0	1
Unknown	0	3	0	0	0	4
TOTAL	82	85	64	163	17	411

TABLE 9

BASAL CISTERN APPEARANCE
AND
PUPILLARY REACTION TO LIGHT

Basal Cistern Appearance	Pupillary response to Light: Both react	One reacts	Neither react	Unknown	Total
Not Obliterated	173	12	9	4	198
Obliterated	92	25	80	5	202
Unknown	6	1	4	0	11
	271	38	93	9	411

(X^2 = 85.95, df = 2 P <0.001)

TABLE 10

THIRD VENTRICLE APPEARANCE
AND
PUPILLARY REACTION TO LIGHT

Third Ventricle Appearance	Both react	One reacts	Neither react	Unknown	TOTAL
Not Obliterated	162	9	9	4	183
Obliterated	107	29	81	5	219
Unknown	6	0	3	0	9
TOTAL	271	38	93	9	411

(X^2 = 76.59, df = 2, p<0.001)

TABLE 11

CONTRALATERAL VENTRICLE DILATATION
AND
PUPILLARY REACTION TO LIGHT

Contralateral Ventricle Dilatation	Both react	One reacts	Neither react	Unknown	TOTAL
Absent	165	17	20	6	208
Present	103	21	70	3	197
Unknown	3	0	3	0	6
TOTAL	271	38	93	9	411

(X^2 = 42.40, df = 2, P<0.001)

TABLE 12

MIDLINE SHIFT
AND
PUPILLARY REACTION TO LIGHT

Midline shift Milimetres (mm)	BOTH react	ONE reacts	NEITHER react	Unknown	Total
0mm	111	6	7	4	128
5mm	68	5	13	2	88
10mm	53	12	16	0	81
15mm	25	13	25	3	66
20mm	10	1	27	0	38
25mm	1	1	4	0	6
30mm	0	0	1	0	1
Unknown	3	0	1	0	4
TOTAL	271	38	93	9	411

TABLE 13

INTER-RELATION BETWEEN INDIVIDUAL CT SCAN FEATURES

CT Scan Features	Midline Shift (mm)					
	0mm	5mm	10mm	15mm	20mm	TOTAL
Basal Cistern Appearance						
Obliterated	7	27	61	59	45	199
Not obliterated	119	56	18	5	0	198
TOTAL	126	83	79	64	45	397
% Obliterated	6%	33%	77%	92%	100%	(50%)
Third Ventricle Appearance						
Obliterated	7	35	68	63	44	217
Not obliterated	121	49	11	2	0	183
TOTAL	128	84	79	65	44	400
% Obliterated	5%	42%	86%	97%	100%	(54%)
Contralateral Ventricle Dilatation						
Present	3	26	61	64	41	194
Absent	124	60	19	2	4	209
TOTAL	127	86	80	66	45	404
% with CVD	2%	30%	76%	97%	93%	(48%)

TABLE 14

INTER-RELATION BETWEEN COMBINATION OF CT SCAN FEATURES

BC*	V3*	CVD*	Midline Shift in millimetres 0mm	5mm	10mm	15mm	20+mm	TOTAL
–	–	–	115	36	4	0	0	155
+	–	–	3	1	2	0	0	6
–	+	–	2	5	1	0	0	8
–	–	+	2	10	4	2	0	28
+	+	–	3	14	11	2	3	33
–	+	+	0	4	9	2	0	15
+	–	+	1	1	1	0	0	3
+	+	+	0	10	45	57	41	153
			126	81	77	63	44	391
% All normal			91%	44%	5%	0%	0%	
% 1 or 2 abnormal			9%	43%	36%	10%	7%	
% all abnormal			0%	13%	59%	90%	93%	
			100%	100%	100%	100%	100%	

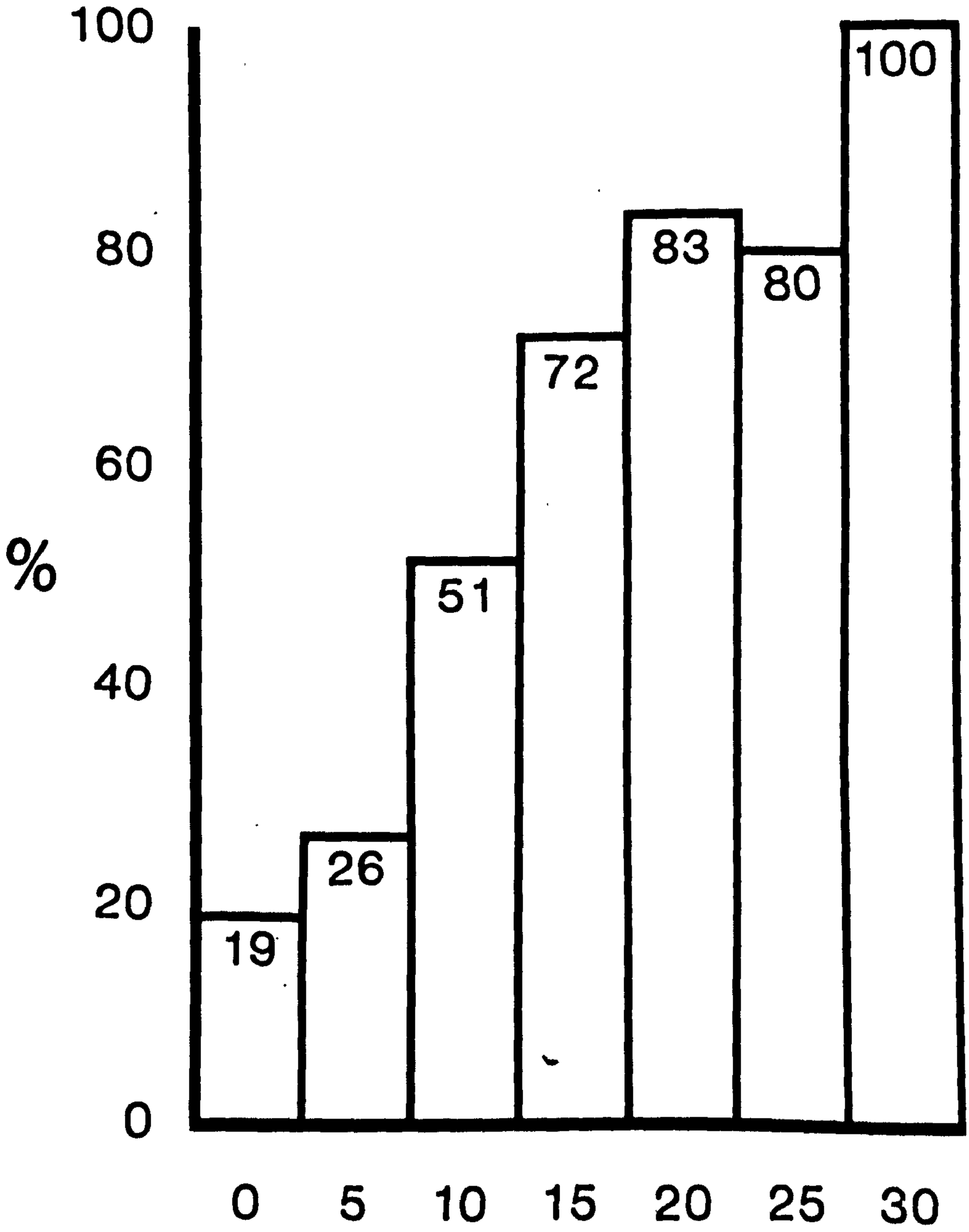
* \+ abnormal CT scan feature (obliterated basal cisterns or third ventricle or CVD)

– normal CT scan feature (basal cisterns or third not obliterated, no CVD)

FIGURE 9

Relationship between coma (GCS <7) and the midline shift.

Coma and Midline Shift



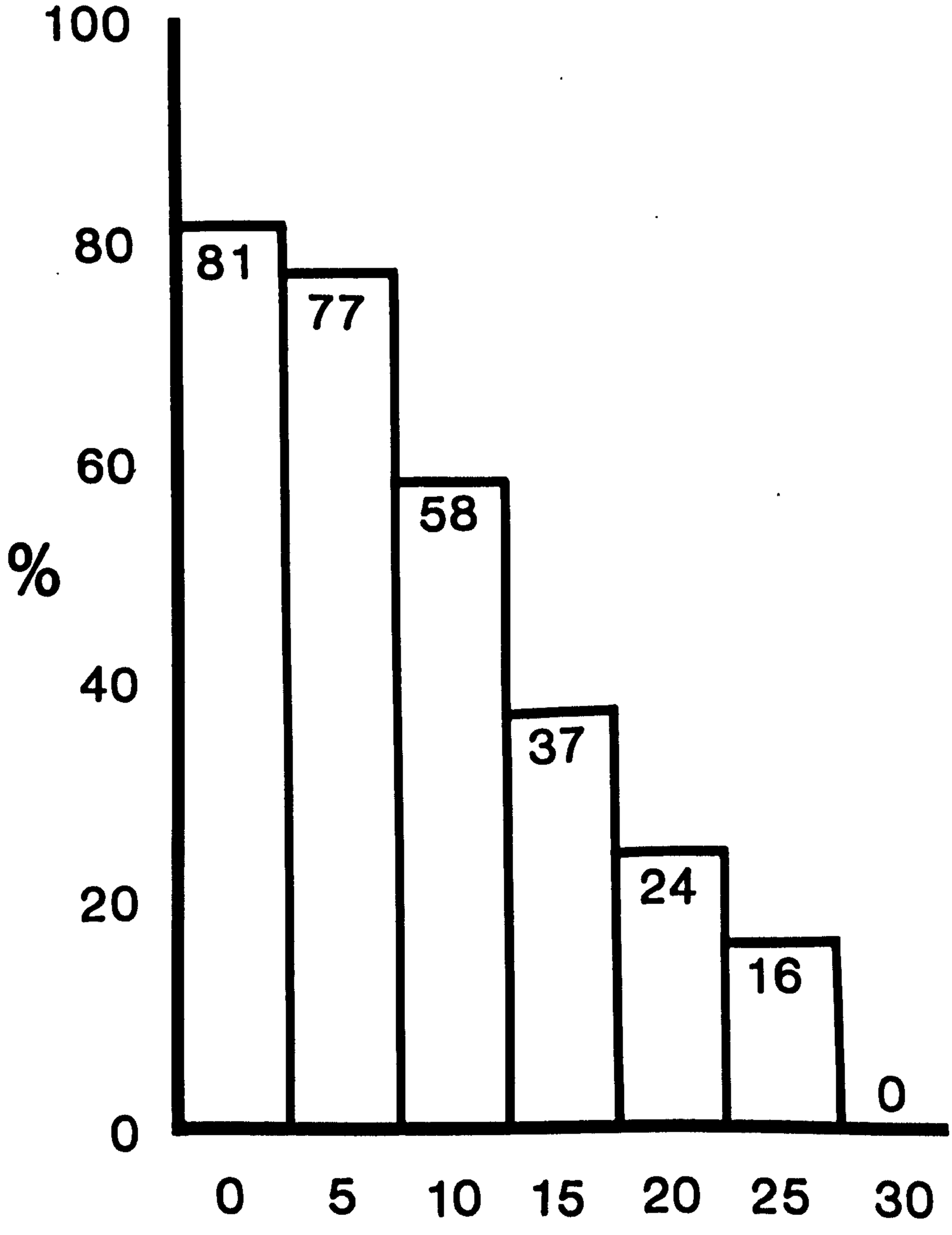
Relationship between coma (GCS≤7) and midline shift in millimetres (mm).

Fig. 9

FIGURE 10

Relationship between midline shift and proportion of patients with both pupils reacting to light

Pupillary response to light and midline Shift

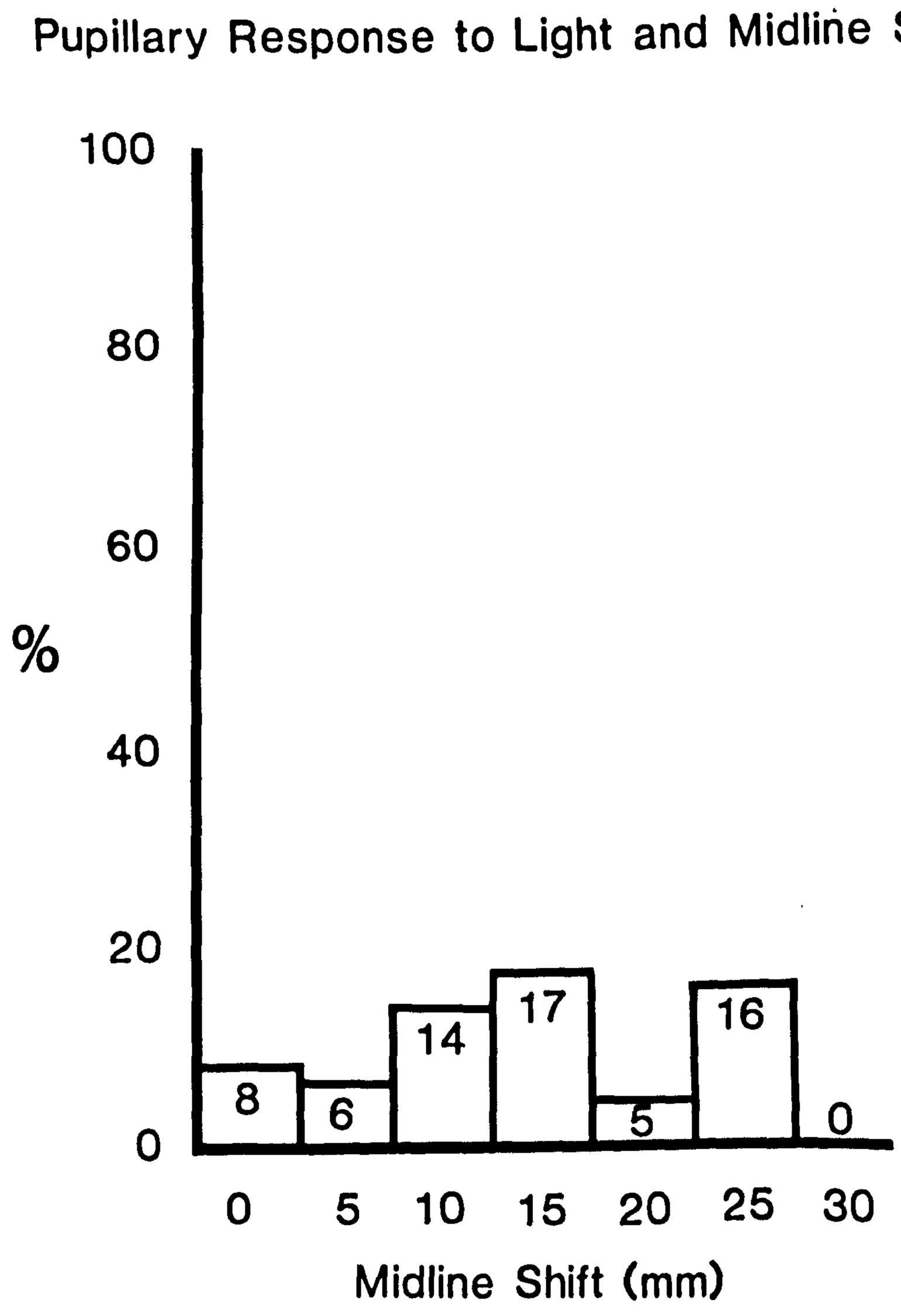


Percentage of patients with both pupils reacting to light in relation to midline shift in millimetres (mm).

Fig. 10

FIGURE 11·

Relationship between midline shift and proportion of patients with one pupil reacting to light



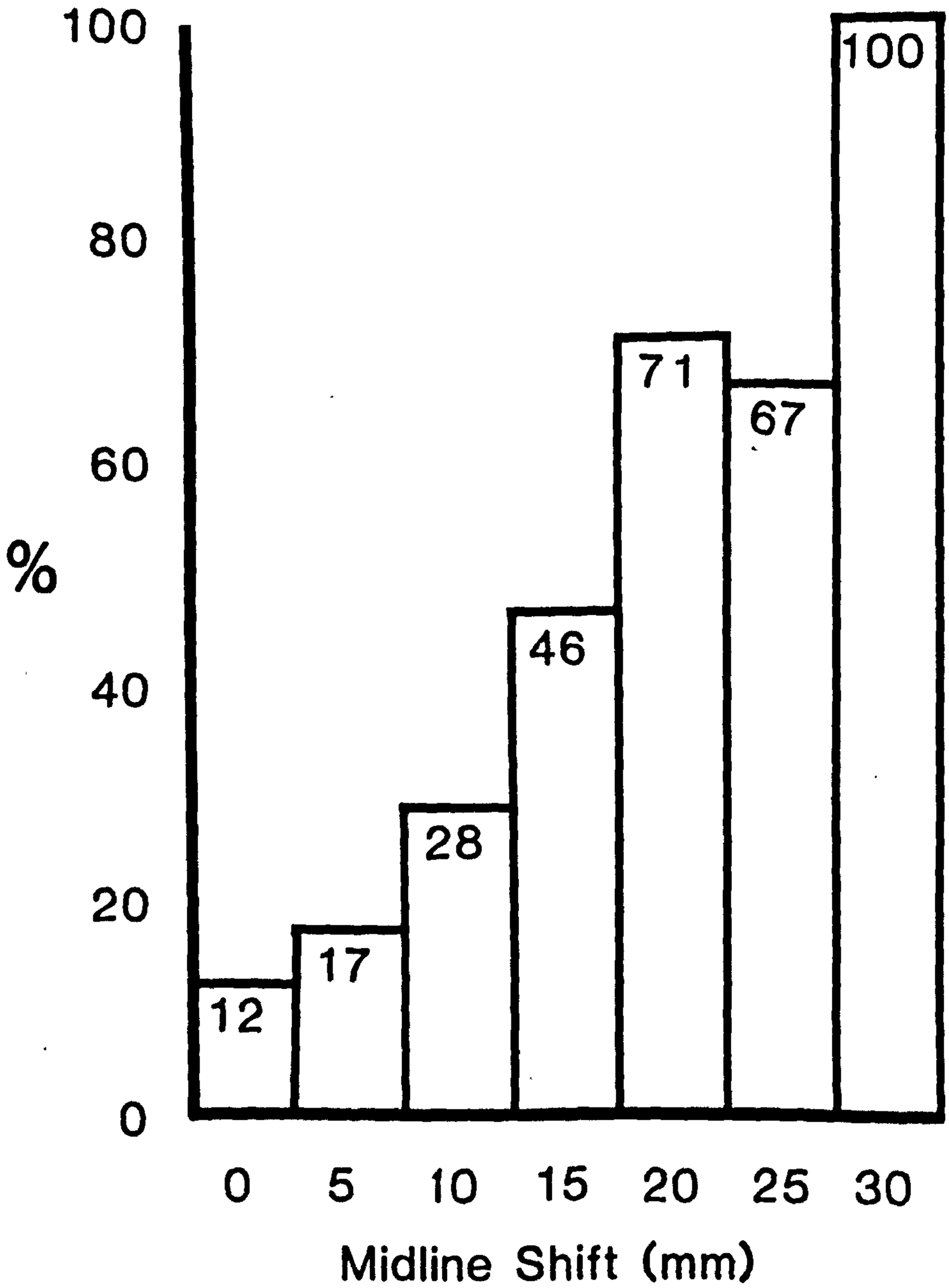
Percentage of patients with one pupil reacting to light in relation to midline shift in millimetres (mm).

Fig. 11

FIGURE 12

Relationship between midline shift and proportion of patients with neither pupil reacting to light

Pupillary Response to Light and Midline Shift



Percentage of Patients with both pupils <u>not</u> reacting to light in relation to midline shift in millimetres (mm).

Fig. 12

CHAPTER 6

CT SCAN FINDINGS AND THE MANAGEMENT OF ACUTE TRAUMATIC INTRADURAL HAEMATOMA

6.1 INTRODUCTION

The observations made in chapter 4 indicated that the initial decision was that following CT scanning half the patients with intradural haematoma did not clearly need operation forthwith. Most of these patients were observed clinically while some had intracranial pressure monitoring. It is a question of considerable practical importance how often clinicians are unable to make up decisions based on initial CT scan. It was shown in Chapter 4 that 25% of the patients with intradural haematoma who were operated upon had that decision delayed because of initial uncertainty of the need for surgery. What CT scan features did those needing immediate surgery have and in what way did they differ from those in whom that decision was uncertain?

In this chapter the CT scan features will be analysed in order to compare the findings in patients considered to need immediate surgery with those in patients who did not have immediate surgery.

6.2 PATIENTS AND METHODS

Patients with intradural haematoma were identified and the type of lesion classified as described in Chapter 3. The CT scan features were those available to the clinicans at the time of the initial decision. The details of interpretation of the CT scan features have also been explained in Chapter 3.

Patients with a pure extradural haematoma have been excluded from this part of the study because the initial decision in these patients was uniformly to proceed to surgery.

6.3 RESULTS

The important issues are: first, to identify the CT findings more prevalent in patients operated early as opposed to those not operated upon early after initial management decisions; and second to find out how the CT scan findings might have affected the final management decision. For these reasons therefore the results will be analysed first to determine the differences in CT scan findings between patients with immediate operation and those who did not have immediate operation. It will be later seen that those who did not have immediate operation were managed by either intracranial pressure monitoring or clinical observations alone. Since the two groups were not randomly subdivided at the initial decision they will be treated together as a group of patients who did not have immediate surgery. Thereafter, in order to evaluate the relative merits of the different CT scan findings in the eventual management decision, each management group will be analysed separately.

a. CT SCAN FINDINGS AND INITIAL MANAGEMENT

a) Type of intradural haematoma (Table 15)

Patients with subdural haematoma were more often operated upon immedately than those with intracerebral haematoma. Of those with subdural haematoma alone 62% were operated upon immediately as were 64% of those with both subdural and intracerebral haematoma but only 34% of those with an intracerebral haematoma. This suggests that the type of lesion has an influence on the decision about immediate surgery.

ii) CT scan features (Tables 16 to 19)

In the patients operated upon immediately, abnormal CT scan features were more frequent than in those not operated upon early. Sixty-nine percent had obstructed basal cisterns or third ventricle and 65% had Contralateral Ventricle Dilatation although only 41% of these patients had a midline shift exceeding 10mm. Of these not operated upon early, normal CT scan features were more frequently observed. Thus 77% had normal basal cisterns, 74% had normal third ventricle appearance as were 79% did not have CVD and 89% a midline shift less than 10 millimetres. The extremes of the CT scan features correlate with the initial management decision.

b. CT SCAN FINDINGS AND FINAL MANAGEMENT

ia) Type of Intradural Haematoma (Table 20)

There were 58 patients who were operated upon after a delay: 50% after clinical observations alone and 50% after intracranial pressure monitoring. Sixteen percent of patients with subdural haematoma had a delayed operation as did 16% of those with both subdural and intracerebral haematoma and 19% of these with intracerebral haematoma only. The type of lesion therefore did not influence the final decision to operate. But in the patients who were never operated upon the differences in the types of lesion can be seen. Of those with subdural haematoma only 20% were never operated and this did not matter whether it occurred alone or in combination with an intracerebral haematoma. Nearly a half (46%) of those with intracerebral haematoma were never operated upon.

ii) CT scan features (Table 21 to 24)

Seventy two percent of those who had delayed surgery after clinical observation did not have obliterated basal cisterns, so too did 62% of those operated after intracranial pressure monitorinng. These findings were the same when the third ventricle was not obliterated. Thus a third of the patients who were eventually operated upon had an obliterated basal cisterns or third ventricle at the initial CT. There were proportionately fewer operated patients with CVD but the trend is similar (Table 23). Around 90% of patients not operated upon had normal ventricle appearances, i.e. no CVD. This was also shown in relation to midline shift (Table 24) in which 91% of patients not operated after clinical observations and 97% of patients no t operated after intracranial pressure monitoring had midline shift of less than 10 millimetres.

6.3 DISCUSSION

It was more often decided that there was need for immediate surgery in patients with subdural haematoma, alone or in combination with intracerebral haematoma, than in patients found to have intracerebral haematoma alone. This may imply that the presence of a subdural haematoma is likely to cause a rise in ICP than the presence of an intracerebral haematoma or that operation is easier, and more successful.

In the final management it did not matter whether the delayed need for surgery was based on clinical observations alone or by intracranial pressure monitoring. The need for delayed surgery also did not differ in the different types

of intradural lesions. But the decision not to operate resulted in about half of those with intracerebral haematoma and about a fifth of those with subdural haematoma, alone or in combination with an intracerebral haematoma, being managed nonsurgically. The less need to operate on intracerebral haematoma may in part be that they have been known to resolve in patients observed for long periods (Suzuki and Takar 1970, McLaurin et al 1970, McKissock et al 1961, Dolinskas et al 1977, Messina and Chernick 1975). But there is always the danger that these patients can deteriorate precipitously (Gudeman et al 1979). This fear has been expressed by others who advocated for a more aggressive approach to these lesions (Becker et al 1977, Diaz et al 1979) and Seeling et al 1981). That subdural haematoma tend to persist in a chronic form (Forbes et al 1978), Scott et al 1977) may also contribute to the differences in the observed operative approach to these lesions.

The pattern of CT scan findings shows a distinction between those considered to need immediate operation and those in whom this was delayed or not done. The majority of patients who had immediate operation had clearly abnormal CT scan features as was described in Chapter 5. The patients in whom surgery was delayed more often did not have abnormal CT scan at the time of the initial decision. Unfortunately repeat CT scans were not usually done at the time of the final management decision so that it is not possible to find out from these patients whether the pattern of CT scan features had changed. My speculation at this stage is that because the CT scan features studied are dynamic then it is quite likely that in

the course of clinical observations or intracranial pressure monitoring these patients might have developed the abnormal CT features seen in the patients operated upon early. This also applied to the majority of patients who were never operated upon. Some of the patients who were not operated upon had clearly abnormal CT scan features. This was because the initial clinical decision was not to operate on the basis of a predictable poor prognosis. They were however, only 3% of the study population and these were in the group observed clinically. In the patients who had ICP monitoring but were never operated upon it was rare to find an abnormal other CT scan feature. Since the final decision to operate upon the patients ICP monitored rested on the level of ICP then it can therefore be said that it is unusual for patients with normal other CT scan features to have clinically significant raised intracranial pressure. This will be examined in more detail in Chapter 7.

TABLE 15

TYPE OF INTRADURAL HAEMATOMA
AND
INITIAL MANAGEMENT

Type of Haematoma	Initial Management: Immediate Operation	No immediate Operation	Unknown	TOTAL
Subdural Haematoma only	86	48	5	139
Intracerebral Haematoma only	47	91	1	139
Both Subdural & Intracranial Haematoma	36	20	0	56
Extradural & Intradural Haematoma	10	0	1	11
	179	159	7	345

TABLE 16

BASAL CISTERN APPEARANCE
AND
INITIAL MANAGEMENT

Basal Cisterns	Immediate Operation	No immediate Operation	Unknown	TOTAL
Not obliterated	49	123	3	175
Obliterated	124	34	4	162
Unknown	6	2	0	8
TOTAL	179	159	7	345

TABLE 17

THIRD VENTRICLE APPEARANCE
AND
INITIAL MANAGEMENT

Third Ventricle	Immediate operation	No immediate operation	Unknown	TOTAL
Not obliterated	44	118	3	165
Obliterated	129	40	4	173
Unknown	6	1	0	7
TOTAL	179	159	7	345

TABLE 18

CONTRALATERAL VENTRICLE DILATATION
AND
INITIAL MANAGEMENT

CVD	Immediate Operation	No immediate Operation	Unknown	TOTAL
Absent	58	125	4	187
Present	117	34	3	154
Unknown	4	0	0	4
TOTAL	179	159	7	345

TABLE 19

MIDLINE SHIFT
AND
INITIAL MANAGEMENT

Midline Shift (mm)	Immediate Operation	No immediate Operation	Unknown	TOTAL
0mm	23	93	2	118
5mm	41	33	4	78
10mm	42	15	0	57
15mm	40	12	0	52
20mm	27	5	1	33
25mm	4	1	0	5
30mm	1	0	0	1
Unknown	1	0	0	1
TOTAL	179	159	7	345

TABLE 20

TYPE OF INTRADURAL HAEMATOMA
AND FINAL MANAGEMENT

	OPERATIVE				NON OPERATIVE		
Type of Haematoma	Immediate Operation	Delayed Operation Clinical	ICPM	Unknown	Clinical	ICPM	
Subdural haematoma only	86	12	10	5	21	5	139
Intracerbral haematoma only	47	15	12	1	39	25	139
Both sudural and intracerebral haematoma	36	2	7	0	7	0	56
Extradural and intradural haematoma	10	0	0	1	0	0	11
	179	29	29	7	67	34	345

TABLE 21

BASAL CISTERN APPEARANCE
AND
FINAL MANAGEMENT

Basal Cisterns	Final Management: Operative				Final Management: Non-Operative		
	Immediate	Delayed Clinical	Delayed ICPM	Unknown	Clinical	ICPM	TOTAL
Not obliterated	49	21	18	3	54	30	175
Obliterated	124	8	11	4	12	3	162
Unknown	6	0	0	0	1	1	8
	179	29	29	7	67	34	345

TABLE 22

THIRD VENTRICLE APPEARANCE
AND
FINAL MANAGEMENT

	Operative				Non-Operative	
		Delayed				
Third ventricle	Immediate	Clinical	ICPM	Unknown	Clinical	ICPM
Not obliterated	44	18	17	3	54	29
Obliterated	129	11	12	4	13	4
Unknown	6	0	0	0	0	1
	179	29	29	7	67	34

TABLE 23

CONTRALATERAL VENTRICLE DILATATION
AND
FINAL MANAGEMENT

Type of Final Management

	OPERATIVE				NON-OPERATIVE		
CVD	Immediate	Delayed Clinical	Delayed ICPM	Unknown	Clinical	ICPM	TOTAL
Absent	58	17	18	4	58	32	187
Present	117	12	11	3	9	2	154
Unknown	4	0	0	0	0	0	4
TOTAL	179	29	29	7	67	34	345

TABLE 24

MIDLINE SHIFT
AND
FINAL MANAGEMENT

Midline Shift(mm)	OPERATIVE				NON-OPERATIVE		
	Immediate	Delayed Clinical	Delayed ICPM	Unknown	Clinical	ICPM	TOTAL
0mm	23	11	12	2	48	22	118
5mm	41	6	7	4	10	10	78
10mm	42	3	8	0	3	1	57
15mm	40	9	1	0	2	0	52
20mm	27	0	1	1	3	1	33
25mm	4	0	0	0	1	0	5
30mm	1	0	0	0	0	0	1
Unknown	1	0	0	0	0	0	1
TOTAL	179	29	29	7	67	34	345

CHAPTER 7

INTRACRANCIAL PRESSURE MONITORING IN THE MANAGEMENT OF ACUTE INTRADURAL HAEMATOMA

7.1 INTRODUCTION

There is controversy about whether all acute intradural haematomas should be treated in the same way or whether a selected policy is more appropriate. Some advocate early and aggressive surgery on all patients found to be in coma (Becker et al 1977). By contrast, others believe that a patient who is clinically stable needs operation only if neurological deterioration occurs (Wiegel et al 1978). There is no doubt that some of the patients observed clinically do recover spontaneously hence obviating surgery (Dolinskas et al 1977). But it is also widely known that deterioration frequently occurs and that when it occurs the results of remedial surgery are disappointing (Gudeman et al 1979), Diaz et al 1979). The ideal approach would be to identify the factors that predict which patients are likely to deteriorate and therefore be operated upon before neurological deterioration occurs.

The suggestion made by Galbraith and Teasdale (1981) appears to offer a more rational approach to the management of patients with clinically 'silent' acute traumatic intradural haematoma. They studied a small selected group of patients and measured the level of their intracranial pressure whilst continuing to monitor them clinically. The decision to operate has been based on the occurrence of neurological deterioration and their experience showed that patients with ICP >30mmHg invariable deteriorated and that only half of those with ICP range 20-30mmHg did eventually require surgery. The results of their study showed that unfavourable outcome occurred twice as often in the

operated patients as in those who did not need to be operated upon. Their conclusion was that patients with ICP >30mmHg should be operated upon before they deteriorated. These findings raise a number of important questions. Do patients in need of surgery have clinical and CT scan features that would make it possible to predict that they were going to deteriorate? Are there clinical and CT scan features that would enable to identify the patients in whom the ICP would be >30mmHg and therefore consistently determine that neurological deterioration will take place.

The results in chapter 5 showed that patients who had impaired consciousness more often had abnormal CT scan findings. Subsequently in chapter 6 it was confirmed that in patients who were operated upon more often also had abnormal CT scan findings. How common then are abnormal CT scan features in patients in whom the intracranial pressure is found to be more than 30mmHg. Another question of considerable clinical implication is if a decision to operate upon patients based on the suggested level of ICP improve the results or are there no appreciable differences?

In this chapter the results will be analysed in order to find out how often intracranial pressure monitoring was used and when it was used how often it led to a decision to operate. The relationship between the findings of monitoring by ICP and the clinical and CT scan features will be examined and how useful intracranial pressure monitoring was in picking out the patients in need of surgery will be determined. The impact on results of the

policy to operate based on the suggested level of ICP will be examined further in Chaper 8 when the factors contributing to outcome will be analysed.

7.2 PATIENTS AND METHODS

The patients in the study were those seen during the three year period following the pilot study by Galbraith and Teasdale. The details of the methods used in relation to intracranial pressure monitoring have already been described in Chapter 3.

7.3 RESULTS

a. Initial Clinical Features and Management

i) Age (Table 25)

The distribution of patients in the different management groups shows that there are no significant differences in age groups.

ii) Initial Glasgow Coma Score (Table 26)

About a quarter of patients who were operated upon immediately were in the highest coma score group. The reason for operation despite their good clinical state was that the neurosurgeons in attendance considered the CT scan lesion to be too 'large' for the patient to be safely observed. The decision in favour of immediate surgery was thus a combination of the level of consciousness and the 'size' of the haematoma.

The results show ICP monitoring was used in patients with a wide range of coma scores. However only two of those so monitored were in the lowest coma score group. The reason was it was uncertain whether the size of the lesion alone was the

cause of the impaired consciousness. The majority of patients were in the higher coma score groups. Of the 13 patients in coma, 5 (38%) were eventually operated upon due to their intracranial pressure characteristics. Half of the patients in the GCS 8 - 10 and also half of those in the GCS 11-15 were operated upon after intracranial pressure monitoring. The initial Glasgow Coma Score alone then could not have predicted which patients would eventually be operated upon after measuring their ICP.

In the group of patients who were clinically observed there were 12 patients in the lowest coma score group. They were not operated upon immediately because their prognosis was considered poor. After a period of observation three of the patients were operated upon. This appeared to be because it was considered that they had improved sufficiently to make operation worthwhile. About three quarters of patients in this group were not in coma. Eventually a third of all the patients observed clinically were operated upon.

iii) Initial and Final Preoperative GCS in Monitored Patients

There were patients who deteriorated in their coma score before operation in those clinically monitored (Table 27) and those ICP monitored (Table 28). In the clinically monitored group there were 6 (29%) of the 29 patients who deteriorated from their initial GCS 11-15 group: two to the GCS group 8-10, and the other 4 patients to GCS <7. The patients who initially were in the GCS group 6-7 were static while two of these in the lowest GCS group had in

fact improved, a factor that prompted their being operated upon. In the ICP monitored patients (Table 28) only 2 (13%) of the 15 patients in the highest coma score group had deteriorated as were three of those in the GCS 8-10 group. Of the 5 in the initial GCS 6-7 group, 2 had improved, two were static and one had deteriorated to a worse group.

b. INTRACRANIAL PRESSURE MONITORING & FINAL MANAGEMENT

a) Duration of ICP Monitoring (Table 29)

A decision to evacuate a haematoma had been reached within 24 hours in 14 (48%) of the patients who required surgery. Another 13 (45%) needed up to 72 hours of monitoring before confirming that operation was required. A decision in favour of surgery had therefore been reached witthin 72 hrs in 93% of patients eventually operated upon. Monitoring beyond 72 hours was done in one patient whose level of ICP had initially remained borderline.

One patient (a failed case) had a borderline ICP but was too restless and actually removed his ventricular catheter before a stable and adequate recording of ICP could be reached. A decision in favour of operation was made instead of inserting a new ventricular catheter.

In the patients who were not operated upon, the decision to stop ICP monitoring had been reached within 24 hours in a third of them. Twenty one patients (62%) of those not operated upon after ICP monitoring were so monitored for 24 - 72 hours. No patients who did not require surgery were

monitored for more than 72 hours. However, two drunken patients were too restless to allow for a safe and meaningful recording. No recording of ICP level was made and there were considered as failed patients. They continued to be monitorded clinically and were not operated upon.

ii) Level of Intracranial Pressure (Table 30)

The highest mean intracranial pressure recorded was less than 20mmHg in 31 (49%) of the patients ICP monitored. Two (6%) of these patients were operated upon. In the first patient there had been deterioration in the coma score after a series of seizures and the patient did not improve in coma score despite adequate respiratory support. In the second patient there was sudden deterioration in the level of consciousness 3 days after ICP monitoring had been discontinued.

Nine patients (15%) had intracranial pressure in the range of 20-30mmHg. Their level of consciousness was closely monitored in the Intensive Care Unit. Three patients were thought not to have improved substantially in their overall level of responsiveness and were therefore operated upon. Two other patients had slight deterioration and also operated upon.

Another patient had sudden deterioration on the sixth day after ICP monitoring had been discontinued and was also operated upon. Thus 6 (67%) of the 9 patients with ICP 20-30mmHg were eventually operated upon.

All the 20 (32%) patients in whom ICP was greater than 30mmHg were operated upon electively when that level of ICP

persisted for more than 15 minutes continuously. Three of these patients deteriorated into coma before they underwent surgery (Table 28). Of those who had deteriorated into the Glasgow Coma Score 3-5, one patient had previously been in the GCS 11-15 while two patients had been in the GCS 8-10. The patients in the GCS group 6-7 were unchanged.

C. HIGHEST LEVEL OF INTRACRANIAL PRESSURE & INITIAL CT SCAN FEATURES (TABLES 31 & 34)

Only three (10%)of the 31 patients with ICP less than 20mmHg had abnormal CT scan features: two with midline shifts of 10mm and 20mm and obliterated basal cisterns while three had an obliterated third ventricle and a dilated contralateral ventricle. Two of these patients were operated upon.

Of the nine patients with ICP range 20-30mmHg, 8 (90%) did not have obliteration of their basal cisterns or third ventricle. None of them had midline shift >5mm or a dilated contralateral ventricle on the initial CT scan.

In the group of 20 patients whose highest ICP exceeded 30mmHg about half of them had either obliteration of basal cisterns or third ventricle or had a dilated contralateral ventricle or a midline shift of at least 10 millimetres.

DISCUSSION

The initial clinical features in the patients who did not have immediate operation did not usefully discriminate between those who eventually required surgery and those who did not need surgery. This may be due to the initial expectation that these patients could be managed without operation. In some of the patients clinically monitored the CT scan lesion had been initially considered not to be significant enough to account for the observed alteration in responsiveness but failure to improve led the clinicians to conclude that the lesion could be important and these were therefore operated upon. The outcome of these patients will be examined in the next chapter.

Intracranial pressure monitoring in this series was used only in one in five patients although it eventually led to a decision to operate in half of those who had delayed operation. In the patients operated upon that decision had been reached in half of them within 24 hours and in over 90% within 72 hours. This time scale indicates that patients do not have to occupy intensive care beds for prolonged periods before a final management decision can be made. This was equally timed in those not operated upon. Another advantage is that by being able to decide within a three day duration then the risk of infection is also reduced.

The decision to operate when the level of ICP was >30mmHg resulted in only a third of such patients deteriorating, compared with all patients in study of Galbraith and

Teasdale. It is to prevent deterioration before surgery that is the aim of this management policy. This emphasises the observation that neurological deterioration is expected to occur when ICP exceeds 30mmHg. The implication of this is that if deterioration is to be avoided before surgery is undertaken then the need for surgery has to be determined at a lower level of intracranial pressure. Patients with ICP in the 20-30mmHg range still pose a problem. The need for surgery in this group of patients was prompted by their deterioration. In the previous series by Galbraith and Teasdale half of their patients in this ICP group did deteriorate before surgery was deemed necessary. In this series two thirds of these patients were operated upon for the same reason as in the previous series. The initial clinical features and CT scan features could not have assisted in determining that these patients were going to deteriorate. As a repeat CT scan at the time of deterioration was not routinely done and it remains to be if they had developed abnormal CT scan features at the time of their deterioration. It would seem to be an advantage to operate early in these patients as at least half of them were likely to deteriorate. Since only a handful of those with ICP less than 20mmHg ever deteriorated in this and the previous series it seems reasonable to decide against early operation in patients in this ICP group.

In examining the relationship between the highest level of ICP and the initial CT scan features, some important observations can be made. Abnormal CT scan features (obliterated basal cistern or third ventricle, dilated

contralateral ventricle) were seen in half of those patients with ICP >30mmHg while also a half had an initial midline shift of at least 10 millimetres. In the patients with ICP of 20-30mmHg it was unusual for the patients to have abnormal CT scan features. This was equally true in the patients with ICP <20mmHg. This suggests that raised intracranial pressure would be expected to be found in patients with abnormal CT scan features. The significance of these findings is that it would be better to operate immediately in patients found to have abnormal CT scans than measure their ICP or observe them and wait for their inevitable deterioration.

TABLE 25

AGE AND TYPE OF MANAGEMENT

	OPERATIVE				NON-OPERATIVE		
		Delayed					
Age (yrs)	Immediate	ICPM	Clinical	Unknown	ICPM	Clinical	
0-9	6	0	1	0	1	2	10
10-19	10	0	4	1	2	14	31
20-29	17	4	2	2	4	3	32
30-39	22	2	3	0	9	5	41
40-49	34	10	3	1	3	13	64
50-59	41	5	7	1	6	7	67
60-69	29	7	7	2	5	9	59
70 +	19	1	2	0	4	14	40
Unknown	1	0	0	0	0	0	1
	179	29	29	7	34	67	345

TABLE 26

INITIAL GLASGOW COMA SCORE

AND

TYPE OF MANAGEMENT

	OPERATIVE				NON OPERATIVE		
		Delayed					
Initial GCS	Immediate	ICPM	Clinical	Unknown	ICPM	Clinical	
3-5	47	0	3	0	2	9	61
6-7	45	5	2	2	6	10	70
8-10	29	8	3	1	8	10	59
11-15	48	15	21	3	18	37	142
Unknown	10	1	0	1	0	1	13
	179	29	29	7	34	67	345

TABLE 27

GLASGOW COMA SCORE (GCS) BEFORE CT SCAN AND IMMEDIATELY PRECEEDING OPERATIONN IN CLINICALLY MONITORED PATIENTS

GCS before CT Scan	Final GCS before operation 3 - 5	6 - 7	8 - 10	11- 15	
3-5	1	2	0	0	3
6-7	0	2	0	0	2
8-10	0	1	2	0	3
11-15	1	3	2	15	21
	2	8	4	15	29

TABLE 28

GLASGOW COMA SCORE (GCS) BEFORE CT SCAN

AND IMMEDIATELY PRECEEDING OPERATION

IN INTRACRANIAL PRESSURE MONITORED PATIENTS

	Final GCS before operation				
GCS before CT scan	3-5	6-7	8-10	11-15	Unknown
3-5	0	0	0	0	0
6-7	1	2	2	0	5
8-10	1	2	4	1	0 8
11-15	1	1	0	13	0 15
Unknown	0	0	0	0	1 1
	3	5	6	14	1 29

TABLE 29

DURATION OF INTRACRRANIAL PRESSURE MONITORING

AND

FINAL MANAGEMENT

Duration	Final Management: Operative	Non-Operative	Total
< 6 hrs	2	3	5
6-12 hrs	4	0	4
12-24 hrs	8	8	16
24-72 hrs	13	21	34
> 72 hrs	1	0	1
Failed	1	2	3
	29	34	63

TABLE 30

LEVEL OF INTRACRANIAL PRESSURE

AND

FINAL MANAGEMENT

	Final Management		
ICP	Operative	Non-Operative	Total
< 20mmHg	2	29	31
20-30mmHg	6	3	9
> 30mmHg	20	0	20
Failed	1	2	3
	29	34	63

TABLE 31

INITIAL BASAL CISTERN APPEARANCE

AND

FINAL INTRACRANIAL PRESSURE

	Intracranial Pressure						
Basal Cistern Appearance	<20mmHg NON OP	<20mmHg OP	20-30 mmHg OP	20-30 mmHg NON OP	>30mmHg	Unknown	TOTAL
Not obliterated	27	1	6	2	10	2	48
Obliterated	1	1	0	1	10	1	14
Unknown	1	0	0	0	0	0	1
TOTAL	29	2	6	3	20	3	63

OP = Operative
NON OP = Non Operative

TABLE 32

INITIAL THIRD VENTRICLE APPEARANCE

AND

FINAL INTRACRANIAL PRESSURE

	INTRACRANIAL PRESSURE						
Third Ventricle Appearance	<20mmHg NON OP	<20mmHg OP	20-30mmHg OP	20-30mmHg NON OP	30mmHg	Unknown	TOTAL
Not obliterated	26	1	6	2	11	2	48
Obliterated	2	1	0	1	9	1	14
Unknown	1	0	0	0	0	0	1
TOTAL	29	2	6	3	20	3	63

OP = Operative
NON OP = Non Operative

TABLE 33

CONTRALATERAL VENTRICLE DILATATION IN INITIAL CT SCAN

AND

FINAL INTRACRANIAL PRESSURE

	INTRACRANIAL PRESSURE						
Contralateral Ventricle Dilatation	<20mmHg NON OP	OP	20-30mmHg OP	NON OP	>30mmHg	Unknown	TOTAL
Absent	27	1	6	3	11	2	50
Present	2	1	0	0	9	1	13
Unknown	0	0	0	0	0	0	0
TOTAL	29	2	6	3	20	3	63

TABLE 34

INITIAL MIDLINE SHIFT

AND

FINAL INTRACRANIAL PRESSURE

Midline Shift	<20mmHg NONOP	<20mmHg OP	20-30mmHg OP	20-30mmHg NON OP	>30mmHg	Unknown	TOTAL
0mm	19	1	4	2	7	1	34
5mm	9	0	2	1	5	0	17
10mm	0	1	0	0	6	2	9
15mm	0	0	0	0	1	0	1
20mm	1	0	0	0	1	0	2
Unknown	0	0	0	0	0	0	0
TOTAL	29	2	6	3	20	3	63

CHAPTER 8

O U T C O M E

8.1 INTRODUCTION

Many factors are known to affect outcome and some of these have already been reviewed in Chapter 2. The overall results of the entire study population were presented in Chapter 4. The outcome of patients with intradural haematoma is the main emphasis in this Chapter. The first part of the analysis of the results in this Chapter will briefly examine how clinical features in this series relate to expected outcome. The CT scan features will now be examined in detail to find out how they relate to outcome.

It has previously been reported that extracranial insults, in particular hypoxia and hypotension, exert a deleterious effect on outcome such that the observed outcome is worse than would have been predicted from the Glasgow Coma Score alone (Kohi et al 1984). In this chapter the relationship between CT scan features and Glasgow Coma Score will be analysed in order to find out how they interact to influence outcome.

The results will also be analysed in relation to the different ways of management, with the comparison of the results in this series and in the Galbraith and Teasdale (1981) series. In this series a level of intracranial pressure >30mmHg was used to determine the need for surgery but in the Galbraith and Teasdale (1981) series neurological deterioration in the ICP monitored patients was used as the indication for operation.

8.2 RESULTS

a. Overall outcome of Patients with Intradural Haematoma

Table 35 shows the outcome of these patients at six months follow up. In all, 41% of these patients had an unfavourable outcome.

b. Clinical Features and Outcome

The age distribution of the patients shows that older patients faired worse than younger patients. About a third of the patients under 50 years and a half of those above 50 years had an unfavourable outcome (Table 36).

For the coma score groups 3-5, 6-7, 8-10 and 11-15, unfavourable outcome occurred in 83%, 61%, 51% and 11% of patients respectively. A worse outcome was associated with a lower coma score as expected (Table 37).

When both pupils reacted to light only 28% of these patients had an unfavourable outcome, compared to 60% and 76% of those in whom one and neither pupil reacted to light respectively (Table 38).

c. CT scan Findings and Outcome

i) Type of Haematoma (Table 39)

As expected the best results were in patients with extradural haematoma in whom only 24% had an unfavourable outcome. Next were the patients with intracerebral haematomas 35%, while half of those with subdural haematoma did badly. The worst outcome was in those who had both extradural and intradural haematoma, 55% had an unfavourable outcome.

iii) CT Scan Features (Tables 40 - 43 and Fig 13)

Abnormal CT scan findings were associated with a worse outcome. Only 24% of those with normal basal cisterns had unfavourable outcome compared to 62% of those in whom the basal cistern were obliterated (Table 40). This is similar to the 24% with normal third ventricle and 57% with obliterated third ventricle with that outcome (Table 41). Equally true were the patients with CVD in whom 56%, compared to only 22% of those without CVD had an unfavourable outcome (Table 42). The results in relation to midline shift are shown in Table 43 and the proportion of patients with unfavourable outcome for each degree of midline shift are shown in Fig 13. It is clear that increasing midline shift correlates with increasing proportion of patients with an unfavourable outcome.

d) Inter-relation between Glasgow Coma Score, CT Scan Characteristics and Outcome

The details of the data are shown in Appendix 2 to 7 while the summary of the analysis are shown in Tables 44 to 46.

It would not be appropriate to divide the patients by both midline shift and several coma score groups, because the number in each group would be too small to enable reasonable conclusions to be made from such an analysis. The other three representative CT scan features will therefore be examined. Patients in whom either the GCS or the status of the CT scan feature being studied or both

were missing are not included in this analysis.

In the patients in the lowest coma score group, the status of the CT scan features did not make any appreciable difference in those who ended up with unfavourable outcome. About 83% of all these patients faired unfavourably irrespective of the appearance of basal cisterns or third ventricles or lateral ventricles. This observation is also true in the next coma score group (GCS 6-7) in which around 60% of these patients faired unfavourably irrespective of the appearance of CT scan features. By definition all patients in these two coma score groups were in coma.

The results of the patients who had a significant impairment in consciousness (GCS 8-10) also show that the appearance of the three CT scan features did not appreciably affect the proportion of patients with unfavourable outcome. About 30% of these patients had unfavourable outcome irrespective of the status of the basal cistern, third ventricle or lateral ventricles.

In the patients with the highest coma score group however, the results are different. About 9% of those with normal CT scan features and 19% of those with abnormal CT scan features had unfavourable outcome. With respect to the presence of contralateral ventricle dilatation the difference in outcome is about three-fold, 7% and 22%.

e) Type of Management and Outcome (Table 47)

Patients who required immediate operation had by far the worst outcome as 51% of them had an unfavourable outcome.

Of the patients operated upon after a delay, 41% of those monitored clinically and 38% of those ICP monitored had unfavourable outcome.

Of the patients not operated upon after ICP monitoring 24% had an unfavourable outcome. The group of patients who were neither operated upon nor ICP monitored included patients in whom a poor outcome was predicted at the time of their initial assessment. The management outcome of that group shows that 28% of them had an unfavourable outcome.

f) Level of Intracranial Pressure and Outcome (Table 48)

A third of patients in the lowest coma score group had unfavourable outcome, seven of whom died. Of the patients who died, 5 died of respiratory related complications; two patients had aspiration following uncontrolled seizures about three months after leaving the INS, one 78 year old patients died of bronchopneumonia three months after discharge from the INS, one died of probable fat embolism while another had severe respiratory insufficiency. Two patients were found dead in bed and their cause of death was unknown as autopsies were not made.

Two thirds of the patients operated with ICP 20-30mmHg also died as did another third of those with ICP >30mmHg. The group of patients with ICP range 20-30mmHg so far appear to compare more unfavourably. Operation in this group of patients still depends on evidence of neurological deterioration or failure to improve.

g) Comparison of Present Series with Galbraith/Teasdale Series

The overall results in the present series reflect the observation that the patients in the previous series were selected while in the present series were not and were initially a neurologically worse group. In all, the operative results are comparable as 38% and 33% of the present and previous series had unfavourable outcome. In the unoperated patients however the outcome of this series was worse.

The differences in the operated and not operated patients in the previous series show that unfavourable outcome was more than twice as often as in the patients who were not operated upon. In the present series however, that difference between those the operated and unoperated patients is significantly reduced to about half as many patients.

In the present series unfavourable outcome in patients with ICP >30mmHg was 35% which is similar to the 33% reported in the previous series (Table 49b). There are thus no differences in outcome between the two groups of patients who had been operate upon after ICP had reached that level.

8.3 DISCUSSION

The observations in this study reiterate the prognostic signficance of age, level of consciousness as determined by the Glasgow Coma Score and pupillary response to light.

Abnormal CT scan features have correlated with unfavourable outcome. This was true in patients at all levels of consciousness. This confirms that obliteration of basal cistern obliteration of the third ventricle, presence of a dilated contralateral ventricle and midline shift exceeding 10mm have prognostic significance, as does a low coma score. Furthermore, in patients in the highest coma score group the presence of abnormal CT scan features carries a worse prognosis that could be determined by the Glasgow Coma Score alone. This finding has an important clinical implication because it is in the patients with the higher coma scores who are allowed to be observed when the significance of the intracranial haematoma is in doubt. These patients may remain clinically silent but they can be regarded as being on the verge of deterioration as indeed often happened. In deciding the type of initial management, therefore, it is important to attach significance to the status of CT scan features; these are perhaps even more important than the assessment of the level of consciousness alone. The need for surgery at any stage (immediate or delayed) was associated with a more unfavourable outcome than when there was no need for surgery. This was so even in the patients having immediate surgery in whom at least half had an unfavourable outcome. Although the results show that patients operated after clinical monitoring were slightly worse off than those after intracranial monitoring the difference was too small to be of statistical significance. It is noted that ICP monitoring did not lead to a worse outcome compared to the time honoured clinical observations.

The type of intracranial lesion does have an influence on outcome as was expected. As such patients with extradural haematoma continue to have the best outcome although it should be noted that none had delayed operation. At the end of the scale are patients with combined extradural and intradural haematoma. This may reflect a severe form of injury required to produce both these lesions. Since a poor outcome has been related to the severity of brain damage (Jennett et al 1975, 1976, 1977, 1979, Bruce et al 1982) then the findings in this study agree with these previous observations.

The overall results of the patients initially managed by ICP monitoring are worse than those reported in the Galbraith and Teasdale series. An important difference between the two series is that the patients were selected in the previous series but they were not selected in the present series. The patients in the present series were neurologically worse than the previous series.

Neurological deterioration was the basis for deciding the need for surgery in the series of Galbraith and Teasdale but in the present series the level of intracranial pressure had been the basis for operation. The finding that the difference in unfavourable outcome between those operated and those not operated is significantly reduced compared to the previous series can be regarded as a success. This may be argued from the opinion that if the patients in the previous series were selected and the need for surgery determined then they should not have been the

gross difference in outcome found between the operated and the non operated patients. But the crucial question is really what happened to the patients operated with ICP >30mmHg because it is that level at which the need for operation was recommended from the results of the previous series. The results of the present series show that there is in fact no difference in outcome between the two series. It can be argued that the present series was neurologically worse than the previous series and therefore a worse outcome was expected. But it was disturbing to find out that some patients with ICP >30mmHg also had deteriorated before surgery. Since deterioration is what one is striving to prevent then failure to do so contributed to a worse outcome than would have been desired. What was even more perturbing was that two thirds of the patients in the ICP range 20-30mmHg did deteriorate and were thererfore operated upon. However, only 5% of those with ICP <20mmHg did also deteriorate in this series as did 10% in the previous series. Retrieval surgery did not lead to a favourable outcome once deterioration had occurred. The level of intracranial pressure previously recommended was a results of the finding that all patients with ICP greater than 30mmHg inevitably deteriorated and that only half of those with ICP 20-30mmHg did deteriorate or need surgery.

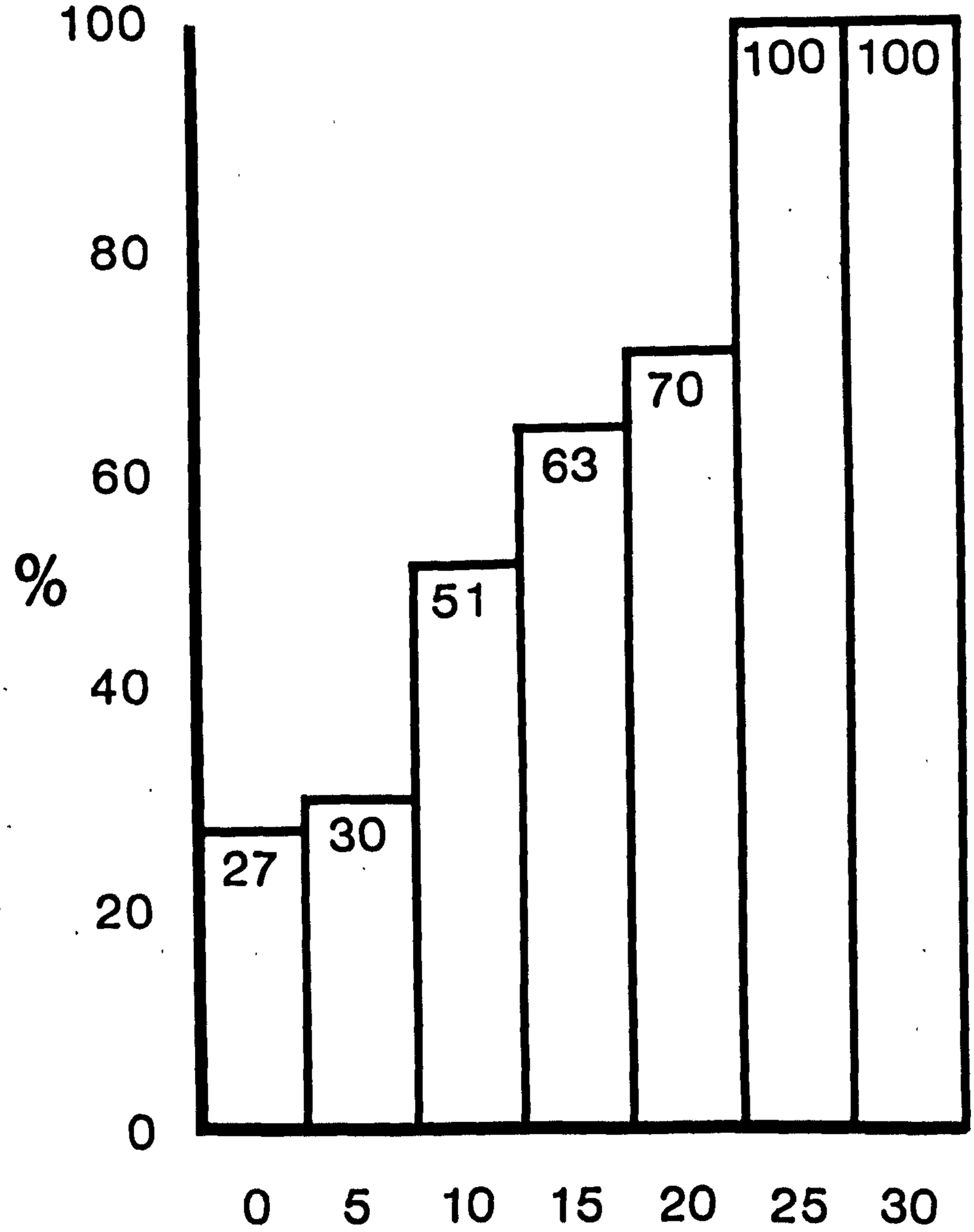
Arising from these results is the question of what really constitutes a clinically significant level of intracranial pressure in patients harbouring an acute traumatic intradural haematoma. It is unequivocally accepted that normal intracranial pressure lies between 0-10mmHg with the

upper limit of normal in the adult being 15mmHg. It can be conceived that intracranial pressures up to 20mmHg were less likely to be of significant clinical sequelae and that ICP of 20-30mmHg is moderately raised and is potentially clinically significant. At the end of this concept is the view that ICP >30mmHg is abnormally high and has inevitable clinical sequelae (except for the remarkably high levels of ICP seen in awake patients with benign intracranial hypertension or pseudo tumour cerebri). From this it can therefore be argued that intracranial pressure above 20mmHg could potentially lead to neurological deterioration and that measures to evacuate a haematoma need to be considered. It remains to be proved that operation of patients with ICP >20mmHg improves outcome but that would be a reasonable expectation.

FIGURE 13

Relationship between unfavourable Outcome and Midline Shift

Unfavourable Outcome and Midline Shift



Relationship between unfavourable outcome (Dead/Vegetative/Severe Disability) and midline shift in millimetres (mm).

Fig. 13

TABLE 35

OVERALL OUTCOME IN PATIENTS
WITH
ACUTE TRAUMATIC INTRADURAL HAEMATOMA

Outcome	Number of Patients	%
Dead	105	36%
Vegetative	4	1%
Severe Disability	35	10%
Moderate Disability	57	17%
Good Recovery	114	33%
Alive (Disability unknown)	23	7%
Lost	7	2%
TOTAL	345	100%

TABLE 36

AGE AND OUTCOME IN PATIENTS
WITH INTRADURAL HAEMATOMA

	Outcome Category							
Age (years)	D	V	SD	MD	GR	Lost	Alive	TOTAL
0 - 9	5	0	0	1	3	0	1	10
10 - 19	5	0	3	4	17	0	2	31
20 - 29	12	0	2	5	9	0	4	32
30 - 39	9	1	5	10	13	1	2	41
40 - 49	11	1	10	14	23	1	4	64
50 - 59	21	1	5	8	22	3	7	67
60 - 69	25	1	3	9	18	1	2	59
70 +	16	0	7	6	9	1	1	40
Unknown	1	0	0	0	0	0	0	1
TOTAL	105	4	35	57	114	7	23	345

TABLE 37

FINAL GLASGOW COMA SCORE (GCS) BEFORE OPERATION/
PRE-CT SCAN AND OUTCOME IN PATIENTS WITH INTRADURAL HAEMATOMA

Final GCS Sum	Outcome Category								% Unfavrourable Outcome
	D	V	SD	MD	GR	L	A	T	
3 - 5	46	1	6	5	3	0	3	64	83%
6 - 7	26	2	18	9	15	0	5	75	61%
8 - 10	15	0	15	17	14	2	5	58	52%
11 - 15	10	1	4	23	81	5	10	134	11%
Unknown	8	0	2	3	1	0	0	14	-
TOTAL	105	4	35	57	114	7	23	345	42%

TABLE 38

FINAL PRE-OPERATIVE/PRE CT SCAN PUPILLARY REACTION TO LIGHT
OUTCOME IN PATIENTS WITH INTRADURAL HAEMATOMA

Pupillary Reaction To Light	Outcome Category D	V	SD	MD	GR	L	A	TOTAL	% Unfavourable
Both react	40	1	24	44	98	7	17	231	28%
One reacts	9	1	5	6	3	0	1	25	60%
Neither reacts	55	2	4	6	8	0	5	80	76%
Unknown	1	0	2	1	5	0	0	9	-
TOTAL	105	4	35	57	114	7	23	345	42%

TABLE 39

TYPE OF TRAUMATIC INTRACRANIAL HAEMATOMA AND OUTCOME IN ALL PATIENTS

Type of Haematoma	Outcome Category D	V	SD	MD	GR	L	A	TOTAL	% Unfavourable
Extradural	10	1	5	8	33	1	8	66	24%
Extradural & Intradural	3	0	3	1	3	0	1	11	55%
Subdural	56	2	11	19	41	2	8	139	50%
Intracerebral	34	0	12	28	53	4	8	139	35%
Subdural & Intracerebral	12	2	9	9	17	1	6	56	41%
TOTAL	115	5	40	65	147	8	31	411	39%

TABLE 40

BASAL CISTERN APPEARANCE & OUTCOME IN PATIENTS WITH INTRADURAL HAEMATOMA

Basal Cistern Appearance	Outcome Category							
	D	V	SD	MD	GR	Lost	Alive	TOTAL
Not Obliterated	25	1	16	41	78	3	11	175
Obliterated	76	3	19	14	34	4	12	162
Unknown	4	0	0	2	2	0	0	8
TOTAL	165	4	35	57	114	7	23	345

TABLE 41

THIRD VENTRICLE APPEARANCE & OUTCOME IN PATIENTS WITH INTRADURAL HAEMATOMA

	Outcome Category							
Third Ventricle Appearance	D	V	SD	MD	GR	Lost	Alive	TOTAL
Not Obliterated	24	1	15	43	71	2	9	165
Obliterated	77	3	19	13	42	5	14	173
Unknown	3	0	0	1	1	0	0	5
TOTAL	105	4	35	57	114	7	23	345

TABLE 42

CONTRALATERAL VENTRICLE DILATATION & OUTCOME
IN PATIENTS WITH INTRADURAL HAEMATOMA

Contralateral Ventricle Dilatation	Outcome Category							
	D	V	SD	MD	GR	Lost	Alive	TOTAL
Absent	35	1	15	43	75	5	13	187
Present	67	3	19	14	39	2	10	154
Unknown	3	0	1	0	0	0	0	4
TOTAL	105	4	35	57	114	7	23	345

TABLE 43

MIDLINE SHIFT & OUTCOME IN PATIENTS
WITH INTRADURAL HAEMATOMA

Midline Shift in Millimetres (mm)	Outcome Category							
	D	V	SD	MD	GR	Lost	Alive	TOTAL
0	22	0	10	31	49	0	6	118
5	15	1	6	16	30	4	6	78
10	20	1	7	5	14	2	8	57
15	22	0	10	3	14	1	2	52
20	20	2	1	2	7	0	1	33
25	5	0	0	0	0	0	0	5
30	1	0	0	0	0	0	0	1
Unknown	0	0	1	0	0	0	0	0
TOTAL	105	4	35	57	114	7	23	345

TABLE 44

FINAL PRE-OPERATIVE/PRE CT SCAN GLASGOW COMA SCORE (GCS)
BASAL CISTERN APPEARANCE & OUTCOME IN PATIENTS WITH INTRADURAL HAEMATOMA

Final GCS	Basal Cistern Appearance	Outcome Category: Unfavourable D/V/SD	Outcome Category: Favourable MD/GR	Lost/Alive	% Unfavourable
3-5	Not Obliterated	5	1	0	83%
	Obliterated	47	6	3	84%
6-7	Not Obliterated	15	10	1	60%
	Obliterated	30	13	4	64%
8-10	Not Obliterated	12	20	3	34%
	Obliterated	6	10	4	30%
11-15	Not Obliterated	10	87	10	9%
	Obliterated	5	16	5	19%
Unknown	Not Obliterated	0	1	0	-
	Obliterated	10	3	0	-
TOTAL*		140	167	30	42%

* Basal Cistern appearance unknown in 8 patients

TABLE 45

FINAL PRE-OPERATIVE/PRE CT SCAN GLASGOW COMA SCORE (GCS), THIRD VENTRICLE APPEARANCE & OUTCOME IN PATIENTS WITH INTRADURAL HAEMATOMA

Final GCS	Third Ventricle Appearance	Outcome Category: Unfavourable D/V/SD	Favourable MD/GR	Lost/Alive	% Unfavourable
3-5	Not Obliterated	4	2	0	67%
	Obliterated	48	5	3	86%
6-7	Not Obliterated	14	10	0	58%
	Obliterated	31	14	5	62%
8-10	Not Obliterated	14	20	3	38%
	Obliterated	5	10	4	26%
11-15	Not Obliterated	7	81	8	7%
	Obliterated	7	23	7	19%
Unknown	Not Obliterated	1	1	0	-
	Obliterated	8	3	0	-
TOTAL*		139	169	30	41%

*This ventricle appearance unknown in 7 patients

TABLE 46

FINAL PRE-OPERATIVE/PRE CT SCAN GLASGOW COMA SCORE (GCS)
CONTRALATERAL VENTRICLE DILATATION (CVD) & OUTCOME
IN PATIENTS WITH INTRADURAL HAEMATOMA

Final GCS	Contralateral Ventricle Dilatation	Outcome Unfavourable D/V/SD	Outcome Favourable MD/GR	Category Lost/Alive	% Unfavourable
3-5	Absent	13	1	1	87%
	Present	40	7	2	82%
6-7	Absent	16	15	0	52%
	Present	28	9	5	70%
8-10	Absent	12	22	5	31%
	Present	6	9	2	35%
11-15	Absent	7	78	12	7%
	Present	8	26	3	22%
Unknown	Absent	3	2	0	-
	Present	7	2	0	-
TOTAL*		140	171	30	41%

*CVD could not be determined in four patients

TABLE 47

TYPE OF FINAL MANAGEMENT & OUTCOME
IN PATIENTS WITH INTRADURAL HAEMATOMA

Type of Management	Outcome Category D	V	SD	MD	GR	Lost	Alive	TOTAL	% Unfavourable
Immediate Operation	65	3	23	18	52	4	14	179	51%
Delayed Operation Clinical Observation	8	1	3	2	11	1	3	29	41%
Delayed Operation ICP Monitoring	6	0	5	5	8	0	5	29	38%
No Operation Clinical Observations	17	0	2	19	29	0	0	67	28%
No Operation ICP Monitoring	7	0	2	11	12	2	0	34	24%
TOTAL	105	4	35	57	114	7	23	345	42%

TABLE 48

OUTCOME, INTRACRANIAL PRESSURE & FINAL MANAGEMENT

Level of ICP	Final Management	Outcome Category D	SD	MD	GR	Alive	Lost	TOTAL
<20mmHg	Operative	0	0	0	0	2	0	2
	Non Operative	7	2	11	9	0	0	29
20-30mmHg	Operative	2	2	1	0	1	0	6
	Non-Operative	0	0	0	2	0	1	3
>30mmHg	Operative	4	3	4	8	1	0	20
Failed	Operative	0	0	0	0	1	0	1
	Non-Operative	0	0	0	1	0	1	2
		13	7	16	20	5	2	63

TABLE 49

COMPARISONS BETWEEN THE PRESENT SERIES
AND THE GALBRAITH/TEASDALE SERIES

A) OVERALL OUTCOME

		D/V	SD	MD/GR	Lost	Total	% Unfavourable
Present Series	Operated	6	5	18	0	29	38%
	Not Operated	7	2	23	2	34	26%
Galbraith/ Teasdale	Operated	2	2	8	0	12	33%
	Not Operated	1	1	12	0	14	14%

B) OUTCOME IN PATIENTS WITH ICP >30mmHg

Present Series	4	3	13	0	20	35%
Galbraith/ Teasdale	1	1	4	0	6	33%

C) NEED FOR SURGERY IN PATIENTS WITH ICP <30mmHg

ICP (mmHg)		Operated	Not Operated	% Operated
<20mmHg	Present Series	2	29	6%
	Galbraith/Teasdale	1	9	10%
20-30mmHg	Present Series	6	3	67%
	Galbraith/Teasdale	5	5	50%

CHAPTER 9

FINAL DISCUSSION

AND

CONCLUSIONS

9.1 THE CURRENT MANAGEMENT OF PATIENTS WITH INTRADURAL HAEMATOMA

There did not appear to be doubt in the minds of the clinicians that surgery was required immediately in about 50% of patients presenting with an acute traumatic intradural haematoma. In a small proportion of patients, about 3%, the clinical state was considered to indicate such a poor prognosis that surgery would not alter the outcome. The problems in discussions about management lay in the remaining patients in whom it was initially expected that recovery was likely to occur without recourse to surgery. Subsequent assessment by clinical observations and intracranial pressure monitoring identified the need for surgery in about half of these patients. Thus three quarters of patients who initially presented with an acute traumatic intradural haematoma eventually required to be operated upon. **The problem remains the early identification of which patients should be operated upon before awaiting neurological deterioration,** the historical clinical hallmark that denotes the need for surgery.

9.2 RELATIONSHIP BETWEEN CT SCAN AND CLINICAL FEATURES

The results emphasise the importance of clinical features in the initial and subsequent assessment of head injured patients suspected of a traumatic intracranial haematoma. Such patients often presented with altered level of consciousness and when definitive measures were delayed further deterioration usually occurred.

The abnormal CT scan features analysed were obliteration of basal cisterns, obliteration of third ventricle, a dilated contralateral ventricle and midline shift exceeding 10 millimetres. Their presence correlated with the observed impairment in consciousness and pupillary dysfunction. However, these features were also found in some patients whose level of consciousness had not significantly altered. This finding implies that these CT scan features precede changes in clinical features. Their presence therefore may provide warning of impending deterioration in the level of consciousness or pupillary function.

9.3 CT SCAN FEATURES AND MANAGEMENT

The practical significance of abnormal CT scan features was best demonstrated in the patients in whom an initial decision in favour of immediate surgery had been made, because of their presenting clinical state. The great majority of these patients had abnormal CT scan features. Furthermore, a significant proportion, between 30-40%, of those patients who eventually required surgery after either clinical monitoring or ICP monitoring, also initially had abnormal CT scan features. It was also found that half of the patients whose ICP was >30mmHg initially had abnormal CT scan features. Had CT scanning been repeated at the time when ICP was greater than 30mmHg it might have shown that all patients with ICP >30mmHg have abnormal CT scan features.

These findings emphasise the conclusion that abnormal CT scan features are an early guide to the need for surgery.

9.4 INTRACRANIAL PRESSURE MONITORING IN THE MANAGEMENT OF PATIENTS WITH INTRADURAL HAEMATOMA

The practical problems in the initial management of a patient with a clinically silent acute traumatic intradural haematoma are first if ICP should be monitored and second is ICP monitoring the best basis on which to decide about the need for operation. It is now a fact that changes in ICP precede changes in clinical state. Monitoring based on clinical features would therefore lead to delayed identification of which patients needed remedial surgery. The results of this study have shown that abnormal CT scan features precede changes in the clinical state. It would therefore be advantageous if such a non-invasive technique predicted changes in clinical state. But the technique of CT scanning does not at present enable us to have a continous display of CT scan features. Intracranial pressure is so far the only available early idex of intracranial dynamics that can readily be displayed continuously. In patients with clinically silent acute traumatic intradural haematoma, ICP monitoring is therefore at present the most practical tool to use in deciding which patients are likely to deteriorate and therefore needed surgery. In patients without a surgically significant intracranial haematoma the technique of ICP monitoring is still a useful guide to changes in intracranial dynamics. This enables therapeutic teams to institute

measures to lower ICP and to assess the efficacy of the different methods used to prevent ICP from rising to life threatening levels.

Which patients with clinically silent acute traumatic intradural haematoma should have ICP monitoring? The results of this study lead to the recommendation that ICP monitoring should be considered in patients in whom the initial CT scan features are not grossly abnormal, that is the basal cisterns and third ventricle are not obliterated, the contralateral ventricle is not dilated and that the midline shift, is present, does not exceed 10 millimetres. The optimum duration of monitoring should be 72 hours because during that period at least 90% of patients would have declared the need for surgery. After that period it is adviseable to perform follow up CT scans at day 3 and day 5. This view reflects the finding that patients with a low ICP who eventually deteriorated did so by the 6th day.

The corollary to this recommendation is that patients found to have abnormal CT scan features should be operated upon immediately - regardless of how good their initial clinical state appears to be. To delay surgery is to await neurological deterioration, which is inevitable.

The ideal objective of monitoring intracranial pressure in patients harbouring a clinically silent acute traumatic intradural haematoma is to identify which patients need surgery before neurological deterioration has occurred. The previous study by Galbraith and Teasdale showed that neurological deterioration was inevitable when ICP exceeded

30mmHg and that 50% and 10% of patients with ICP of 20-30mmHg and <20mmHg respectively also eventually deteriorated. They suggested that if ICP was >30mmHg it was better to institute surgery before neurological deterioration occurred. This suggestion was the basis of the management of the patients ICP monitored in the present series. It was disturbing to find out that some patients with ICP >30mmHg still deteriorated before surgery. In the patients with ICP <30mmHg neurological deterioration was still the basis for further management. About two thirds of the patients with ICP 20-30mmHg in this series deteriorated before operation was deemed to be necessary. This suggests that the present recommended level of ICP at which to operate is too high. This could explain the failure to substantially improve on the result as was found out in this study. It can be hypothesised from this study that operation based on ICP >20mmHg is likely to result in improvement in outcome.

9.5 APPLICATION OF FINDINGS TO RELATED PROBLEMS

For several decades there has been disagreement as to the optimum method of treating patients with a spontaneous intracerebral haematoma, especially those due to systemic hypertension or of a cryptogenic cause. In 1961 McKissock and co-authors had shown that the results of surgery were not better than conservative treatment. For those who continued to advocate for surgical treatment the depressed level of consciousness was the main indication for operating while denying surgery for those with mild symptoms or a poor prognosis (Luessenhop et al 1967, Paillas et al 1973, Tedeschi et al 1975, Kaneko et al 1977).

Few reports have studied intracranial pressure characteristics in patients with spontaneous intracerebral haematoma. Janny and co-workers (1978) were only interested in measuring ICP in order to 'observe' the natural history of this disorder while Duff and co-workers (1981) did so in order to evaluate the efficacy of medical means of maintaining adequate cerebral perfusion pressure in patients treated non-surgically, also suggesting that osmotic agents were useful in lowering ICP. Although Papo et al (1979) had shown that raised intracranial pressure was found in patients in coma, the eventual decision in favour of surgery in that series was based on the level of consciousness alone. The level of intracranial pressure was not used as an indication for surgery and the CT scan features not even mentioned.

The results in this thesis may be relevant also to patients with a spontaneous haematoma, which does behave as a space occupying lesion akin to a traumatic intradural haematoma. It is suggested that it would be profitable to examine the relevance of CT scan features to management decisions. Thus the presence of abnormal CT scan features (obliterated basal cisterns, obliterated third ventricle, dilated contralateral ventricle, or midline shift exceeding 10mm) may be considered as indications to evacuating the haematoma. In the patients who are found to have normal CT scan features at the time of initial assessments, I believe that measurement of intracranial pressure will be a useful guide to the need for surgery, which should be considered if the level of ICP exceeds 20mmHg.

9.6 THE FUTURE

Recommendations have been made in this thesis of the application of intracranial pressurre monitoring in the initial management of patients with clinically silent acute traumatic intradural haematoma. Whether final managmement is based on the CT scan and ICP criteria suggested will improve the results remains to be proven.

There have been recent significant investigative radiological advances. CT scan definitions and scan time have greatly been improved. Nuclear Magnetic Resonance (NMR) or Magnetic Resonance Imaging (MRI) so far provide the finest anatomical definitions possible. Positron Emission Tomography (PET) provides both anatomical and physiological (metabolic) definitions of intracranial structures. How useful these recent developments are in the initial assessment, management and prognositication of head injured patients has yet to be determined.

Appendix 1

PROTOCOL FOR

NON OPERATIVE HAEMATOMA STUDY

HAEMATOMA STUDY - NON OPERATED CASES

1. HAEMATOMA STUDY NUMBER (INCLUDING CENTRE CODE IN COLUMNS 2 AND 3) — 1 | | | | | | | — 1-7
2. NAME (SURNAME FIRST) — | | | | | | | | | | | | | | | | | | | | — 8-19
3. UNIT NUMBER — | | | | | | — 20-25
4. DATE OF INJURY — | | | | | | — 26-31 (DAY MONTH YEAR)
5. DATE OF CT SCAN — | | | | | | — 32-37

CODING INSTRUCTIONS

N. B. TIMING ON FORM IS FROM CT SCAN

EYE OPENING
4=SPONTANEOUS
3=TO SOUND
2=TO PAIN
1=NIL

BEST MOTOR RESPONSE
6=OBEY
5=LOCALISE
4=NORMAL FLEXION
*3='ABNORMAL' FLEXION
2=EXTENSION
1=NIL

VERBAL RESPONSE
5=ORIENTATED
4=CONFUSED
3=WORDS
2=SOUNDS
1=NIL

MOTOR PATTERN IN RIGHT/LEFT LIMB
1=NO RESPONSE
2=EXTENSION
*3=ABNORMAL FLEXION
4=BETTER TYPE OF FLEXION OR LOCALISING. BUT WEAKER THAN THE OTHER SIDE
5=BETTER TYPE OF RESPONSE AND NORMAL STRENGTH

*-SPASTIC. DECORTICATE. STEREOTYPED ABNORMAL HAND CLENCHING OR LEG ALSO EXTENDS

USE 4/5 TO INDICATE ASYMMETRY

EYE SIGNS

PUPIL REACTION
1=EQUAL REACT
2=UNEQUAL REACT
3=ONLY ONE REACTS
4=NEITHER REACTS <2MM
5=NEITHER REACTS 2-4MM
6=NEITHER REACTS >4MM
7=NEITHER REACTS UNEQUAL

SPONTANEOUS EYE MOVEMENT
1=ORIENTING
2=ROVING CONJUGATE
3=ROVING DYSCONJUGATE
4=LATERAL DEVIATION
5=NONE
6=OTHER

SIDE OF LARGER OR DILATED PUPIL/ LOCAL FACTORS AFFECTING PUPILS
1=NEITHER/NONE
2=RIGHT
3=LEFT
4=BOTH

OCULOCEPHALICS
1=ORIENTATING
2=FULL/CONJUGATE
3=DYSCONJUGATE/MINIMAL
4=ABSENT

OCULOVESTIBULARS
1=NYSTAGMUS
3=CONJUGATE(AT LEAST ONE SIDE)
3=BILATERAL DYSCONJUGATE
4=ABSENT

PERSONAL DATA

6. SEX
   1=MALE
   2=FEMALE

☐ 38

7. AGE (IN YEARS)

☐☐ 39-40

INJURY DATA

☐ 41

8. TYPE OF INJURY
   1=MOTOR VEHICLE
   2=PEDESTRIAN
   3=RTA OTHER (MOTORCYCLE, PEDAL CYCLE)
   4=SPORT
   5=WORK
   6=ASSAULT
   7=DOMESTIC (+ FALL FROM WINDOW)
   8=FALL UNDER INFLUENCE OF ALCOHOL
   9=OTHER (INCLUDES GUNSHOT)

9. INFLUENCE OF ALCOHOL OR OTHER DRUGS ON INITIAL ASSESSMENT
   1=NO
   2=SUSPECTED
   3=DEFINITE : SPECIFY_____

☐ 42

10. LUCID INTERVAL (=TALKED AT SOME STAGE BEFORE OPERATION)
   1=NONE
   2=PARTIAL - WORDS/CONFUSED
   3=TOTAL - SENSIBLE/ORIENTATED

☐ 43

11. PLACE OF INITIAL ADMISSION
   1=TO OTHER HOSPITAL
   2=SAME HOSPITAL, NON NSU
   3=TO NSU
   4=ER TO THEATRE

☐ 44

12. BEST STATE
   0=NOT KNOWN
   1=BEFORE FIRST ADMISSION
   2=BETWEEN FIRST ADMISSION AND NSU
   3=AFTER NSU

☐ 45

TIME FACTORS

TIME FROM INJURY UNTIL:-

13. FIRST ADMISSION TO ANY HOSPITAL
14. ADMISSION TO NEUROSERVICE
15. OPERATION
16. ONSET OF DETERIORIATION
17. TIME FROM FIRST ADMISSION TO NEUROSERVICE
18. TIME FROM ONSET OF DETERIORATION TO OPERATION
19. TIME FROM ONSET OF DETERIORATION TO NEUROSERVICE

CODING FOR 13-19
1=<1 HOUR
2=1-3 HOURS
3=3-6 HOURS
4=6-12 HOURS
5=12-24 HOURS
6=24-72 HOURS
7=>72 HOURS
8=GRADUAL
9=DID NOT OCCUR/ NOT RELEVANT

☐ 46
☐ 47
☐ 48
☐ 49
☐ 50
☐ 51
☐ 52

EXTRACRANIAL COMPLICATIONS

20. CHEST INJURY
21. OTHER TRUNK INJURY
22. LIMB INJURY
23. FACIAL INJURY

CODING FOR 20-23
1=NO
2=MINOR
3=MAJOR - REQUIRING HOSPITAL ADMISSION

☐ 53
☐ 54
☐ 55
☐ 56

CRANIAL INJURY

24. FRACTURE OF SKULL (X-RAY, CLINICAL OR OBSERVATION)
1=NONE
2=VAULT
3=BASE-(X-RAY OR ORBITAL/MASTOID HAEMATOMA OR CSF LEAK)
4=BOTH

☐ 57

25. SIDE OF FRACTURE
1=RIGHT
2=LEFT
3=BILATERAL
4=NOT KNOWN

☐ 58

26. EPILEPSY (BEFORE OPERATION)
1=NO
2=FOCAL
3=GENERAL
4=BOTH

☐ 59

INVESTIGATIONS

27. EMI/ANGIO
    1=NEITHER
    2=EMI
    3=ANGIO
    4=BOTH

☐ 60

28. SHIFT
    1=NO
    2=TO LEFT
    3=TO RIGHT

☐ 61

29. SHIFT SIZE
    1=<0.5 CM
    2=0.5-1.0 CM
    3=1.0-1.5 CM
    4=>1.5 CM

☐ 62

30. CONTRALATERAL VENTRICLE
    1=NORMAL
    2=SMALL
    3=DILATED

☐ 63

31. BASAL CISTERNS
    1=ABSENT
    2=PRESENT

☐ 64

EMI RESULT

	RIGHT	LEFT	POST FOSSA	
32-34. INCREASED DENSITY	☐	☐	☐	65-67
35-37. REDUCED DENSITY	☐	☐	☐	68-70
38-40. HAEMATOMA	☐	☐	☐	71-73

32-34. INCREASED DENSITY
    1=EDH
    2=SDH
    3=ICH
    4=1+2
    5=1+3
    6=2+3
    7=1+2+3

35-37. REDUCED DENSITY
    1=SDH
    2=IDH
    3=BOTH

38-40. HAEMATOMA
    1=EXTRADURAL
    2=SUBDURAL
    3=INTRACEREBRAL
    4=1+2
    5=1+3
    6=2+3
    7=1+2+3

HAEMATOMA STUDY NUMBER [2][ ][ ][ ][ ][ ][ ] 1-7

PRE-CT CLINICAL FEATURES

COMA SCALE

		1ST	ADM HOSP	ADM NSU	BEST PRE-CT	FINAL PRE-CT	
41-44.	EYE OPENING (1-4)		☐	☐	☐	☐	8-11
45-48.	BEST MOTOR RESPONSE (1-6)		☐	☐	☐	☐	12-15
49-52.	VERBAL RESPONSE (1-5)		☐	☐	☐	☐	16-19

MOTOR RESPONSE PATTERNS

53-60.	RIGHT SIDE (1-5)	☐	☐	☐	☐	20-23
57-60.	LEFT SIDE (1-5)	☐	☐	☐	☐	24-27

EYE SIGNS

61-64.	PUPIL REACTION (1-7)	☐	☐	☐	☐	28-31
65-68.	SIDE OF LARGER/DILATED PUPIL (1-4)	☐	☐	☐	☐	32-35
69-72.	LOCAL FACTORS AFFECTING PUPILS (1-4)	☐	☐	☐	☐	36-39

EYE MOVEMENTS

73-76.	SPONTANEOUS (1-6)	☐	☐	☐	☐	40-43
77-80.	OCULOCEPHALICS (1-4)	☐	☐	☐	☐	44-47
81-84.	OCULOVESTIBULARS (1-4)	☐	☐	☐	☐	48-51

HAEMATOMA STUDY NUMBER [3][ ][ ][ ][ ][ ][ ] 1-7

POST-CT SCAN CLINICAL FEATURES

COMA SCALE

		START ICP MON	24HR BEST	24HR WORST	2-3D BEST	4-7D BEST	
85-89.	EYE OPENING (1-4)	☐	☐	☐	☐	☐	8-12
90-94.	BEST MOTOR RESPONSE (1-6)	☐	☐	☐	☐	☐	13-17
95-99.	VERBAL RESPONSE (1-5)	☐	☐	☐	☐	☐	18-22

MOTOR RESPONSE PATTERNS

100-104.	RIGHT SIDE (1-5)	☐	☐	☐	☐	☐	23-27
105-109.	LEFT SIDE (1-5)	☐	☐	☐	☐	☐	28-32

EYE SIGNS

110-114.	PUPIL REACTION (1-7)	☐	☐	☐	☐	☐	33-37
115-119.	SIDE OF LARGER/DILATED PUPIL (1-4)	☐	☐	☐	☐	☐	38-42
120-124.	LOCAL FACTORS AFFECTING PUPILS (1-4)	☐	☐	☐	☐	☐	43-47

EYE MOVEMENTS

125-129.	SPONTANEOUS (1-6)	☐	☐	☐	☐	☐	48-52
130-134.	OCULOCEPHALICS (1-4)	☐	☐	☐	☐	☐	53-57
135-139.	OCULOVESTIBULARS (1-4)	☐	☐	☐	☐	☐	58-62

140. TEMPORAL PATTERN OF RESPONSIVENESS IN FIRST 24 HOURS ☐ 63

1=NO CHANGE
2=IMPROVING
3=DETERIORATING
4=FLUCTUATING

HAEMATOMA STUDY NUMBER [4][ ][ ][ ][ ][ ][ ] 1-7

PRE-CT AUTONOMIC ABNORMALITIES

CODING FOR 141-146
1=NEVER PRESENT
2=TEMPORARY, NOT FINAL PRE-CT
3=ABNORMAL AT FINAL PRE-CT

141.	BP>160	[ ] 8
142.	BP<60	[ ] 9
143.	PULSE<60	[ ] 10
144.	PULSE>120	[ ] 11
145.	APNOEA (NOT INDUCED BY RELAXANT DRUGS AND LONG ENOUGH TO REQUIRE AT LEAST TEMPORARY VENTILATION)	[ ] 12
146.	RESPIRATORY FREQUENCY>30	[ ] 13

POST-CT AUTONOMIC ABNORMALITIES

CODING FOR 154-180
1=NO
2=TEMPORARY
3=UP TO END OF EPOCH

		24HRS	2-3D	4-7D	
147-149.	BP>160	[ ]	[ ]	[ ]	14-16
150-152.	BP<60	[ ]	[ ]	[ ]	17-19
153-155.	PULSE<60	[ ]	[ ]	[ ]	20-22
156-158.	PULSE>120	[ ]	[ ]	[ ]	23-25
159-161.	APNOEA	[ ]	[ ]	[ ]	26-28
162-164.	RESPIRATORY FREQUENCY>30	[ ]	[ ]	[ ]	29-31
165-167.	LOW PACO2 (<30)	[ ]	[ ]	[ ]	32-34
168-170.	HIGH PACO2 (>50)	[ ]	[ ]	[ ]	35-37
171-173.	LOW PAO2 (<50)	[ ]	[ ]	[ ]	38-40

TREATMENT

		PRE-CT	POST-CT 24HR	POST-CT 2-3D	POST-CT 4-7D	
174-177.	STEROIDS 1=NONE 2=<20MG DEXAMETHASONE DAILY 3=>20MG DEXAMETHASONE DAILY 4=UNKNOWN OR SHOCK DOSE	[ ]	[ ]	[ ]	[ ]	41-44
178-181.	OSMOTICS 1=NONE 2=ONE DOSE 3=REPEATED DOSE 4=UNSPECIFIED	[ ]	[ ]	[ ]	[ ]	45-48

TREATMENT CONTINUED

182-185. TRACHEOSTOMY/TUBE/VENTILATION (EXCLUDING TEMPORARY OR TERMINAL) ☐ 49-52
1=NO
2=INTUBATED
3=TRACHEOSTOMY
4=2+CONTROLLED VENTILATION
5=2+PATIENT TRIGGERED
6=3+CONTROLLED VENTILATION
7=3+PATIENT TRIGGERED

186-189. DRUGS POSSIBLY AFFECTING OBSERVATION ☐ 53-56
1=NO
2=YES SPECIFY _____

TIME TO

190. OBEY
191. SPEAK
192. EYES OPEN TO PAIN
193. RECOVERY FROM ABNORMAL MOTOR PATTERN

1=<6 HOURS
2=6-12 HOURS
3=12-24 HOURS
4=24-72 HOURS
5=4-7 DAYS
6=8-14 DAYS
7=15-28 DAYS

☐ 57
☐ 58
☐ 59
☐ 60

OUTCOME

194. BEST AT ANY TIME
195. 3 MONTHS
196. 6 MONTHS
197. 1 YEAR

1=DEATH
2=VEGETATIVE STATE
3=SEVERE DISABILITY
4=MODERATE DISABILITY
5=GOOD RECOVERY
6=OUT OF HOSPITAL, LOST TO FOLLOW-UP (=4 OR 5)
IF 2/3 INDISTINGUISHABLE CODE 7

☐ 61
☐ 62
☐ 63
☐ 64

N. B. SEVERE- CONSCIOUS BUT DEPENDENT, IE. REQUIRING THE HELP OF ANOTHER PERSON DURING EVERY 24 HOURS.
MODERATE- INDEPENDENT BUT DISABLED.

198. TIME TO DEATH AFTER CT SCAN ☐ 65
1=<24 HOURS
2=2-3 DAYS
3=4-7 DAYS
4=8-14 DAYS
5=15-28 DAYS
6=>28 DAYS

199. UNPREDICTABLE COMPLICATION ☐ 66
1=NO
2=PRE- CT
3=POST-CT

APPENDIX 1a

Letter of correspondence to Family Physicians and/or next of kin of patients in non operative study

INSTITUTE OF NEUROLOGICAL SCIENCES, GLASGOW

Department of Neurosurgery
Mr. A. Paterson
Prof. Bryan Jennett
Mr. J. W. Turner
Mr. T. A. Hide
Prof. G. M. Teasdale
Mr. S. L. Galbraith
Mr. D. Mendelow

Southern General Hospital
1345 Govan Road
Glasgow G51 4TF
Telephone : 041-445 2466
Ext :—

_____

_____

_____

_____

_____

The above mentioned patient was admitted to this Institute on _____ after a head injury. As he/she has not been seen again for follow-up, could you kindly please provide us with the following information:-

1.	Lives independently	YES	NO
2.	Requires help at least once every 24 hours	YES	NO
3.	Physical Disability	YES	NO
	If Yes - State Disability		
4.	Mental Disability	YES	NO
5.	Able to, or returned to work/school	YES	NO
6.	Has he/she developed epilepsy	YES	NO
	If so - Focal/Generalised/Both	YES	NO
7.	Taking anti-epileptic drugs	YES	NO
	State when started		
8.	Died	YES	NO
	State cause		

Thank you for your cooperation.

Yours sincerely,

APPENDIX 2

Final pre-operative/pre CT Scan Glasgow Coma Score Basal Cisterns Obliterated, & Outcome in Patients with Intradural Haematoma

	Outcome Category							
Final Pre-Op/ Pre CT Scan GCS	D	V	SD	MD	GR	Lost	Alive	TOTAL
3 - 5	41	1	5	4	2	0	3	56
6 - 7	18	2	10	3	10	0	4	47
8 - 10	5	0	1	4	6	2	2	20
11 - 15	4	0	1	1	15	2	3	26
Unknown	8	0	2	2	1	0	0	13
TOTAL	76	3	19	14	34	4	12	162

APPENDIX 3

Final pre-operative/pre CT Scan Glasgow Coma Score
Basal Cisterns Not Obliterated & Outcome
in Patients with Intradural Haematoma

	Outcome Category at six months							
Final GCS	D	V	SD	MD	GR	Lost	Alive	TOTAL
3 - 5	4	0	1	1	0	0	0	6
6 - 7	7	0	8	5	5	0	1	26
8 - 10	8	0	4	12	8	0	3	35
11 - 15	6	1	3	22	65	3	7	107
Unknown	0	0	0	1	0	0	0	1
TOTAL	25	1	16	41	78	3	11	175

APPENDIX 4

Final pre-operative/pre CT Scan Glasgow Coma Score (GCS) Third Ventricle Obliterated and Six Month Outcome in Patients with Intradural Haematoma

	Outcome Category							
Final GCS	D	V	SD	MD	GR	Lost	Alive	TOTAL
3 - 5	42	1	5	3	2	0	3	56
6 - 7	18	2	11	4	10	0	5	50
8 - 10	4	0	1	3	7	2	2	19
11 - 15	6	0	1	1	22	3	4	37
Unknown	7	0	1	2	1	0	0	11
TOTAL	77	3	19	13	42	5	14	173

APPENDIX 5

Final pre-operative/pre CT Scan Glasgow Coma Score (GCS) Third Ventricle Not Obliterated & Outcome in Patients with Intradural Haematoma

	Outcome Category							
Final GCS	D	V	SD	MD	GR	Lost	Alive	TOTAL
3 - 5	3	0	1	1	0	0	0	6
6 - 7	7	0	7	5	5	0	0	24
8 - 10	10	0	4	13	7	0	3	37
11 - 15	3	1	3	22	59	2	6	96
Unknown	1	0	0	1	0	0	0	2
TOTAL	24	1	15	43	71	2	9	165

APPENDIX 6

Final pre-operative/pre CT Scan Glasgow Coma Score (GCS) Contralateral Ventricle Dilated* & Outcome in Patients with Intradural Lesions

	Outcome Category							
Final GCS	D	V	SD	MD	GRR	Lost	Alive	TOTAL
3 - 5	35	1	4	4	3	0	2	49
6 - 7	14	2	12	3	6	0	5	42
8 - 10	4	0	2	2	7	1	1	17
11 - 15	7	0	1	4	22	1	2	37
Unknown	7	0	0	1	1	0	0	9
TOTAL	67	3	19	14	39	2	10	154

* CVD unknown in 4 patients: 3 died, 1 severe disability

APPENDIX 7

Final pre-operative/pre CT Scan Glasgow Coma Scale (GCS) Contralateral Ventricle Not Dilated & Outcome in Patients with Intradural Haematoma

	Outcome Category							
Final GCS	D	V	SD	MD	GR	Lost	Alive	TOTAL
3 - 5	11	0	2	1	0	0	1	15
6 - 7	11	0	5	6	9	0	0	31
8 - 10	9	0	3	15	7	1	4	39
11 - 15	3	1	3	19	59	4	8	97
Unknown	1	0	2	2	0	0	0	5
TOTAL	35	1	15	43	75	5	15	187

R E F E R E N C E S

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