COMPUTED TOMOGRAPHY OF HEAD INJURIES : OBSERVER RELIABILITY AND NEUROPATHOLOGICAL CORRELATION

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ABSTRACT

Head trauma constitutes a substantial problem. Major advances have occurred in understanding the pathophysiology, epidemiology and long term sequelae of brain injury. Research efforts have lead to the introduction of more sophisticated neuroimaging techniques which have made the diagnosis of the lesions associated with head injury earlier and more frequent by, generating a better understanding of the injured brain.

This thesis has been carried out to examine the reliability and the validity of the CT scan diagnosis. A comprehensive definition of the lesions occuring on a CT scan was the basis for testing reliability and the validity.

The reliability or the consistency of describing CT scan appearances by different levels of observers was tested by, comparing 13 observers scores of the CT scans appearances of 28 head injured patients. It was found that the reliability of a CT scan diagnosis could be improved if better definitions of the lesions were achieved.

To test the validity of the radiological-pathological correlatins, 102 fatal head injured patients were studied and their worst CT scans and post-mortem pathological results were compared. The results showed that, the previously held idea that CT simply and directly shows living pathology must be in doubt.

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CHAPTER I

INTRODUCTION

The management of severely head injured patients has been greatly advanced over the last decade. This is the result of several developments in knowledge and understanding of pathological processes, of epidemiology, of the importance of ischaemia and secondary insults and advances in investigative procedures, in particularly computed Tomography.

Computed Tomography (CT) now is the main method of diagnostic assessment of head injured patients. It can confirm the occurrence of traumatic brain damage in head injury and the occurrence or threat of intracranial complications. It provides an index of prognosis, and its results are used to classify series of cases for reporting . Crucial clinical decisions and influential scientific conclusions rest upon the findings of the CT scan .

This thesis examines two important aspects of the role of the CT scan in head injuries. First, reliability : the consistency of reporting of CT appearance by different observers. Second, validity: the evidence from post-mortem for the existence of the radiological - pathological correlation that underpin the interpretation of CT scan findings.

1.1 Head injury

Several facts highlight the importance of head injury as a clinical and social problem, Attention to the study of head injuries has risen as the death rate from heart disease and stroke have fallen by 22% and 32% respectively while the death rate from trauma has risen by approximately 1% each year since 1977.(1) In multiple trauma, the head is the most commonly injured part, and in fatal accidents, injury to the brain is found in nearly 75% of victims autopsied.(2) Head injuries without other injuries are even more common. In Britain 200-300/100,000 of population are admitted to hospitals every year, of which the percentage is higher in men than women and in younger rather than older people.(3,4) In Scotland 3% of all admission to all acute hospital beds are head injuries and these account for 1% of bed usage.(5)

The definition of a head injury takes different forms and there is controversy about choosing words. Some define a head injury as any event with a blow or impact on the head or even face others require evidence of presumed brain involvement i.e. concussion with loss of consciousness, post-traumatic amnesia or neurological signs of brain injury, or of impact sufficient to cause a skull fracture even without altered consciousness.

Jennett and Teasdale (5), indicated that there is no absolute criteria that define a head injury, and it is difficult in many instances to avoid some reference to severity, whether this is defined administratively or in biological terms. The difficulty of the latter is that the scalp, skull and brain can each be injured without involvement of the other, but it is the brain damage actual or potential that is the key for medical

interest in blows to the head and the term craniocerebral trauma is therefore a reasonable synonym for head injury.

1.2 Imaging investigation of craniocerebral trauma

Traditionally patients with brain diseases had a clinical diagnosis made on the basis of history, physical findings and specialised neurodiagnostic tests ,but the procedures provided limited information and did not always lead to a definite diagnosis. Most of the previous information about clinical correlates with disease resulted from autopsy studies, but such patients were a rather special subgroup of all clinical cases (those who had died). As a result, a great deal of the information about what was happening inside the head in self limiting, non-fatal injuries and even fatal head injuries prior to death, was unavailable, This was changed by the revolution in neurological investigation that followed the practical application of the theory of how computer technology could be used to produce a series of cross sectional images of the body.

In 1972 the British physicist Godfrey Hounsfield, and Radiologist Dr James Ambrose were the first to develop and apply the CT scan. The first report was presented at the British Institute of Radiology in April 1972, and the first CT scan was instillated at Atkinson Morley's Hospital in Wimbledon, United Kingdom (6,7) .

The CT scanner consists of an X-ray tube and a detector opposite the tube, the X-ray beam, either very narrow (a pencil beam) or very wide (fan beam) is passed through each slice of interest in the patients body, the exit beam then striking

the detector opposite the tube which measures and records its intensity. As the tube moves around the patient, the sequence is repeated, with the amount of radiation actually reaching each detector being measured for each different exposure. The resulting data are then stored and analysis in a computer (6&7). The attenuation coefficient is represented by a different shade of grey, A hard copy is then made.

A CT scan is essentially an electronic image; this means that the diagnostic picture is formed by using an electronic detection device instead of film opposite the X-ray tube.

The advantages of using an electronic device for detecting photons is that the efficiency of utilising of photons presented in it is 80%, where it is only 5% for the X-ray films. So the advantages of CT scan are

1- Its ability to distinguish between two similar objects slightly different in density.

2- It provides a slice largely eliminating photon interactions above and below this slice.

3- It permits a more efficient utilisation of the X-ray beam that lowers the dose of radiation.

Since 1972 a wide experience in the use of this instrument has accumulated It is now possible to construct what is often thought to be akin to a 'living autopsy' and to examine the gross structure of the brain in any type and at any time after a head injury or even any illness. This has made day to day management of patients safer and better; it has also enlarged knowledge about brain disease .

In 1974 (the year of the description of Glasgow Coma Scale for the evaluation of coma through clinical signs and symptoms by Teasdale and Jennett) (8), CT scanners became more widely available, This was followed by a major alteration in the preference for special neurodiagnostic tests ; ultrasound detection of the cerebral midline was abandoned completely except in infants ; ventriculography and pneumoencephalography virtually disappeared and EEGs were considerably reduced. Cerebral angiography has continued because of its role in vascular disorders but even this is being challenged by Magnetic resonance imaging (MRI)

MRI is a relatively recent development which is significantly different from existing methods of radiographic imaging. The clinical application of MRI started in 1982 and the first picture of a human head was produced in 1978 (9) Like CT it is a non-invasive computer assisted image technique but differs in the type of energy used. MRI depends on the physical phenomenon of spinning subatomic particles of nuclei called nucleons . The multiplanar capabilities of MRI lead to better demonstrate of extra-axial haemorrhage located subfrontally, subtemporally or along the tentorium and it is more sensitive than CT in detecting non haemorrhage lesions, especially areas of shear injury (10). However, CT has advantages compared to MRI :

1- The high field strength magnetic environment makes life support of critically ill patients more difficult

2- Acute haemorrhage may be difficult to identify on MRI.

3- The time taken to perform MRI with current apparatus is typically twice the time needed for CT . The time needed for the patient to be immobile during exposure is 2 seconds or less for a CT slice , where MRI requires the patient to be immobile for 4 to

6 minutes. Although faster techniques are becoming available (e.g. ECHO-planar imaging), they will still usually need sedation which is undesirable in a recent head injury because it may mask a change in the neurological condition of the patient.

4- Rapid examination is important, If a prior medical history is unavailable. MRI is contraindicated in patients with electronic implants, any metal or ferromagnetic foreign bodies ,thus if a patient with serious intracranial injury requires immediate surgery, CT is preferred.

5- The MRI is more expensive than CT, the cost of MRI is twice to three times the cost of CT.

6- MRI may be most useful in evaluating patients with a head injury during the subacute stage, when it may elucidate the severity of parenchymal image and inform prognosis.

1.3 CT in head injury

CT remains the investigation of choice for the acute head injured patient, It provides the necessary information safely with minimal inconvenience to the patient. This has been achieved by the advancement in CT technology in the last two decades.

The first generation of CT scan, the EMI scanner which was commercially available in 1973. It was suitable only for head scanning and the scanning time was 4 minutes per pair of slices. The images were produced as Polaroid pictures and the tissues exhibiting the lowest density were shown as black areas, Only the most gross lesions were detected. The first generation was slow but still offered a tremendous tool for neuroradiological diagnosis.

By 1975 the second generation,(EMI scanner, CT 1010) was developed which was a modified fan beam scanner. The head was confined in a water bag and 10-30 seconds for each slice were required. This second generation scanner offered a significant reduction in the time required for scanning, however its 10-30 seconds scan time was still too long to prevent artefacts caused by respiration or intestinal movements and vascular pulsations. EMI was soon not the only manufacturer of CT scanner and EMI it self was taken over by General Electric in 1979.

The third generation scanner offered a wide-angle scan beam with multiple detectors, the scanning time for each slice was reduced to about 5 seconds or even less. General Electric manufactured this generation (CT 9800) scanner in 1983.

In the fourth generation machines, the rotational scan system, the tube is rotating 360 degrees very rapidly with a wide fan beam and fixed detector. the scanning time for each slice became 5 seconds or less. This was the type of machine used in this study.

The most recent development is of spiral CT scanning, a scan time of less than a second.

1.4 Technical advancements

Developments over the years have provided high resolution digital radiographs with improvement in the quality and sharpness of the image , current scanners allow the resolution of structures of less than 0.8 mm. As slices become thinner, minute lesions can be detected and the time becomes shorter and shorter. The

speed of the scanner is directly related to safety, The lesser the time of the exposure to radiation the safer the scanner, the speed of the scanner also enhances the sharpness of the image and decreases the artefacts. The most troublesome artefact are kinetic artefacts that are usually produced by the patient movement, respiratory excursions, intestinal peristalsis or pulsation of the heart and blood vessels.

Although CT contrast media are much used in tumour detection, contrast media are not routinely used for initial imaging of the head injury. Contrast enhancement is more useful to rule out an isodense collection in a follow up scan.

The pathological features of head injuries displayed by CT imaging are discussed in the second chapter of this thesis , Initially the findings on CT scan were classified as extraparenchymal and intraparenchymal lesion in the form of EDH , SDH , ICH , contusions. Shearing lesions were described by Zimmerman (8), Stritch, and Peerless, the term DAI subsequently being introduced by Adams and Gennarelli (11). After 1980 , CT sign of increased ICP attracted a lot of attention because of the frequency of raised ICP and its potential to produce further brain dysfunction and damage by decreasing cerebral blood flow and promoting brain shift and herniation . Recently traumatic subarchnoid haemorrhage has attracted attention as an adverse prognostic sign and its possible relations to traumatic arterial spasm and cerebral ischaemia .

1.5 Indications for CT

There is widening use CT imaging of head injury, including those who are clinically stable , however it is important that patients be subjected to a test only when cost , inconvenience and risk are outweighed by the potential value of the result. Several factors have addressed the indications for CT in head injury . Recently Dacey et al (11) concluded that clinical data alone are insufficient for determining which patients are at risk of deterioration due to delayed complication and they predicted a 50% cost saving if CT scans were used to determine the necessity for hospital admission .

In the last decade, several sets of guidelines or criteria for scanning have been produced, based upon varying degrees of evidence and date . The most definitive studies came from Mendelow ,Teasdale and colleagues in Glasgow (12,13). These showed that a linear skull fracture substantially increases the probability that an intracranial haematoma is present. The presence of a fracture accompanied by any impairment of brain function depression of consciousness, focal neurological signs or epilepsy, is an absolute indication for urgent CT scanning . It is now widely accepted that the presence of skull fracture alone may be an indication for CT , likewise persisting neurological dysfunction even without a fracture . The urgency with which CT is obtained depends upon the grade of severity of head injuries

1- In severe head injury (GCS 8 or less) patients CT should be carried out as soon as the patient is judged safe to be moved from emergency room as the incidence of Intracranial haemorrhage is found to be up to 40% (13).

2- In moderate injury (GCS = 9 to 12) CT should be carried out soon after admission and immediately if the patient has a skull fracture , has focal neurological deficits or has had seizures (14).

3- In mild head injury (GCS > 12) CT should also be carried out early if the patient has a skull fracture , has focal neurological deficit or has seizures . even if these factors are absent CT should be carried out if the patient does not regain the full score of 15 points on GCS within 24 hours of admission to hospital .

Recently some controversy has arisen over what really constitute a low risk patient , Stein and Ross (15), identified CT abnormalities in 185 of patients with GCS of 13 to 15 of whom 5% required neurosurgical intervention. When patients with a GCS of 13 were analysed separately , 40% had CT abnormalities and 10% required operative intervention . The authors felt that with such a high frequency of CT abnormalities , head injured patients with GCS score 13 should be classified as moderate rather than low in severity and risk. They recommended CT for any patient with GCS of 14 or 15 who has experienced transient loss of consciousness or amnesia and for all patients with GCS of 13, regardless of clinical findings . It appears that in North American practice, skull x-rays play little or even no role in the routine evaluation of head injury and all patients are considered as a candidates for CT scanning

The American college of surgeon advanced trauma life support guidelines for CT scanning state, all head injured except for patients with a trivial head injury will require CT scanning at some time .

Since 1980, in the UK, increasing numbers of head injuries have been transferred to neurosurgical units and this has led to an improvement in results . Recently an increasingly liberal policy for scanning of head injury is being employed ,

with CT before transfer to the Neurosurgical Unit as CT machines become more available in district hospitals.

The reliability and the validity of the CT scan are of utmost value to the diagnosis , treatment and prognosis of head injured patients. Two important questions remain to be answered in order to judge the reliability and validity of the CT scan in head injury

1- How consistent is the interpretation of CT by several observers . Surprisingly, apart the very early study by Galbraith (16) , restricted to haematomas, there are no studies of observer variation in interpretation of the CT of head injury

2- How accurate are the CT scan finding or how well does the CT scan depict the pathology of head injury . Likewise comparison of the finding of CT scan of head injuries and pathology findings has been the topic of very few studies (17,18)

The paucity of published work on the topics of the reliability and validity of the CT images of head injured patients in clinical practice and research work stimulated for this and is the basis of this thesis.

CHAPTER II

LITERATURE REVIEW

The first report of CT of head injury was by **JAMES AMBROSE** (7), Using CT scanning he found that in intracranial haemorrhage the associated tissue damage will usually result in some surrounding oedema and therefore reduction of tissue density, but once clotting has occurred, there is a great increase in average density, enabling the lesion to be recognised, and the extent and relation to other structures defined. In chronic subdural haematoma he was able to describe a lens shaped area of either high or low density and displacement of the ventricular system or pineal body, A thin poorly defined low density line marking the boundary between haematoma and brain was also stated.

Ambrose stated that in head injury it may be important (but difficult) to distinguish between cerebral contusion , laceration , or on one hand oedema and on the other hand an intracerebral haemorrhage. He classified the abnormalities seen on CT scan according to the density alterations they exhibit in the pictures:

1- Lesion with an average density higher than that of normal tissue.

2- Lesion with an average density lower than that of the surrounding normal tissue.

3- Lesion with the same density as normal tissue.

Ambrose further showed that in cranio-cerebral trauma, the CT scan provided an easy method of distinguishing between extradural , subdural or

intracerebral haematomas and cerebral oedema resulting from brain damage based on a higher density than normal for the extradural, subdural and intracerebral haematoma, and also identifying the displacement and distortion of the ventricular system and pineal body may add information about the nature of the abnormalities (19).

DUBLIN AND FRENCH (20), Retrospectively studied 200 cases of documented head injury. They found that using the CT scan, differentiation between oedema and a surgically correctable lesion as a well defined haematoma was greatly improved and it also permitted the earlier detection of the intraventricular haemorrhage They eliminated the term acute , subacute and chronic and preferred instead to refer to extracerebral collections as increased density , isodensity or decreased density in relation to normal brain. They found that a subdural haematoma may actually increase in density during the first 1 to 3 days after trauma, and that evaluation of intracerebral haematoma should be based on change in mass effect rather than CT density. They highlighted the fact that a clinical picture of "brain stem contusion" could be associated with a surprisingly high number of surgically correctable lesions. French and Dublin (21), then studied 1000 consecutive cases of head injury. They related clinical severity to CT findings the more severely injured more often had an abnormal scan : less than 44% of those with deep coma and abnormal motor movement had a normal scan and this was found in only 15% of those in coma with lateralising signs. They highlighted the fact that a clinical picture of "brain stem contusion" could be associated with a surprisingly high number of surgically correctable lesions.

IN 1977 KOO AND LA ROQUE (22), studied 457 patients who presented with a history of head trauma. Of these, 165 patients showed CT scan features of an acute or chronic abnormality. They indicated that CT scan detected cerebral oedema from traumatic injury much earlier "in a matter of hours", than oedema resulting from other causes such as ischaemia. They considered that a helpful CT sign in diagnosis was that a contusion is invariably associated with brain oedema while a small extra-cerebral collection is usually not.

IN 1978 ZIMMERMAN ET AL. (23), reviewed the CT scan findings in 286 patients with acute crania-cerebral trauma to see if CT scan carried prognostic value. They classified the CT scan findings:-

1- No focal abnormalities

A- Normal or minimal abnormality

B- General cerebral swelling

C- Diffuse shearing injury of cerebral white matter .

2- focal abnormalities .

A -Intracerebral abnormalities .

Haemorrhage contusion .

Intracerebral haematoma .

Focal cerebral swelling .

B- Extra-cerebral abnormalities .

Acute subdural haematoma

Epidural haematoma .

They reported that the small haemorrhagic lesions which are commonly associated with diffuse white matter damage of impact type , can often be seen on the CT scan if examinations are repeated with thin cuts. They indicated that CT scan led to prompt and effective treatment. Thus patients with CT scan findings consistent with shearing injury (small haemorrhage in the corpus callosum and dorso-lateral quadrant of the brain stem) invariably had a poor prognosis, whereas the paediatric patient with general cerebral swelling had a good prognosis. Investigation by CT scan led to a significant improvement in mortality rate for intracerebral , subdural and epidural haematomas.

They further classified a parenchymal lesion into cortical contusion and intracerebral lesions, which ranged from large haematomas in continuity with cortical contusions (burst lobe), subcortical lesions, haematomas in the basal ganglia and small haemorrhage lesions considered to be related to tissue tearing, particularly when located in the parasagittal white matter, the corpus callosum or the dorsolateral quadrant of brain stem.

KISHORE ET AL. (24), correlated CT findings and ICP (only to patients with unequivocally normal initial CT scan and those with haemorrhagic lesions), they used the broad division of classification of CT scan into normal, haematoma either extracerebral or intracerebral. They found that of patients with a normal initial CT , 38% had ICP of 20 mmHg. or less, and in 17% ICP was controllable and between 21 and 40 mm Hg. during the monitoring period, of patients with extra-axial haematomas 40% had ICP between 21-40 mm Hg. and 17% had severe and controllable ICP (> 40

mm Hg.), of patients with an intracerebral haematoma, 33% had ICP between 21-40 mm Hg. and 20% had severe intracranial hypertension, in 2 out of 3 uncontrollable, of the patients with both lesions, 67% had ICP between 21-40 mm Hg., and 33% had ICP ≥ 41mm Hg. (71% uncontrollable).

They indicated that patients with a haematoma have higher incidence of elevated ICP, most often severe (> 40 mm Hg.), and , occasionally patients with normal initial CT develop intracranial hypertension, however, 83% of the patients with normal CT scan did not develop high ICP. They highlighted the division of intracranial lesions into extracerebral and intracerebral, and of the extracerebral into subdural and extradural collections. They showed in their series that some clinically useful assumptions could be made from the CT scan findings . A majority of patients with CT scan demonstrating haemorrhagic lesions had raised ICP , whereas 83% of patients with a normal initial CT scan did not have raised ICP. (98% of these patients had normal ICP during the first day after trauma and 91% did not have raised ICP until the third day after trauma or later).

SADHU ET AL. (25), reported that raised intracranial pressure did not relate to CT features of asymmetrical ventricular compression , midline shift or obliteration of the mesencephalic cistern , fissures or sulci . The clearest correlation with raised ICP in their 21 cases of head injury was dilatation of the contralateral temporal horn.

IN 1983 LOBATO ET AL. (26), on the basis of experience with 277 severely head injured patients using CT scans and continuous measurement of intracranial

pressure. They outlined eight basic anatomical patterns of lesion, which they believed had clinical and physiopathological significance and provide useful prognostic information and facilitated improved therapeutic decision making in severely head injured patients .

The patterns identified were:-

1- Pure extra-cerebral haematoma. 2- Extra-cerebral haematoma plus acute hemispheric swelling. 3- Single brain contusion , whether or not associated with a neighbouring extra-cerebral haematoma. 4- Multiple unilateral brain contusion , whether or not associated with subdural haematoma. 5- Multiple bilateral brain contusion. 6- General brain swelling whether or not associated with small extra-cerebral haematoma. 7- Diffuse axonal injury. 8- Normal CT scan. They found that 84% of patients with pattern 1 had recovered within 2 weeks after surgery, and 16% died after several days, with pattern 2 the large preoperative midline shift persist, even increased, after surgery, 100% of those patients had severe intracranial hypertension, and 100% of the patients died within 2-7 days, with pattern 3, partial lobectomy was performed for 56% of patients with pure contusion because of intracranial hypertension, 11% developed severe intracranial hypertension, 77.7% had good recovery, and 22% had poor recovery, with pattern 4 rising ICP occurred in 78% of those cases and 60% of the patients had severe intracranial hypertension, and 75% of patients died, with pattern 5 raised ICP occurred in 82% of those cases and 42% developed severe intracranial hypertension, the mortality rate in this pattern was 50% of the cases, with pattern 6 , 87% of the patients had good outcome, with pattern 7, 86% of the patients had poor outcome and with pattern 8 they indicated that no patients with this CT pattern had intracranial hypertension, 68% had good outcome,

and 32% had poor outcome. They concluded that CT patterns in severely head injured patients are indicative of the severity of injury and correlate with both ICP and final outcome, however the CT picture alone does not give an absolute prognosis.

MURPHY AND TEASDALE (27), found a striking correlation between CT scan appearance (obliteration of basal cistern and third ventricle) and a raised intracranial pressure in patients with a diffuse head injury .They studied the relation between ICP and CT scan findings in head injured patients : the ventricular volume, and the appearances of the third ventricle and basal cisterns. They utilised three groups of patients: the first group patients with diffuse injuries and their CT scans did not show a significant intracranial clot , the second group was patients with an "occult" intracranial clot and in stable clinical condition and "non-operative" management and the third group was postoperative patients who had an acute traumatic intracranial haematoma evacuated and their ICP was monitored and CT repeated postoperatively. They found that neither in the patients with a diffuse injury nor in those with an occult haematoma was there evidence of a relationship between ventricular volume and ICP. and in patients with an occult haematoma or patients studied postoperatively the appearances of the basal cisterns and the third ventricle were not related to the level of ICP.

The clearest correlation they found was between the CT scan appearance of obliterated basal cisterns and the third ventricle and a raised ICP in patients with diffuse injury. Disappearance of the third ventricle is a sensitive and reliable index of brain swelling; failure to see the basal cistern may indicate that the basal subarachnoid space

has become obliterated , because of downward shift , when cerebrospinal fluid is seen in both structures the raised intracranial pressure is rarely above 20 mm Hg. However, CT scan can not be used to provide an index of raised intracranial pressure in patients with an occult haematoma .

VAN DONGEN K. ET AL. (28), studied retrospectively 121 severely head injured patients who were in coma when admitted to NSU, to assess whether a relationship exists between CT scan findings made on admission or 24 hours after injury, and outcome of these patients after six months or one year survival , and to establish whether CT findings add prognostic information to that supplied by clinical state according to the protocol used by the International Data Bank. They classified the CT findings of their patients into 4 categories, taking into account the type and size of intracerebral or extracerebral lesion, the state of basal cistern and ventricles and the presence of midline shift.

They found that the individual CT findings combined with clinical features allowed 62% predictions of survival or death higher than by CT or clinical alone and that, in this combination, the state of the basal cistern was the main discriminator. Also, the state of the basal cisterns, whether compressed, obliterated or open was a powerful prognostic indicator and strongly correlated with the pupil reactivity to light.

TOUTANT S. ET AL. (29), studied the information on the appearance of the basal cisterns as seen on the first scan of 218 consecutive severely head injured patients entered into the second phase of the National Pilot Traumatic Coma Data

Bank, to describe the value of the appearance of the basal cisterns in early prediction of outcome in severely head injured patients. The outcome was determined either at the time of death or 90 days or more following injury. They concluded that the basal cistern appearances can be an independent and potent predictor of outcome within the first few hours of injury; in other words if the basal cisterns were absent or compressed or normal the rate of poor outcome was 85%, 64%, and 44% respectively.

LIPPER M. ET AL. (30), used the size of a haemorrhagic lesion on the initial CT scan to predict outcome of patients with haemorrhage lesions. They stated that as the number of CT scan slices on which a haemorrhage lesions was seen increased, there was a corresponding decrease in good outcome and increase in poor outcome. Thus 80% of patients with normal CT scan in their series had a good outcome, while 20% had poor outcome. and 100% of patients with both extra and intra-axial haemorrhagic lesion ended up with poor outcome, 75% of patients who developed bilateral lesions within the first week were found to have a poor outcome, 80% of patients with corpus callosum lesion had a poor outcome, and the extent of midline shift used provided a good prognostic indicant, of patients with a midline shift $\geq$ 3.8 mm, 63% had a poor prognosis.

BAYKANER K. ET AL. (31), They showed that all the chronic haematoma cases were arterial and subacute cases were venous in origin. The mortality rate for patients with an extradural haematoma was 9.4% when CT was used for diagnosis compared with the mortality rate of 33% for conventional diagnostic

facilities. They reported a gradual decrease in mortality with the increasing availability and use of CT scanning .

HARA M. ET AL. (32), showed that in patients with an intracranial lesion it is possible to estimate the occurrence of raised Intracranial pressure on the bases of CT scan findings concerning midline shift , size of the lesion , contralateral ventricular dilatation and Evans ratio, provided that the nature of the lesion and the patients age are taken into account .

BULLOCK R. TEASDALE G. (33), considered the role of CT scan in the context of management of intracranial collections. They indicated that the classification of parenchymal lesions is difficult especially the distinction between contusion, haematoma and haemorrhages. In deciding if a mass lesion is surgically "significant", they advised that the features used should include the site, size and constitution of the haematoma, and the current clinical state of the patient, but pointed to increasing emphasis on the secondary space-occupying consequences as indices of whether surgery is appropriate. They also highlighted the grave risk of midline shift using enlargement of the temporal horn is an early index to this significant effect.

EISENBERG H. ET AL. (34), related the initial CT scan in patients with a head injury to abnormal intracranial pressure and to death. They demonstrated that CT scan findings indicating herniation , either transtentorial or subfalcine were strongly associated with the risk of elevated ICP and with death .

They also found that a normal CT scan (6%) as well as abnormal CT scan (21%) not demonstrating abnormal mesencephalic cisterns , midline shift mass effect or subarchnoid haemorrhage were associated with a low risk of elevated ICP and death .

They concluded that the status of the mesencephalic cisterns and degree of midline shift were very strongly associated with high ICP and death. Another conclusion was that extracerebral masses are more ominous predictors than are intracerebral masses and that all other CT scan features are of less importance, with the possible exception of subdural haematoma .

They classified the CT scan abnormalities to:-

1- Normal CT scan.

2- Mass lesion high or mixed density collection (Intracerebral or extracerebral) $\geq$ 15 cc.

3- Bilateral diffuse swelling, defined as compressed or obliterated mesencephalic cisterns and/or small ventricles without midline shift (defined as midline shift) $\leq$ 3 mm

4- Diffuse swelling with shift > 3 mm .

5- Other abnormalities .

Also they indicated that subarachnoid haemorrhage is associated with a more severe injury and only occasionally could be a target for treatment such as when it result in hydrocephalus.

MARSHALL L. ET AL. (TCDB) (35), using the same patient population, extended the work of Eisenberg. They enunciated the approach now widely known as the Traumatic Coma Bank System, they believed this could be used

both as a research and clinical tool in association with predictors of neurologic status. This classification aims to guide early management and when used in conjunction with other risk factors, predict the outcome.

2.1 Conclusion

Computed Tomography gained more and more importance as it became widely used as an essential tool in investigation of head injured patients, the widening use led to more understanding of the lesions and a drawing of definitive line between normal and abnormal. The nature of the abnormal lesions became more clear since the first description by (Ambrose 1973). The diagnostic accuracy of CT exceeds that of other investigation as cerebral angiography, and in some studies the rate of accuracy for an abnormal CT scan reaches 100%. The CT scan reveals significant lesions in severely head injured patients quickly, and permits more and rapid accurate identification of haematomas which sometimes are life threatening. The earlier detection of an extradural haematoma resulted in a significant improvement in outcome, and a striking decline in mortality rate from 29% in pre CT scan era compared to 12% after CT. As CT scans became more frequently used an extradural haematoma became detected in some minor head injuries. However CT of limited ability in detecting chronic haematomas that are isodense where the distinction between the haematoma and adjacent cortex is lost.

The CT is also of limited value in its ability to reveal cerebral contusions especially when adjacent to bone ,although haemorrhagic contusions are the single most frequent traumatic parenchymal lesions identified on CT scan. The ability of CT scan to demonstrate shearing injury is also limited, it does not directly show the numerous torn axons and, the diagnosis is indirectly made from the presence of small haemorrhage in specific areas.

Abnormalities on CT scan are also useful in predicting raised ICP. Any abnormality in the symmetry of the ventricular system was shown to be associated with a subsequent intracranial hypertension. The CT has the ability to predict elevated ICP, from midline shift on the CT, and the appearance of the III ventricle, basal cisterns on the initial CT scan. Also the CT findings enhance the ability to predict outcome. however the absence of a mass effect on the CT scan does not role out raised ICP. The CT scan now is able to detect small lesions within the brain, and also it has been used for classification of head injury. Nevertheless the identification of mass lesions remains the most important role in management of head injury.

CHAPTER III

TRAMATIC INTRACRANIAL LESIONS DISPLAYED BY COMPUTERISED TOMOGRAPHY

The interpretation of CT and its use in the classification of head injury is based upon multiple pillars, one of the most important is a good understanding and a clear definition of the appearances on the CT scan produced by different pathologies in head injury.

3.1 Technique

It is necessary to ensure that the patient is scanned in a such a way that all the required information will be available and that any potential further harm to the patient is minimised while the scan is carried out (38). Any necessary resuscitative measures must be instituted and continued during scanning and attending staff should be protected from radiation hazards. Ideally the patient should be scanned in the supine position but if the patient is restless a decubitus position may be adopted and still provide diagnostic images

The standard protocol for cerebral CT should extend from the foramen magnum to the vertex and be angled parallel to the orbitomeatal line to avoid irradiating the lens of the eye (39). In the posterior fossa the slice thickness should not

exceed 5 mm in order to minimise partial volume artefact, to optimise the signal to noise ratio and to improve contrast resolution. If a slice thickness of 10 mm is used, small haematomas will be missed.

Examination provides accurate definition of the radiological pattern of injury especially if the patient is co-operative. Subsequent scans can then be performed with thicker (10 mm) slices and reduced cumulative radiation dose. The image can be displayed in different fashions by altering window and level function on the computer console to emphasise differences in tissue attenuation (Table 1). The ability to vary window and level function is an important tool in the diagnosis of many pathologic processes, particularly small extra-axial collections lying adjacent to cranium.

TABLE 1

WINDOW AND LEVEL SETTING OF COMPUTED TOMOGRAPHY

	Width	Level
Narrow "brain" (window)	80	40
Blood (window)	150	60
Bone (window)	2000 - 4000	500

The physics of CT imaging are beyond the scope of this thesis. In brief the CT maps the way in which different tissues absorb or attenuate the beam of X-ray and so can normal structures and pathology. Blood clot, because it contains large compacted protein molecules has a high electron density and relatively little water, absorbs the X-ray more than normal brain and so is displayed as a dense or white area of increased beam attenuation. Conversely, oedema or ischaemia because of a relative increase in water content and hence a reduction in electron density are displayed as dark areas of reduced beam attenuation. Normal grey and white matter have attenuation between these two extremes and as grey matter is more cellular and vascular it has a higher attenuation than white matter.

Developments in CT scanners have significantly improved CT image quality and reduced the time needed for scanning and reconstruction of the images. Even faster dynamic scanning programmes have been developed that provide acquisition of data from the entire volume being imaged prior to reconstruction and image production. A minimum scan time of between one and two second per slice is now routine. Both soft tissue and bone window can be obtained from the same data set and 3 dimension reconstructions are possible.

3.2 Indications

The Guidelines of Practice for Head Injuries Management in United Kingdom were set up in the early 1980s, when the extremely restricted scanning facilities. That only those at high risk could undergo CT scanning.

As CT scans become more widely available, the indications for CT after head injuries are also widening and continue to yield positive findings, This indicates a policy of scanning any patient with persistent impairment of consciousness or any neurological signs irrespective of whether or not a skull fracture is present[40]. Most neurosurgeons believe that if CT scanning is available it must be done before operation[5-41-42-43]. Nothing can be be gained by delaying the CT scan to save minutes in neurosurgical units.

During the period of this study, the guidelines for investigation by skull x-ray and CT that applied as outlined in (Table 2).

TABLE 2

CRITERIA FOR SKULL RADIOLOGY AFTER RECENT HEAD INJURY

Clinical	*Recommendations*
- Asymptomatic - no loss of of consciousness. (low risk)	No skull x-ray, discharge with advice to responsible second person.
- Loss of consciousness or amnesia at any time - Neurological symptoms or signs - CSF or blood from nose or ear - Suspected penetrating injury - Scalp bruising or swelling - Difficulty in assessing the patients (e.g. alcoholic intoxication, epilepsy)	Skull x-ray or immediate CT if appropriate

3.3 Follow-up CT

The close correlation between a deterioration in the clinical condition and new CT abnormality necessitates follow-up scanning whenever the patient's condition fails to improve or worsens. Sometimes post-operative follow-up scanning is required to evaluate the effect of surgery and to serve as a baseline for future changes.

Based upon experimental and clinical evidence, the pathology of head injury is divided traditionally into two main types, the damage sustained at the moment of injury (primary brain damage) which takes the form of cerebral contusion and diffuse axonal injury and that produced by complicating processes (secondary brain damage). Some secondary damage may be initiated at the moment of injury and some develops sometime after the injury itself. The principal secondary abnormalities are haematomas, , brain swelling, raised ICP, hypoxic brain damage, hydrocephalus, and infection (Table 3).

The other principal approach to the classification of traumatic brain damage is to consider the damage as focal and diffuse, in the current era of CT scanning, the various types of focal brain damage can usually be identified during life, but identification of the precise nature of diffuse brain damage is more difficult. (44).

TABLE 3

CLASSIFICATION OF TRAUMATIC BRAIN DAMAGE

Primary injuries
(occurring at the time of impact)
Contusions
Diffuse shearing injury

⇓

Intracranial haematomas
Extradural
Subdural
Intracerebral
Subarachnoid
Intraventricular

⇓

Secondary Intracranial consequences
Swelling
Raised intracranial pressure
Ischaemic damage
Herniation
Infection

⇑

Systemic complications
Hypotension
Hypoxaemia
Epileptic seizure.

In this chapter the CT findings in head injury are discussed with respect to their radiological appearances and how CT may point to pathophysiological process. The specific anatomical traumatic lesion are :

3.4 CONTUSION

Contusions are bruises of the brain formed by coalescence of petechial haemorrhages caused by direct contact with the inner table of the skull. They are the most common complication of the head injury and have a great variety of appearances which change with time.

Mechanistically, contusion are classified as coup and contracoup lesions. the "coup lesion" lies under the point of impact or fracture, and "contracoup" lesions are remote from impact site, most often situated in the inferior frontal lobes and anterior temporal tips.

Pathologically, a contusion is a localised traumatic necrotic and haemorrhagic lesion characteristically more severe at the crest of a gyrus, but often extending through the cortex into the subcortical white matter.(45) Some investigators make a distinction between a contusion and laceration on the bases that in a contusion the pia is intact but in a laceration the pia and the underlying brain are torn. Contusions occur characteristically at the frontal and temporal regions, where the brain comes in contact with the skull base, and less frequently at the inferior surface of the cerebellum,

however, contusion may occur elsewhere as well. Despite contusion being common, they cannot be invoked as the cause of loss of consciousness at the time of injury nor in the maintenance of a comatose state. However a contusion may cause focal seizures and neurological or psychological deficits related to the site of the contusion.

It is particularly important that contusions can cause neurological deterioration after lucid interval.(11,46) Contusion may be associated brain oedema. The focal brain oedema associated with parenchymal contusion begins about 24 hours after injury, and steadily increases over the next seven days, after that slowly resolves.(47) On CT scan contusions usually have a mottled or salt and pepper appearance, appearing as ill defined or homogenous areas of increased attenuation (haemorrhage) with interspersed areas of low attenuation (oedema) in the typical subfrontal and temporal regions. The appearance is due to multiple small haemorrhages associated with areas of oedema and tissue necrosis. Haemorrhage is always present pathologically, but may be only microscopic and undetectable on CT.

3.5 Diffuse axonal injury (DAI)

Holbourn(48) in 1943 used a gelatine model of the brain to study the mechanism responsible for blunt head trauma. He emphasised that both direct injury (usually produced by skull fractures that directly lacerate or contuse the subadjacent brain) and indirect injuries (resulting from shear-strain deformation) were characterised changes in shape without changes in volume. Strich provided further support for the

shearing mechanism and was first to define clearly the occurrence of diffuse damage of cerebral white matter.[49]

Adams and Gennarelli were the first to introduce the term diffuse axonal injury.[50] Diffuse axonal injury occurs more easily with oblique and lateral head impact than with sagittal impact. The rotational component of acceleration/deceleration head injury produces diffuse cortical/subcortical disconnection, maximal in the periphery of the brain. As the severity of rotational forces increase, the more central areas of the brain (brain stem) become involved. A shearing injury of the corpus callosum is caused by less force than that resulting in a brain stem shearing injury. The brain tissue density and the rigidity in relation to the adjacent tissue is an important determinant of whether an area of brain is vulnerable to DAI.

The mechanism of head injury can be helpful in clinically distinguishing between DAI and an expanding mass lesion. High speed motor vehicle accidents are the most common cause of DAI (acceleration/deceleration injury) while falls from standing positions have rarely been associated with DAI.[48,51,27] Skull fracture and extra-axial haematoma are less frequent in DAI than in other non-DAI injuries. DAI injury is a microscopic diagnosis, the macroscopic clue is the frequent association, in more severe DAI, with tissue tears visible at PM or on CT.

Microscopically in those surviving a DAI for a more than 24 hours, retraction ball formation may be observed in white matter of cerebral hemisphere, cerebellum and brain stem. If a patient survives slightly longer, microglial clusters can be seen. If a

patient survives for prolonged periods, Wallerian degeneration of fibre tracts, which are sheared at the time of injury, are detectable.[5,51] Macroscopically, depending upon the time of survival from injury to death, tissue tears may be seen as small haemorrhagic areas or as shrunken yellowish scarring.

DAI is a diffuse type of injury and is the commonest cause of coma in the absence of an intracranial haematoma, mass lesion or ischaemic insult. Until recently DAI lesions were thought to be complete immediately. Some studies now shown that this is not the case and this therefore opens a therapeutic window through which it may be possible to limit the extent of damage.

Clinically DAI exists as a spectrum varying from transient cerebral dysfunctions to deep coma. DAI occurs in almost one half of severely head injured injury patients, causes more than one third of head injury deaths and is the greatest cause of severely disabled and vegetative survivors.

For CT scans to detect acute white matter shearing injury, there must be a haemorrhagic lesion. Most DAI lesions are small (often microscopic) and < 30% are haemorrhagic.[10] The CT scan findings that correlate with shearing injury are haemorrhages within the corpus callosum and/or the rostral brain stem or at the junction of the cortical grey / white matter.

3.6 EXTRADURAL HAEMATOMA (EDH)

This occurs in the potential space between the inner table of the skull and the dura matter. These two structures are normally tightly adherent from suture to suture. The blood comes from torn meningeal arteries or veins or from a laceration of the dural sinuses.

Traditionally, EDH[52] were separated into three categories, depending upon when they presented:

Acute - less than 24 hours, but up to 72 hours

Subacute - more than 24 hours and less than 10 days

Chronic - more than 10 days to 3 weeks.

In the majority of EDH the middle meningeal vessels are the cause, in the region of the squamous portion of the temporal bone. An EDH is more commonly found in the temporal and tempero-parietal regions (70%) than in frontal regions,(10 to 12%), but the latter often results in rapid neurological deterioration that requires urgent intervention.

An EDH has been reported to occur in 0.2% to 12% of all patients with head trauma, defined on the severity of the initial injury[52,53,54,55,56] The incidence peaks in the second and third decade of life, with < 6% occurring in patients > 60 years of age and < 3% occurring in patients > 3 years of age.[3]

An EDH is sometimes associated with other intracranial abnormalities, most commonly a contusion or SDH in 68%, and skull fracture in 82% of patients[57]. A SDH is much more common than EDH in the supratentorial regions, but an EDH is more common than SDH in the infratentorial region. 3% to 13% of EDHs are venous than arterial in origin [54], but these represent 25% to 38% of posterior fossa traumatic injuries.

The classic CT appearance of an acute EDH is a well-defined biconvex, high attenuation extra-axial mass that causes well-defined focal brain compression. Many extradural clots are now seen as mixed or even mainly low attenuated lesions. This is because the blood is still liquid, clot is still forming, and does not yet resemble the dense dehydrated mature haematoma.

The inhomogeneity of density within the haematoma is due to active clot formation and/or renewed bleeding, which is usually of no clinical significance. Norman et al.[58] studied the relative attenuation characteristics of venous properties of liquid and clotted blood, measured in Hounsfield units (HU). In this study 100% liquid blood measured 47.8 HU. 100% clotted blood measured 74.5 HU, and in 50/50 mixture of blood the density measured 61 HU.

A typical acute EDH measures 50 to 70 HU. Normal brain parenchyma measures - 30 HU. The variety of density patterns in acute EDH is related to the proportion of clotted and non-clotted blood.(23)

Unlike haemorrhage into the subdural space, a chronic EDH is rare. It shows a similar low or mixed attenuation pattern with dural marginal enhancement after contrast on CT scan. Rarely an EDH will develop as a delayed clot at a site previously unsuspected to be injured.

3.7 SUBDURAL HAEMATOMA (SDH)

Subdural haematoma is a collection of blood located above the arachnoid and beneath the inner layer of dura. More than 50% of SDHs are caused by traumatic disruption of the superficial cortical veins as they bridge over to major venous sinuses (59-60) In most cases a SDH accumulates only until the ICP rises to the level above the venous pressure.

Subdural haematomas are significantly more common after falls and assaults than after motor vehicle accidents, in which deeper tissue shearing injuries occur.(59). However, direct impact to the head is not necessary for the formation of SDH. Although a SDH is most commonly due to the tearing of the bridging cortical veins, it can also arise from a direct contusion to the underlying cerebral parenchyma, and if

associated with an adjacent intracerebral haematoma, the lesion is termed a "burst lobe". This is most commonly found in frontal and temporal poles.

Some 95% of acute SDH in the series of Forbes et al[61] were associated with significant mass effect upon the underlying tissue and lateral ventricles. The patient with bilateral (balanced) SDH is an exception and a typical pattern of ventricular compression and no midline shift is seen in these patients. These lesions are visible as bilateral collections. Unilateral SDH may cause a lesser degree of mass effect if there is underlying parenchymal volume loss e.g. due to old age, and alcoholism[27].

The presence or absence of associated parenchymal injury divides acute SDH into simple or complicated. A "simple" SDH occurs without underlying parenchymal injury and accounts for 45% of all acute SDH. A "complicated" SDH, with a more severe injury underlying cerebral injury. has been associated with a 53% mortality compared with the 21% of a simple SDH.[62]

A subdural haematoma is often associated with contusion, lacerations and intracerebral haematoma. The majority of these "complicated" lesions are located in the temporal and frontal regions. In these cases the superficial cortical vessels are often the source of the haemorrhage. The majority of simple SDH are located over the cerebral convexity, especially the parietal region.[63] However, SDH located beneath the

tentorium is relatively uncommon in adults, but occurs more commonly in neonates.(64)

Prefrontal and subtemporal SDH probably result from rupture of veins that enter the petrosal sinus, lateral sinus or cavernous sinus. Only six cases of clival SDH have been reported and all patients had an additional SDH in another location.(65)

Interhemispheric SDHs are usually unilateral and are produced by bleeding from bridging veins between superior sagittal sinus and the parieto-occipital cortex. The two hemispheric subdural spaces do not communicate across the midline except in open injury.(66)

On CT scan a typical acute SDH appears as a homogenous hyperdense crescentic mass located between the skull and the brain parenchyma. More than six hours is required for subdural blood to dissect around the hemisphere and assume its typical shape described above.(65) Subdural collections thicker than a few millimetres are ovoid rather than crescentic when viewed in coronal plane.

A posterior fossa SDH can appear quite subtle on CT scan. The cerebellum can appear slightly swollen and abnormally dense, with the increased attenuation being due to layering of blood over the cerebellar folia by the extra axial mass. SDH may be isodense or hypodense on CT in the acute phase. This is probably due to acute

anaemia and haemodilution, some times associated with resuscitation from multiple injuries, before clot formation is complete.

Considering densities of SDH collections on CT scan, Moler and Ericson[67] described that 25% of cases in their series (87 patients) showed an isodense lesion between one week and three months after injury, with a peak in the two to six weeks period following trauma. In such patients the diagnosis must rest upon the identification of associated mass effect.

3.8 INTRACEREBRAL HAEMATOMA (ICH)

A discrete haematoma within the brain substance, as distinct from contusion with diffusion of blood into the neural parenchyma, is considered to be an intracerebral haematoma. It is usually initiated at the moment of injury, but enlarges over time. It is therefore regarded as secondary phenomena and may lead to raised ICP, brain herniation and brain stem distortion.

Before the era of CT scan, Jamieson and Yelland[62] reported the incidence of ICH as 0.6% but this was limited to surgically evacuated cases. Zimmerman [23] reported an incidence of 4 - 23% on CT scan. An ICH can be caused either by direct rupture of a perforating cerebral vessel, or result from coalescence of contusional areas. Most ICH are located in the frontal and temporal lobes, 80 to 90% in the white

matter of the temporal and frontal lobes. In one study, 80% of ICH were unilateral, 20% were bilateral and 60% were associated with other lesion.(20)

An ICH reaches its maximum size within 12 hours in 84% of patients. An ICH may not be present on the admission scan but develop later. This is termed a delayed haematoma and it was originally thought to be rare phenomenon but after more patients were scanned several hours or even days after injury, delayed haematoma become more commonly detected.

The classical CT appearance of ICH is of high attenuation rounded or irregular collection of blood within the brain parenchyma (white) surrounded by oedema (dark); as the clot ages the oedema increases and clot become isodense.

3.9 INTRAVENTRICULAR HAEMORRHAGE (IVH)

An Intraventricular haemorrhage is observed after extension of an intracerebral haemorrhage into the ventricles. If the bleeding is minor, increased density is seen in the region of the occipital horns, with a fluid level. In extreme cases the entire ventricular system is filled with blood. A large intraventricular blood load frequently impedes the outflow of CSF and is associated with a lower admission GCS (68). Early signs are a circumscribed or generalised dilatation of individual ventricle segments or the entire ventricular system, with a decrease of the periventricular brain density.

The incidence of intraventricular haemorrhage is about 2 to 3%[69] but in patients with severe head injury the incidence is higher. Isolated traumatic ventricular haemorrhage are rare and attributable to rupture of ependymal vessels, it is seen more often in children than in adults.[69, 70]

3.10 SUBARCHNOID HAEMORRHAGE (SAH)

Subarchnoid haemorrhage is found in 25 to 33% of the severely head injured patients (68,71), It is most frequently seen in association with cerebral contusion, and skull base fracture.

In a review of 1,367 deaths due to blunt injuries to the head, a SAH was found in nearly all the autopsy specimens.[72] In 88% of these cases the haemorrhage was considered clinically insignificant and consisted of a tiny layer of venous blood, derived from injury to small cortical veins passing through the subarchnoid space. It is now evident that a worse outcome follows a head injury complicated by SAH, even if this is only the CT scan abnormality (34,73)

One possible serious consequence of SAH is that it might induce cerebral vasospasm. In one series data obtained during cerebral angiography for traumatic injuries demonstrated significant vasospasm in 5% to 31% of patients.[74] However, others indicated that SAH was not related to vasospasm. (75,76)

On CT, SAH appears as serpigenous, linear area of high attenuation conforming to the sulcal and cisternal spaces. Contusions might or might not be present. The sensitivity of CT in detecting SAH depends on the volume of the

extravascular blood, patient haematocrit and the time interval from injury, so it is uncommon for CT scan to detect SAH more than one week after injury.[77] A typical site for subarchnoid haemorrhage to accumulate after head injury is within the interpeduncular cistern.[78,79] Another common location is the Sylvian fissure.

The signs of the pathophysiological processes of H.I. on the CT scan are:

3.11 INTRACRANIAL PRESSURE (ICP)

ICP or CSF pressure is defined as a steady state level reference to atmosphere, the normal pressure is below 15 mmHg upon which there are superimposed cardiac and respiratory components. Becker (80) reported the high prevalence of raised ICP in head injury, a relationship to poor outcome and pointed out that in half of head injury fatalities, death finally occurred due to an uncontrollable increase in ICP. Raised ICP both indicates brain damage and, on its own account produces further brain dysfunction and damage by reducing cerebral blood flow (CBF) and promoting brain shift and herniation.

The measurement of ICP by a needle introduced into the CSF space was reported by Dawson.(81) An ICP pressure value of more than 20 - 30 mmHg is the most widely used range for initiating the therapy in head injured patients, a sustained ICP above 60 mmHg is almost always fatal. In head injury cerebral blood flow (CBF) is usually disturbed when the ICP is above 40 mmHg. An increase in ICP can occur with midline shift of the brain or with brain swelling. Basal cistern and III ventricle obliteration is related to elevation of ICP > 20 mmHg,(36) the third ventricle usually becomes obliterated before the basal cistern.(34)

The features on CT scan that predict increased ICP are ipsilateral ventricular compression, dilatation of the contra-lateral trigone and temporal horn, after the Foramen of Monoro is obliterated and a downward herniation leading to obliteration of the basal cisterns and / or third ventricle as a result of significant downward herniation (82). Isolated masses within the posterior fossa can cause acute obstructive hydrocephalus by compression of the fourth ventricle. The fourth ventricle should always be identified and to be central and symmetrical.

3.12 BRAIN SWELLING

Traumatic brain oedema is an increase in brain volume of all or part of the brain. Focal swelling is commonly related to a contusion or ICH. Unilateral hemispheric swelling is most commonly seen with an acute-cerebral collection usually a SDH. The diagnosis of a diffuse "swollen" brain should not be made from the size of the lateral ventricles because they have a wide range of normal size. The appearance of third ventricle and basal cistern should be the main criteria for a diagnosis of "swelling" leading to increased ICP, as used in the classification of Marshall et al. (37).

3.13 POST-TRAUMATIC ISCHAEMIA

The brain's oxygen requirement is enormously high. The brain represents only 2% of the body weight but it receives 15% of the total cardiac output. Oxygen supply to the brain can be decreased by a reduction in blood oxygen content (hypoxia) or because of impaired tissue perfusion (ischaemia) (83). The CNS vasculature has

two unique properties that protect the brain from ischaemia. The first is autoregulation and the second is chemoregulation. Head injury can result in a loss of cerebral autoregulation and sometimes chemoregulation, and the injured area becomes a pressure-passive zone which leads to cerebral ischaemia, cerebral oedema and intracranial haemorrhage if an episode of arterial hypotension occurs.

Brain ischaemia is common in fatal head injury and pathologically the severity of ischaemic brain damage is graded as being severe in 27% of cases, moderately severe in 73% and mild in 30%. (27) Signs of ischaemia are rarely seen on the CT scan in life.(20,72) The patterns seen at autopsy are focal, multifocal, boundaryzone.

An SDH is often associated with the rapid onset of mass effect and vascular compromise (occlusion or spasm) can cause diffuse low attenuation on the CT scan on the same side as the lesion.

Post-traumatic vasospasm predisposes the injured brain to watershed infarction. Occlusion of the distal branches of ACA, MCA and PCA is potentially more dangerous than occlusion of ICA or vertebral arteries. Post-traumatic infarction caused by cerebral vasospasm has been reported to occur in 5% to 7% of patients after head injury.

Vasospasm typically causes ischaemic cortical injury and generally spares the deep white matter.(10)

The changes in the brain that result from hypoxia contribution to further cerebral swelling and exacerbate elevated intracranial pressure (ICP). As ischaemic infarction in general is due to interruption of the blood supply to a portion of the brain, the CT finding within the first few hours is usually unremarkable and seen as low attenuation area giving darker appearance than the usual brain. Arterial vascular territory ischaemia is the most common recognised ischaemic complication on CT scan.

The earliest manifestations of ischaemia on CT (six hours post-traumatic) include a subtle loss of grey matter-white matter differentiation, mild mass effect and a slight decrease in attenuation value. Within eight hours the manifestation becomes more defined and a further decrease in attenuation values takes a wedge-shaped area. The density of the lesion becomes progressively lower over the succeeding weeks. Petechial haemorrhage can be seen in the subacute stage (1-2 weeks) and causes the infarct to appear slightly heterogeneous. There appears to be a direct relationship between the size of the ischaemic area and the probability of haemorrhagic transformation of the infarct.(79)

CHAPTER IV

OBSERVER VARIABILITY

4.1 Introduction

An assessment of an investigation is usually based on considerations of accuracy, sensitivity and specificity . However the clinical utility of an imaging study also depends on its reliability or the consistency with which the study is interpreted in the same way either on different occasions or by different observers (84). The consistency of observation made by different observers in interpreting the same study or a group of studies is termed "interobserver reliability or agreement" (84,85) , its converse is termed "interobserver variability" (86) . Consistency of observation made by a single observer of the same material on different occasions is known as intra-observer reliability. Although the accuracy of CT in detecting traumatic intracranial haematoma has been determined (16) , the interobserver reliability of CT across a range of traumatic brain injury has not been measured .

Inter-observer variation has been of concern for several decades. A major impetus to awareness of the phenomenon was a study in the late 1940s by Yerushalmy (87), which compared the value of different types of chest x-rays for mass screening purposes. In the years since this study, the phenomenon of variability among observers, as well as inconsistency by the same observer, has been documented in many

settings and has become well recognized in medical observations, including radiological observation. Discrepancies between different observers are to be expected. These can be diminished but probably never absolutely eliminated. Clinically observers variability studies played an important role in the developing Glasgow Coma Scale (88, 89,90, 91, 92, & 93), and in judging the reliability of classifications of subarachnoid haemorrhage (94). Neuroradiological studies of observer variability include grading the amount of blood on computed tomogram after subarchnoid haemorrhage (95).

The importance of observer variability in reporting CT scans of head injuries includes communication between different practitioners, leading to improved outcome because of appropriate management. Observer variability is also important in research.

Inter-observer variation may arise from several sources : an observer may miss an abnormality or think he has found one where none is present, observers may disagree about what is normal or, conversely abnormal, this may be due to unclear definitions of the pathology present on the CT scan and hence a poor classification. Observer variability is influenced also by the ability of observers and their training, and by the type and quality of the scan .

Improvement in the scanning technology has been going on since the first scan machine EMI 1010 in 1973, The imaging of the cases in this study was carried out with the Philips tomoscan.

4.2 PATIENTS

Thirty patients with different severity of head injury, all of whom were transferred to the Neurosurgery Unit in the Institute of Neurological Sciences in Glasgow between 1985 and 1990, were chosen for this study, and only the files 23 patients been traced and reviewed.

The head injury was due in 9 cases to a road traffic accident, in 10 cases to a fall (two of them because of epilepsy), 3 cases were due to assault, , and one case was due to hitting a wall.

When admitted to the neurosurgical unit, 7 patients had a GCS of 8 or less i.e. severely injured, 7 patients scored between 9 and 12 i.e. moderately injured, 4 patients scored 13 or more i.e. mildly injured, 5 patients were intubated and could not be scored..

16 patients had one CT scan and 7 had more than one CT scan.

9 patients underwent an operation and 14 cases had no indication for surgical treatment.

4.3 METHOD

Thirty CT scans of the previously discussed patients were collected, Their CT scans were extracted from files and copies were made for use in this study. The CT scans had been taken on the Philips tomoscan machine in the form of ten to fifteen axial non contrast slices, as discussed in chapter two. Two CT scans were eliminated one was of poor quality and the other one was a CT scan of stroke rather than head injury, leaving 28 CT scans from patients with a closed head injury.

4.3.1 OBSERVERS

13 observers (Table 4) were deliberately selected to have different levels of experience, and to represent different specialties within the Neurosciences. In addition one of the observers was a senior consultant from Netherlands and the other was a consultant trained in Germany Observers were chosen in this way to investigate the influence of experience, because many acute decisions depend upon senior-house officers or registrars observation of CT scan findings, as well as review by senior colleagues.

The participants were asked to code their observations on each CT scan separately on a detailed proforma , covering most of the specific intracranial pathological lesions known to result from a head injury. We also included the commonly used methods of classification discussed in chapter II

TABLE 4

THE OBSERVERS

Consultant	**4**	One Neuroradiology
Senior Registrar (Neurosurgery)	**3**	
Senior Registrar (Neuroradiology)	**1**	
Registrar Neurosurgery	**3**	
Registrar Neuroradiology	**1**	
Senior House Officer	**1**	
	13	

A) The observers were required first to decide if the CT scan was normal, if it showed a surgical mass lesion or if it showed other abnormalities, either with or without signs of raised intracranial pressure.

B) Scans were also coded by using classification of the Traumatic Coma Data Bank (37).

C) The specific lesions included in the proforma were mass lesion or contusions, their numbers and sites, using the classification employed in the European study of nimodipine in head injuries (71).

D) CT signs indicating raised intracranial pressure (midline shift, the state of mesencephalic cisterns and the third ventricle) were included .

E) Subarachnoid haemorrhage was coded using the classification employed in the European study of nimodipine in head injury, by subarachnoid classification described by Fisher (96), and also by a detailed classification of subarachnoid haemorrhage designed for the purpose of this study.

F) The state of the lateral ventricles and the presence of blood within the ventricular system was also included in the proforma.

4.4 ANALYSIS

There are two main kinds of observers variability, First is the amount that observers vary from one to another when reporting on the same scan, the focus of this study . Second is the amount one observers varies, when he /she reports more then once on the same scan, this is less important in the current context.

The overall agreement is the proportion of occasions in which observers agree on the presence or absence of an abnormality. Specific agreement is the proportion of patients about whom observers agree on the presence of an abnormality, that is the conditional probability that all observers will report an abnormality present , given that one observer did so.

There is no gold standard against which one might make a comparison and observers variation is usually assessed by looking at all possible observers pairs. As Spitzer and Fleiss (97) pointed out that the agreement rate of two equally skilled physicians regarding the presence of an abnormality in a series of cases is in part a function of the proportion of cases each physician consider abnormal, if two physician each consider half of the cases abnormal they will agree 25% of the time by chance alone , if each considers 80% of the cases abnormal, they will agree 64% of the time by chance alone. Since the proportion of cases considered abnormal varies across studies, the level of agreement expected by chance varies across studies . Thus an overall agreement rate of 80% in one study may be 55% greater than expected by chance , and an overall agreement rate of 30% in another may also be 55% more than chance.

Because of the effect of chance on observer agreement , a statistic has been developed that takes in account of the contribution of chance agreement (98) . It can be thought of as the proportion of the observed agreement (corrected by chance) divided by the maximum possible agreement in excess of chance (99,100, 101), and is widely used in analysis.

$$K = (Po - Pc) / (1 - Pc)$$

Where Po is the observed proportion of agreement.

Pc is the chance expected proportion of agreement .

The Kappa is zero if the agreement of observers is equal to that due to chance .

Kappa is one (1.0) equal perfect agreement between observers .

Kappa of (-1.0) would equal perfect disagreement between observers

Kappa between zero and one indicates that there is more agreement than is to be expected from chance , The null hypothesis that the physicians only agreed by chance was tested by means of a standard normal value Z (100).

Z value corresponds with a tail probability smaller than 0.02, meaning that each Kappa is significantly greater than 0.0. In otherwords, the observed proportion of agreement Po is significantly greater than chance proportion of agreement Pc.

The P values measures the strength of evidence in scientific studies by indicating the probability that a result at least as extreme as that would occur by chance.

When, Kappa is significantly greater than chance , the degree of variability or conversely agreement is to some extent reflected in the value of Kappa. Unfortunately, the absolute value obtained for Kappa in a study is influenced by a number of factors such as the number of patients included and the observers participating, thus a universally accepted subdivision of Kappa score has not been established . We have chosen, partly arbitrarily, partly in relation to the values we found and partly in the light of previous work, to refer to the degree of agreement, we found if significant as either low, medium, or high, equated with Kappa values of < 0.4, between 0.4 and 0.6, and > 0.6.

4.5 RESULTS

We found that overall, observers usually agreed more than could be expected by chance in the assessment of most of the CT scan features of head injured patients. Nevertheless, the scoring given by the observers showed differences in almost every case. Sometimes the differences were small but in other cases, less commonly, conflicting, even completely contradictory, observations were recorded. Some observations, over the series of patients, appeared to provoke relatively more disagreement than others. When an observation could be assigned to one of three or more choices, there was considerable disagreement.

4.5.1 Classification of CT scan features.

4.5.1.1 Major diagnosis

Table 5, shows that the assignment of the CT scan to one of the four major categories (normal CT scan; abnormal with a surgical mass lesion; abnormal without or with signs of raised intracranial pressure) produced significant agreement. However, the overall Kappa value 0.51 was only moderate. The observation most frequently recorded was of a surgical mass lesion, noted in 41% of recordings and associated with a relatively high Kappa value of 0.71 Although only 11% of observations assigned the CT scan to the normal category, this was associated with the next highest Kappa value,0.60. Although an abnormal scan without signs of raised intracranial pressure was the second highest recorded, its Kappa value was lower 0.41. Relatively few scans were assigned to the category of abnormal with raised intracranial pressure, but this was associated with the lowest Kappa value of all (0.14).

TABLE 5

MAJOR DIAGNOSIS

Major Diagnosis	No. Of Observations	%	Kappa Value	Z Value
Normal CT scan	31	0.11	0.60	8.11
Surgical Mass Lesion	115	0.41	0.71	3.80
Abnormal No SML, No Raised ICP	110	0.40	0.41	2.25
Abnormal No SML, Raised ICP	22	0.08	0.14	2.18

The overall results of Major Diagnosis

No. of observations	278
Kappa	0.51
Z	17.38
P	< 0.0010.

These findings may indicate that a particular source of disagreement is the distinction between the presence or absence of raised intracranial pressure in patients with an abnormal scan.

4.5.1.2 Traumatic Coma Data Bank Classification

Table 6 shows that overall the assignment of scans to the Traumatic Coma Data Bank classification had a moderate degree of agreement, the Kappa value of 0.56, being similar to that for the major diagnosis considered previously. This agreement was highly significant and greater than chance as (Z value was 26.23). Again the most frequent observation was of a surgical mass lesion (in approximately 32% of observations) and this was associated with a very high Kappa value 0.87. The number of recordings of normal appearances was very similar to that in the preceding section but in the context of the Traumatic Coma Data Bank, was associated with a higher Kappa of 0.73. The assignment to abnormal, with brain swelling 8.9%, although significant was again associated with a Kappa 0.44 indicative of only a moderate degree of agreement, and abnormal without swelling was second most frequently recorded 30.26% and had (Kappa 0.44 and Z 3.00). Diffuse injury with a shift was not recorded on any occasion. Some 15.45% of observations were assigned to an unevacuated surgical mass lesion and this was associated with a very low rate of agreement 0.16.

TABLE 6

TRAUMATIC COMA DATA BANK RESULTS

Traumatic Coma Data Bank Classification	No. Of Observations	Kappa Value	Z. Value
DI. I. (Normal)	36	0.73	8.82
DI. II	82	0.44	3.00
DI. III (swelling)	24	0.44	6.46
DI. IV (shift)	0	-	-
Evacuated Mass Lesion	87	0.87	5.66
Non Evacuated Mass Lesion	42	0.16	1.79

The overall results of TCDB.

No. of observations	272
Kappa	0.56
Z	26.23
P	< 0.0010.

Because the majority of observations in this classification are of either category 2 or 5, these have been further subdivided. Thus, diffuse injury without swelling can be subdivided into a single lesion or multiple lesion, and multiple lesions as either unilateral or bilateral (Table 6a). We have found that overall this further distinction produced a very low Kappa value 0.034 not significant. This may reflect a difficulty in distinguishing between single and multiple unilateral lesions because bilateral lesions were associated with a relatively high Kappa value. Intraparenchymal lesions can also be assigned as superficial , deep or present in both locations (Table 6b). This distinction in the patients studied, was of limited value because few patients (9) had only deep lesions. Overall the Kappa value was 0.47 and this was significant. The assessment of scans showing both superficial and deep lesions was associated with a relatively high (Kappa value 0.72 and Z value was 1.98), whereas that associated with observations of superficial lesions was only moderate (Kappa value of 0.40 and Z value of 0.95). The data suggests that distinction simply between two categories, superficial only and deep lesions with or without superficial lesions may prove preferable.

TABLE 6a

SUBDIVISIONS OF DIFFUSE INJURY

TCDB Classification (DI II)	No. Of Observations	Kappa Value	Z Value
Single	31	0.19	0.48
Multi: Unilateral	16	0.03	0.15
Multi: Bilateral	26	1.01	3.06

The overall results.

No. of observations	73
Kappa	0.44
Z	7.41
P	< 0.001

TABLE 6b

SUBDIVISIONS OF DIFFUSE INJURY

TCDB Classification (DI II)	No.Of Observations	Kappa Value	Z Value
Superficial	35	0.40	0.95
Deep	9	0.04	0.24
Both	30	0.72	1.98

The overall results

No. of observations	74
Kappa	0.47
Z	6.61
P	< 0.001.

4.5.2 Types of haematoma and contusion.

The identification of either an extradural or subdural haematoma was made respectively on 36 and 33 occasions and produced high Kappa values 0.94 and 1.295, Z value was 2.40 and 3.59 respectively. An intracerebral haematoma was recorded on only 12 occasions and was associated with a low Kappa of 0.24. Z was 1.38 (Table6c). When the more complex system of assessment of the specific location of the haematoma was employed (Table 7) lower rates of agreement were found, with only moderate, Kappa values, ranging from 0.30 or 0.40 to 0.60 for subdural and extradural haematoma. In the subtentorial region, posterior fossa lesions were scored too infrequently for meaningful values.

Within the Traumatic Coma Data Bank system (Table 6c), only on 12 occasions was an intracerebral haematoma recorded, with a low Kappa value of 0.24, and identification of the signs of an intracerebral haematoma, in a detailed breakdown (Table 7) produced Kappa values ranging from 0.20 to 0.35.

The presence or absence of cortical contusions in different locations (Table 7) also produced only moderately degrees of agreement with values ranging from 0.04 to 0.51 for right or left regions, with again the occurrence of observations of a posterior fossa contusion to be too infrequent for meaningful analysis.

TABLE 6c

TYPES OF HAEMATOMA

Evacuated Mass Lesion	No. Of Observations	Kappa Value	Z. Value
Extradural Haematoma	36	0.94	2.40
Subdural Haematoma	33	1.30	3.59
Intracerebral Haematoma	12	0.24	1.38

The overall results of Evacuated Mass Lesion

No. of observations	81
Kappa	0.94
Z	16.36
P	< 0.001

TABLE 7

(HIT II) HAEMATOMA VERSUS CONTUSIONS

HIT II	No. Of Observations	Kappa Value	P Value
EDH Rt	276	0.48	< 0.0100
EDH Lt	276	0.60	< 0.0010
EDH PF	276	0.02	< 0.9900
SDH Rt	276	0.30	< 0.0500
SDH Lt	276	0.56	< 0.0010
SDH PF	276	0.09	< 0.9900
ICH Rt	277	0.35	< 0.0010
ICH Lt	276	0.20	< 0.1000
ICH PF	276	0.34	< 0.9900
CONT. NO. Rt	276	0.51	< 0.0010
CONT. Lt	275	0.22	< 0.0500
CONT. PF	276	0.04	< 0.9900
CONT. Rt	276	0.21	< 0.9900
CONT. Lt	276	0.29	< 0.3000
CONT. PF	276	0.02	< 0.9900

4.5.3 Midline shift

Table 8, overall midline shift was recorded on 104 occasions 37.68% of the observations, with over half of these being of a marked degree. There was overall a relatively high agreement about the presence and extent of shift, Kappa was 0.61 , and Z value was 11.72. The presence of a marked degree of shift was associated with a particularly high Kappa value 0.80 and Z value of 7.25. Lesser degrees of shift produced a relatively low level of agreement Kappa value 0.31 and Z value 3.17.

To assess the value of a binary division, the observation were collapsed. Combining non or minimal mid-line shift < 5mm , and distinguishing mid-line shift more than 5 mm led to Kappa value of 0.77.

Comparison of no mid-line shift with any degree of mid-line shift led to a Kappa value of 0.66.

TABLE 8

MID-LINE SHIFT

Mid-Line Shift	No. Of Observations	Kappa Value	Z Value
No M-L shift	172	0.66	2.24
< 5 mm	47	0.31	3.17
> 5 mm	57	0.80	7.25

The overall results of Mid-Line shift

No. of observations	276
Kappa	0.61
Z	11.72
P	< 0.0010.

4.5.4 CSF spaces

4.5.4.1 The basal cisterns

Table 9 were recorded to be compressed or obliterated on 106 occasions 37.68% of observations. There was however, overall only a moderate degree of agreement Kappa value was 0.49 and Z value was 9.70. This reflected the relatively low Kappa value 0.16 and Z value 1.51 for recording of compressed cisterns.

By contrast, relatively higher Kappa values were obtained for both normal cisternal appearances (Kappa value 0.59, Z value 2.06), and for obliterated cisterns (Kappa value 0.65 and Z value 5.99).

4.5.4.2 The third ventricle

Table 9 The body of the third ventricle was considered to be abnormal on 118 occasions 42.75%. The assessment of third ventricle appearances was associated overall with a high degree of agreement (Kappa 0.65 and Z value 15.35). As with the basal cisterns, agreement was high about the most abnormal appearance, obliteration (Kappa 0.81 and Z value 7.07), but also was considerably greater for intermediate abnormality compressed (Kappa 0.45 and Z value 4.02), than for the basal cistern appearances.

TABLE 9

APPEARANCES OF CEREBROSPINAL FLUID SPACES

	Basal Cisterns			Body Of The Third Ventricle		
CSF Spaces	**No. Of Observ-ations**	**K Value**	**Z Value**	**No. Of Observ-ations**	**K Value**	**Z Value**
Normal	169	0.59	2.06	158	0.69	2.61
Compressed	51	0.16	1.51	58	0.45	4.02
Obliterated	55	0.65	5099	60	0.81	7.07

The overall results

	B.S.	III V.
No. of observations	275	276
Kappa	0.49	0.65
Z	9.70	15.35
P	< 0.001	

4.5.4.3 The lateral ventricles

Table 10, The lateral ventricles were considered to show abnormalities with the size or symmetry in almost half the cases. Overall there was a moderate degree of agreement about the assessment of lateral ventricles (Kappa value 0.45 and Z value 8.98) for the right ventricle and (Kappa value 0.52 and Z value 14.75) for the left ventricle, however low values of Kappa were associated with the infrequently recorded findings of a compressed or dilated ventricle relative to the contralateral.

4.5.6 Intraventricular haemorrhage

Table 11 Intraventricular haemorrhage was assessed to be present on only 38 occasions 13.77% of the observation. There was a moderate degree of agreement overall, (Kappa value 0.53 and Z value 3.85), with absence of Intraventricular haemorrhage having a (Kappa value 0.63 and Z value 1.09) and assignment of CT scan to the presence of blood in more than one ventricle produced moderate to good agreement (Kappa value 0.57 and Z value 9.46).

TABLE 10

APPEARANCES OF LATERAL VENTRICLES

	Rt. Ventricle			Lt. Ventricle		
LATERAL VENTRICLES	**No. Of Observ-ations**	**K Value**	**Z Value**	**No. Of Observ-ations**	**K Value**	**Z Value**
Normal	164	0.48	1.72	137	0.54	2.37
Compressed relative to normal	62	0.48	4.12	96	0.55	3.96
Compressed relative to contralateral	7	0.04	0.51	16	0.18	3.06
Dilated relative to normal	30	0.56	7.52	21	0.38	5.95
Dilated relative to contralateral	12	0.13	2.06	5	0.16	1.80

The overall results

	Rt.V.	Lt.V.
No. of observations	275	275
Kappa	0.45	0.52
Z	8.98	14.75
P	< 0.001	

TABLE 11

APPEARANCES OF OF INTRAVENTRICULAR HAEMORRHAGE

Intraventricular Haemorrhage	No. Of Observations	Kappa Value	Z Value
No	238	0.63	1.09
One ventricle	20	0.31	4.94
> one ventricle	18	0.57	9.46

Overall calculations

No. of observations	276
Kappa	0.53
Z	3.85
P	< 0.001.

4.5.5 Subarachnoid haemorrhage

Observer agreement, although significant, was relatively low about the presence and distribution of traumatic subarachnoid haemorrhage, Using Fisher classification Table 12 of subarachnoid haemorrhage the overall Kappa value was 0.31, and by using the classification employed in the European Study of Nimodipine in Head Injury (HIT II) (Table 13), the Kappa value was 0.29 and Z value was 5.35, subarachnoid haemorrhage was recorded in approximately 38.4% of cases and considered to be present in 106 assessments, 70 observations recorded a focal subarachnoid haemorrhage with a Kappa of only 0.09 and Z value of 1.03. Using the detailed assessment schedule for assessment of the distribution of subarachnoid haemorrhage, devised for this study (Table 14), a similar low degree of agreement was observed (overall Kappa 0.27 and Z value 7.73). Recordings of the distribution focal cortical. SAH were associated with a particularly low level of agreement

(Kappa value of 0.08 and Z value of 1.25).

TABLE 12

APPEARANCES OF SUBARACHNOID HAEMORRHAGE UISING "FISHER CT CLASSIFICATION"

Fisher CT Classification	No. Of Observations	Kappa Value	Z Value
No SAH	172	0.41	1.35
Diffuse or thin, no vertical layer > 1mm	62	0.33	2.74
Clots or vertical layer > 1mm	17	0.06	0.94
Intracerebral or Intraventricular clots	19	0.16	2.59

The overall calculation

No. of observations	270
Kappa	0.31
Z	5.49
P	< 0.001.

TABLE 13

APPEARANCES OF SUBARACHNOID HAEMORRHAGE UISING (HIT II)

SUBARACHNOID HAEMORRHAGE	No. Of Observations	Kappa Value	Z Value
No SAH	170	0.39	1.35
Diffuse SAH	47	0.36	3.68
Focal SAH	70	0.09	1.03
Blood at tent.	19	0.12	1.99

The overall calculation

No. of observations	276
Kappa	0.29
Z	5.35
P	< 0.001.

TABLE 14

APPEARANCES OF SUBARCHNOID HAEMORRHAGE USING "GLASGOW INSTITUTE OF NEUROLOGICAL SCIENCES CLASSIFICATION"

GINS Classification	No. Of Observations	Kappa Value	Z Value
No SAH	170	0.39	1.36
Isolated tentorial blood	17	0.11	1.85
Focal basal SAH	9	0.22	3.26
Diffuse basal SAH	14	0.13	2.11
Focal cortical SAH (sylvian or sulcui)	24	0.08	1.25
Diffuse cortical SAH	20	0.37	5.88
(3 or 4)+(5 or 6)	21	0.23	3.65

The overall calculation

No. of observations	275
Kappa	0.27
Z	7.73
P	< 0.001.

4.5.7 Observer experience

Comparison of the Kappa values obtained using the TCDB classification associated with individual observers, grouped according to seniority (Table 15), showed moderate levels of inter-individual agreement. The Kappa values were lower for the group of six registrars than the group of seven consultant or senior registrars 0.50 as against 0.71, and also Z value 3.27 and 2.57 . The group of three neuroradiologists (one consultant; one senior registrar; one registrar), showed a relatively high level of agreement Kappa value was 0.65 and Z value was 1.83, as compared with the group of 10 surgeons (three consultants; three senior registrars; three registrars and one senior house officer) Kappa value was 0.42 and Z value was 2.43.

TABLE 15

OBSERVER EXPERIENCE

The Observers	No. Of Observations	Kappa Value	Z Value
Consultant / SR	7	0.71	3.27
Registrar	6	0.50	2.57
Surgeon	10	0.42	2.43
Radiologist	3	0.65	1.83

4.6 DISCUSSION :

Differences in findings due to variation among observers is an inescapable part of medical practice. There is no single method for analyzing the results of studies of observer variability. The method that we employed in this study is widely accepted and has two essential advantages , it is not invalidated by missing data an inevitable complication of a study using so many observers , and it facilitates comparisons between scales with different numbers of components .

Although the use of the CT scan in classification of head injuries has been presented in number of studies (37) to the best of our knowledge the inter-observer reliability in detailed classification of CT scan of head injury in classification has not been measured previously.

Although Kappa is widely accepted as the most appropriate statistic for measuring inter-observer agreement or reliability, it has also been recognized that it over compensates for chance agreement by giving the observers no credit for determining the appropriate frequency of abnormal findings in the group being studied(102).

Kappa varies from -1.0 (perfect disagreement) through 0.0 (chance disagreement) and 0.99 (perfect agreement) (98). A specific value has been agreed upon as being indicative of significant or satisfactory inter-observer agreement .(103,104,105)

Landis and Koch (103) suggested that a Kappa value of 0.81 indicates "almost perfect agreement" , while values between 0.61 and 0.80 are "substantial" between 0.41 and 0.60 "moderate" , and between 0.21 and 0.40 "fair".

This is comparable to the approach in the present study in which values < 0.4 were regard as low , between 0.4 and 0.6 as moderate, and > 0.6 as high.

Nargard et al (105) considered that Kappa values of more than 0.75 are excellent and those ranging from 0.40 to 0.75 are fair to good .Generally speaking, the Kappa value found in studies of imaging procedures have been in the fair to good range depending on the findings or signs being evaluated (105,102).

An important consideration in understanding the significance of a measure of inter-observer variability in assessing an imaging study is the conspicuity or obviousness of the finding or abnormality being assessed . The more straight forward the assessed finding is, the less subject it is to varying interpretation by different observers. On the other hand , we would expect the inter-observer agreement for diagnosis of a less straight forward finding to be lower (105). In this study the abnormalities assessed varied in their ease of diagnosis. It seems likely that the finding of a surgical mass lesion is relatively conspicuous and is diagnosed with more agreement than more subtle abnormalities e.g. CT scan with raised intracranial pressure. Thus the Kappa value for mass lesion was 0.71 and that for abnormal CT scan with raised intracranial pressure was only 0.14.

The overall kappa for the "major diagnosis" was moderate with the lowest agreement Kappa 0.14 recorded for the category "abnormal CT scan with no mass lesion and with signs of high ICP". This may be attributed to the low number of observation (Number of observations was 22), but even with this small number of observations the Kappa value is significant as the Z value was 2.18 . The agreement about midline shift in this study was good Kappa value 0.61 , and there was higher agreement between the observers in scoring the Midline shift more than 5mm Kappa value 0.88 than the scoring the Midline shift less than 5mm Kappa value 0.31 .

There is moderate agreement between observers using TCDB classification and it was highly significant as Z value 26.23. However the Kappa value for DI III (swelling) showed only moderate agreements Kappa value 0.44 . The agreement between the observers about the state of the basal cisterns also was only moderate Kappa value 0.49; it was good for the obliterated basal cisterns Kappa value 0.65 , but much less for compressed Kappa value 0.16.

The agreement between observers was high when they judged the third ventricle Kapppa value 0.65 higher than for the basal cisterns. Observers agreed highly on an obliterated third ventricle Kappa value 0.81 , but less for compression Kappa value 0.45 .

The agreement about SAH using different classifications was only fair. Agreement was better for IVH Kappa value 0.53, and even more high for the presence of blood in more than one ventricle Kappa value 0.57 and Z value 9.46.

The low interobserver agreement in grading SAH using the more detailed classification, probably attributed to the complexity of descriptions. It is difficult by CT scan to identify small amounts of blood in different anatomical locations in the same slice. The tentorium cerebelli and posterior Interhemispheric fissure may normally have a slightly increased density on a non enhanced CT scan and this should not be mistaken for subarachnoid blood , CT scan assignment of SAH should be made without a too detailed discreption.

Agreement regarding normality was usually greater than agreement about abnormality, and agreement for dichotomous judgments (present/absent) usually higher than judgments about continuous or qualitative multiple (94,97,98) , although this not so always (100) . In this study, the Kappa value for diffuse injury II in TCDB classification (no pathological lesion is seen) was 0.73, where as for detailed classification of subarachnoid haemorrhage the Kappa value was low 0.27 .

Pairs of physicians with more training relevant to the test task will agree more often than pairs of observer physicians with less training (94,85) . However and perhaps reassuringly in this study the Kappa values for juniors were not far from the most senior staff, not surprisingly radiologists have a higher Kappa value 0.65 than the others 0.42.

The observation of the head injuries that have adequate reliability are the ones with less detailed subdivision and with clear definition the major diagnosis ,

Traumatic Coma Data Bank classification ,mid-line shift , third ventricles , interventricular haemorrhage . However for subarachnoid haemorrhage the Kappa value was 0.29, what ever system was used.

The present study clearly documents that attention to the observer variability problem is essential in clinical management investigation of patients with closed head injury. The reliability of CT scans in diagnosis of head injuries could be improved if the proforma for reporting the CT scan followed standardized protocols, by careful definition of the lesion and also by supplying atlases of the different lesions which can be seen on the CT scans of head injured patients .

CHAPTER V

COMPARISON OF FINDINGS ON COMPUTED TOMOGRAPHY AND NEURO-PATHOLOGY

5.1. Introduction

Computer Tomography scanning has the ability to provide rapid, non-invasive evaluation of the pathophysiological effects of head injury, It guides early medical and surgical intervention and since its introduction the outcome of patients has improved (26). The morbidity and the mortality rates have been lowered in patients who have been studied by CT scan compared with patients evaluated before CT scans became available (5).

Clinicopathological reports on human closed head injury are common, but the correlation between CT scan findings on head injury and post-mortem pathological findings has been rarely reported. Surprisingly little work so far has been done in this area. Only one study has been published in 1989, from Barcelona Spain, which reviewed the initial CT scans of 24 patients with a severe head injury in whom the post-mortem examination demonstrated signs of diffuse axonal injury . The study supported the suggestion by other authors that DAI is a frequent finding in patients with severe head injury. They concluded that wider and more extensive studies are needed to

differentiate between cases with severe primary DAI and patients in which DAI is not present (12).

For this thesis the findings in CT of a series of head injured patients who later died were compared with the post-mortem pathological findings of the same patient. The CT scan was analyzed in relation to features such as mass lesion, midline shift, CSF spaces, subarachnoid haemorrhage and ischaemic infarction, and using classifications of head injury into mass lesion and diffuse injury , and also with a classification systems used in previous section the "Major Diagnosis" and Traumatic Coma Data Bank classifications (37).

The opportunity for this comparison study presented in Glasgow, because a consecutive series of severely head injured cases, who had been transferred to neurosurgery unit alive but later died, had received a comprehensive post-mortem neuropathological study.

5.2. Cases

102 patients were studied who had died due to head trauma between 1987 and 1989, after having been transferred to the neurosurgical unite. The regional NSU in Glasgow provides the only service covering a population of 2.7 million in the West of Scotland and accepts patients with head injuries after they have been initially dealt with by other clinical services . More than 500 head injured patients are transferred each year to this NSU. Annually some 50 patients die in the NSU because of head trauma, constituting 10% of the admitted cases (106).

5.3 Features of series

5.3.1 Age

The number of cases are distributed fairly evenly through the ages between new-born and 77 years of age. The mean age was 35.17

5.3.2 Sex

Of the 102 Patients included in this study, 69 were male (67.6%) , and 33 were female (32.4%), with the ratio of males to females being nearly 2:1.

5.3.3 Cause

The cause of head injury varied, in 49 patients it was due to a road traffic accident, in 46 patients due to fall and in the rest due to other causes.

Table 16

FEATURES OF THE CASES

Total	**102**
Age Mean	**35**
Sex	
Male	**69**
Female	**33**
Cause of injury - RTA	**50**
GCS ≤ 8 Admission	**71**
NSU	**92**
Haematoma evacuated	**40**
Time injury to death	
> 1 day	**31**
2-7 days	**52**
8-28 days	**19**
Number of scans	
1 scan	**63**
≥ 2 scans	**39**

5.3.4 Clinical features

The severity of the injury was evaluated using the Glasgow Coma Score, where severe head injury is defined as a patient who scores 8 or less, moderate head injury as a patient who scores between 9 and 12, and a mild injury when a patient scores 13 or more (107). The GCS at the referral hospitals showed that 69.6% of the cases had suffered a severe head injury, 21.6% a mild head injury and only 6.9% a moderate injury. In 2% of cases a GCS total score could not be given because the patients had been intubated or was dysphasic, etc.

The GCS was assessed after admission to the Neurosurgery Unit, showed that, now, 92% cases were classified as a severe head injury, only 3.9% cases as a mild head injury and 5.9% as a moderate head injury . None of the cases which were included in this study were patients who had died on arrival to the Neurosurgery Unit, Most of the cases (63.4%) had not spoken after the injury, in the remainder who had a "lucid interval" 21.4% were recorded to have been confused and 15.2% were orientated.

63 patients had one CT scan and 39 patients had more than on CT scan

40 cases were operated for evacuation of a haematoma.

5.3.5 Time of death

31 patients lived for more than one day, 52 patients between 2 - 7 days, and 19 patients lived between 8 - 28 days.

5.4 CT scan Interpretation

The CT scans of the 102 patients were collected, as well as the worst of the follow up scans of the 39 patients with more than one scan. The CT were scans performed on a Philips 350 tomoscan with 6mm contiguous slices. For those patients with only initial CT scan film, these features were studied, and for those with a second scan 39 , the initial and follow-up CT scans were compared and the worst appearances were analysed. The differences between the first and worst in these cases are described subsequently.

Comparison between CT and Pathology are based on the admission scan in those with only one scan and worst in those with two scans. Each CT scan was assessed by my self and an experienced neuroradiological consultant and the findings were recorded. The two set of observations were compared and discussed until an agreement was reached about the findings in each case.

5.5 METHODS

5.5.1 CT Method

163 cases underwent post-mortem pathological examination. However only 102 of these cases were included in the study, the other patients were eliminated because their CT scan films could not be traced by the department of Neuroradiology.

The coding of each CT scan was summarised on the proforma that was used in the observer variability study, where the CT scan was interpreted in respect of the presence of focal lesions, number, size and location, TCDB classification, SAH, and brain ischaemia. The CT scan coding performas of the 102 patients were entered in the main frame computer of Glasgow University.

5.5.2 Pathological Method

The post-mortem examinations were performed by a Senior Consultant in the Glasgow Institute of Neurological Sciences. All the PM pathological reports of the patients enrolled in our study were reviewed by my self. The results of the post-mortem pathological studies were coded on a proforma and entered to the main frame computer of Glasgow University.

5.5.3 Techniques

The brain was removed by a neuropathologist who ascertained the state of the dura and the presence of either extradural or subdural blood. The skull was cut immediately above the supraorbital ridge, horizontally, and with the help of the malleable retractor, the dura was carefully separated from the vault of the skull. Care

was taken to avoid damage to the dura, superior sagittal sinus or the underlying cortex. The skull cap was removed. The dura was opened. The falx was freed from the crista galli of the cribriform plate, the frontal poles were retracted gently and the pituitary stalk and the internal carotid arteries were cut. The attachment of the tentorium to the skull was also cut to expose the posterior fossa, then the cranial nerves and the vertebral arteries were divided, the upper end of the spinal cord was transacted and the brain was gently removed from the skull. After preliminary observation, the brain was fixed by being placed in a 2 gallon polythene bucket containing neutral 4% formaldehyde in normal saline, and kept hanging freely , care was taken to insure that no surface of the brain was in contact with the floor or sides of the bucket. The solution (neutral 4% formaldehyde in normal saline) was changed after 3 days and then weekly through the interval of 3-4 weeks. Then the brain was washed in a running water for about an hour.

The intact fixed brain was examined again for external abnormalities such as generalised atrophy, abnormalities of the meninges and tonsillar or tentorial herniations. The dissection of the brain was carried out by cutting it in horizontal planes to correlate with the appearances in imaging and blocks were taken for microscopical examination.

5.6 Analysis

Because the study is essentially descriptive, because there are a large number of comparisons and many of these are based on small numbers, the data were not analysed statistically .

5.7 RESULTS

5.7.1 CT scan findings

The CT scan findings on the first scan, coded according to the by TCDB was that in 2 patients (1.9%) no pathological lesion was seen, 17 patients (16.7) had DI II, 21 patients (20.6%) had DI III (swelling), 5 patients (4.9%) had DI IV (shift), and 57 patients (40.2%) had a mass lesion (table 16).

41 patients (40.2%) showed midline shift more than 5mm., 81 patients (79.4%) showed abnormal basal cisterns, 24 compressed, and 57 obliterated. An abnormal III ventricle was recorded 83 patients (81.4%), of those 22 were compressed and 61 were obliterated. 55 patients (65.7%) showed SAH, and 45 patients (44.1%) showed a subcortical (deep) lesion.

The findings on the worst CT scan using TCDB classification were as follow: no patient had a normal scan, 11 patients had DI II, 28 patients had DI III " swelling", 5 patients had DI IV "shift" and 58 patients had a mass lesion ,

44 patients showed midline shift more than 5mm., 82 patients showed abnormal basal cisterns, 22 patients showed compressed III ventricle and 71 were obliterated , 15 patients showed focal and 44 diffuse SAH, and subcortical "deep" lesion were seen in 45 patients.

Table 17

CT SCAN FINDINGS

Traumatic Coma Data Bank

	Initial	Worst
DI I (normal)	2	0
DI II	17	11
DI III	21	28
DI IV	5	5
Mass lesion	55	56
Contusion	52	52
Deep Lesion	45	45
Mid-line Shift	41	44
Cisterns	81	92
III Ventricle	83	93
Intraventricular Haemorrhage	48	48
Subarchnoid Haemorrhage	55	67

Table 18

NEURO-PATHOLOGICAL FINDINGS

Contusion		
	- moderate	67
	- severe	6
Diffuse Axonal Swelling		
	- mild	7
	- moderate	6
	- severe	10
Raised Intracranial Pressure		83
Brain swelling		52
Hypoxic Brain Damage		
	- minimal	53
	- moderate	26
	- severe	13

5.8 Neuropathological findings

The PM neuropathological examination showed that 90 patients (88.2%) had a contusion, 73 moderate to severe. In 23 patients (22.5%) diffuse axonal injury was diagnosed on basis of macro and microscopical examinations. 83 patients (81.4%) showed signs of raised ICP , 55 patients (53.9%) showed brain swelling, 92 patients (90.2%) showed evidence of Hypoxic Brain Damage, 53 minimal, 26 moderate and 13 severe.

5.9 CT - Neuropathological correlations

5.9.1 MAJOR DIAGNOSIS

Correlation of Major Diagnosis on worst CT scan and pathological lesion on PM.

The Major Diagnosis on the worst CT scan of the 102 cases was cross-matched with the various post-mortem pathological findings (Table 19).

Contusion

Of the 52 patients with a surgical mass lesion on the worst CT scan, 38 (73.1%) were found at PM to have moderate to severe contusions, 15 (26.9%) patients had none or minimal contusions. Of 11 patients with an abnormal CT scan but no surgical mass lesion, or signs of raised ICP, 6 had moderate to severe contusions. Of 37 patients with an abnormal scan with no surgical mass lesion, but signs of raised ICP, 28 had moderate to severe contusions, and only 9 patients had none to minimal contusions at post-mortem.

Table 19

Major Diagnosis

PM Results		Surgical Mass Lesion n= 53	Abnormal No Significant mass lesion, No raised Intracranial pressure n= 11	Abnormal No Significant mass lesion, raised Intracranial pressure n= 37
Contusion	**None**	**7**	**2**	**3**
	Minimal	8	3	6
	Moderate	**34**	**6**	**26**
	Severe	4	-	2
Diffuse Axonal Injury	**None**	**30**	**6**	**19**
	Mild	2	1	4
	Moderate	**2**	**2**	**2**
	Severe	5	2	3
Intracranial pressure	**Normal**	7	**6**	**6**
	Raised	46	5	31
Brain Swelling	**None**	**24**	**6**	**15**
	Present	28	5	21
Hypoxic Brain Damage	**None**	**7**	-	**3**
	Minimal	24	5	24
	Moderate	**15**	**4**	**6**
	Severe	7	2	4
Pathological Cause of Death	**P.B.D.**	**16**	**6**	**21**
	E.I.L.	25	2	11
	O.I.C.	**3**	**2**	**2**
	E.C.	2	-	-

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

Diffuse Axonal Injury

Only 9 patients out of 53 patients with a surgical mass lesion on the CT scan showed DAI at PM pathology (5 severe, 2 moderate, and 2 mild). Of 11 patients with an abnormal CT scan, but no surgical mass lesion and no signs of raised ICP, 5 showed DAI at PM (2 severe, 2 moderate, and 1 mild). Of 37 patients with abnormal initial CT scan, no surgical mass lesion but with signs of raised ICP 9 patients showed DAI at PM (3 severe, 2 moderate, and 4 mild).

Post-mortem pathological finding indicating dynamic pathophysiology

The Major Diagnosis on the worst CT scan of the 102 cases was cross-matched with the post-mortem pathological findings indicating dynamic pathophysiology (Table19).

Raised Intra-cranial Pressure

In 83 (81.4%) of the 102 patients showed PM evidence that there had been raised ICP before death, 19 (18.6%) showed no signs of raised ICP. Of the 53 patients with a surgical mass lesion on the worst CT scan, 46 (86.8%) showed signs of raised ICP at PM. Of the 11 patients with an abnormal CT scan, but no surgical mass lesion and no signs of raised ICP, 5 showed signs of raised ICP at PM. Of the 37 patients with an abnormal scan, with signs of raised ICP, 31 (83.8%) exhibited signs of raised ICP on PM.

Brain Swelling

The cross matching of major diagnosis on the CT scan with brain swelling on the post-mortem revealed that of the 53 patients with a surgical mass lesion, 28 had brain swelling at PM. Of the 11 patients with an abnormal CT but without a surgical mass lesion or signs of raised ICP, 5 of them had brain swelling at the PM. Of the other 37 patients with signs of raised ICP on the worst CT scan, 21 showed brain swelling on PM.

Hypoxic Brain Damage (HBD)

46 (86.8%) patients out of 53 with a surgical mass lesion showed HBD on PM (24 minimal, 15 moderate, and 7 severe), all the patients with an abnormal scan with no surgical mass lesion and no signs of raised ICP showed HBD (5 minimal, 4 moderate, 2 severe) and of the 37 patients with signs of raised ICP on the worst CT scan, 34 exhibited HBD at PM (24 minimal, 6 moderate, 4 severe).

Pathologist's opinion on Cause of Death

Of the 53 patients with a surgical mass lesion on the CT scan, 16 were considered to died have from primary brain damage, 25 from an expanding intracerebral lesion, 3 from other intracranial complications, 2 from extracranial complications, and in 7 the cause was uncertain. Of the 11 patients with an abnormal CT scan but no surgical mass lesion and no raised ICP, 6 died from primary brain damage, 2 died from an expanding intracranial lesion, 2 from other intracranial complications, and in one the cause was uncertain. Of the 37 patients with signs of raised ICP on the worst CT scan, 21 died from primary brain damage, 11 from an

expanding intracranial lesion, 2 from other intracranial complications and in 3 the cause was unknown.

Comments

Contusions were a common finding at PM, they were present in all CT groups and only 12 cases did not show contusion at PM. Signs of Diffuse Axonal Injury were found in about 1/4 of patients with significant mass lesion but were more frequent in cases with diffuse injury, especially those without signs of raised intracranial pressure. Raised Intracranial pressure was present in most cases, and even in a half of the cases without CT signs of raised ICP, indicating that most cases suffer raised ICP before death. Brain Swelling was less frequent than raised ICP in all groups ,except for diffuse injury without CT signs of raised ICP. Hypoxic Brain damage was present in 90% of all cases, and was Moderate-Severe in about half of the cases of diffuse injury without CT signs of raised ICP. In 1/3 of patients with a surgically significant mass lesion, the pathologists opinion that death was due to primary brain damage, but more than a half died potentially avoidable deaths due to the effects of cerebral compression. Most patients with diffuse injury without CT signs of raised ICP were confirmed to have died from primary brain damage. However 13 cases without surgically significant mass were considered to have died from an expanding intracranial lesion

Table 20

TRAUMATIC COMA DATA BANK CLASSIFICATION

PM Results		Diffuse injury II n= 11	Diffuse injury III (swelling) n= 28	Diffuse injury IV (shift) n= 5	Evacuated Mass Lesion n= 54	Non Evacuated Mass Lesion n= 2
Contusion	**None**	**2**	**3**	-	7	-
	Minimal	4	4	1	8	-
	Moderate	**5**	**19**	**4**	**35**	**2**
	Severe	-	2	-	4	-
Diffuse Axonal Injury	**None**	**5**	**13**	**4**	**31**	**1**
	Mild	1	3	-	3	-
	Moderate	**3**	**2**	-	**1**	-
	Severe	2	3	-	5	-
Intracranial pressure	**Normal**	**6**	**6**	-	7	-
	Raised	5	22	5	47	2
Brain swelling	**None**	7	**12**	**1**	**25**	-
	Present	4	15	4	28	2
Hypoxic Brain Damage	**None**	-	**3**	-	7	-
	Minimal	5	17	2	27	1
	Moderate	**4**	**5**	**1**	**14**	**1**
	Severe	2	3	2	6	-
Pathological cause of death	**P.B.D.**	**7**	**18**	**2**	**15**	-
	E.I.L.	2	7	2	26	2
	O.I.C.	**2**	**1**	**1**	**3**	-
	E.C.	-	-	-	2	-

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

5.9.2 TRAUMATIC COMA DATA BANK CLASSIFICATION

Correlation between the application of the Traumatic Coma Data Bank classification to the worst CT scan and Primary Pathological Lesions seen On PM.

Table 20, Shows the cross matching of the TCDB classification on the worst CT scans of the 102 patients with the occurrence at PM of lesions of "primary" brain damage

Contusions

The cross matching of the TCDB method of coding with signs of contusions at post-mortem showed that of the 11 patients with DI II on the worst CT scan, 9 showed contusion on PM (4 minimal and 5 moderate), of the 28 patients with DI III (swelling), 25 showed contusion (4 minimal, 19 moderate and 2 severe), each of the 5 patients with DI IV (shift) had contusions (1 minimal and 4 moderate) and of the 54 patients with a surgical mass lesion, 47 showed contusions (8 minimal, 35 moderate and 4 severe)

Diffuse Axonal Injury

Cross matching of the results of the TCDB coding showed that of the 11 patients with DI II on the worst CT scan, 6 had signs of DAI (1 mild, 3 moderate and 2 severe), 5 had no signs of DAI and 7 could not be classified. Of the 28 patients with DI III (swelling) on the worst CT scan, 8 showed DAI (3 mild, 2 moderate and 3 severe), 13 had no signs of DAI and 1 patient could not be classified. Of the 5 patients with DI IV (shift), none of them had DAI and 1 patient could not be

classified. Of the 54 patients with a surgical mass lesion, 8 of showed DAI (3 mild, 1 moderate and 5 severe), 31 patients had no signs of DAI, and 14 patients could not be classified.

Traumatic Coma Data Bank Classification of CT scan findings and post-mortem features of pathological dynamic processes

Table 20, Shows the correlation between the Traumatic Coma Data Bank coding of the worst CT scan with post-mortem evidence of dynamic processes

Raised Intracranial Pressure

Of 11 with DI II on the CT scan 5 had signs of raised ICP at PM. Of 28 patients with DI III (swelling), 22 had signs of raised ICP at PM, and all 5 patients with DI IV (shift) on the CT scan had signs of raised ICP at PM. Of the 54 patients with a surgical mass lesion, 47 had signs of raised ICP at PM.

Brain Swelling at PM.

Of the 11 patients with DI II, 4 had brain swelling at PM, of 28 patients with DI III (swelling), 15 of them had brain swelling and one patient was not classified. Of the 5 patients with DI IV (shift) 3 had brain swelling at PM and of the 54 patients with a mass lesion, 28 had brain swelling at PM and one patient could not be classified.

Hypoxic Brain Damage (HBD)

Of the 11 patients with DI II on the worst scan, all showed HBD on PM (5 minimal, 4 moderate and 3 severe), of the 28 patients with DI III (swelling), 25 showed

HBD (17 minimal, 5 moderate and 3 severe), each of the 5 patients with DI IV (shift) had HBD (2 minimal, 1 moderate and 2 severe). Of the 54 patients with a mass lesion, 47 had HBD (27 minimal, 14 moderate and 6 severe).

Pathologist's opinion of the Cause of Death

Of the 11 patients with DI II, 7 were considered to have died from primary brain damage, 2 from an expanding intracranial lesion, 2 from other intracranial complications. Of 26 patients with DI III (swelling), 18 were considered to have died from primary brain damage, 7 patients died due to an expanding intracranial lesion and one from other intracranial complications, of the 5 patients with DI IV (shift), 2 patients died from primary brain damage, 2 died from an expanding intracranial lesion and 1 from other intracranial complications Of the 54 patients with a mass lesion, 15 patients were considered to have died from primary brain damage, 26 from an expanding intracranial lesion, 3 from other intracranial complications and 2 from extracranial complications.

Comments

The findings with the TCDB classification echo the finding for the "major diagntic grouping". Contusions were found in all groups, minimal-moderate contusions in half of those with DI II, moderate or worse in more than 2/3 of the other groups. Diffuse Axonal Injury was not found in DI IV (shift), and was found in a smaller percentage of patients with a mass lesion than in the other groups. Raised ICP was found in half of the cases with DI II (diffuse injury without swelling), and in 90% of the other groups. In the TCDB classification DI III and DI IV refer to Diffuse injury with swelling and in

DI IV with shift , but at PM no swelling was found in 41%. Hypoxic Brain Damage was, surprisingly, absent or minimal in 2/3 of DI III (swelling), and in this group 2/3 of deaths were considered to be due to primary brain damage.

Table 21

CONTUSION

PM Results		No Contusion n= 50	Contusion Of No Mass Lesion n= 16	Contusion Of Mass Lesion n= 36
Contusion	**None**	**9**	**2**	**1**
	Minimal	14	3	-
	Moderate	**26**	**10**	**31**
	Severe	1	1	4
Diffuse Axonal Injury	**None**	**27**	**7**	**22**
	Mild	2	1	4
	Moderate	**5**	**1**	-
	Severe	5	3	2
ICP	**Normal**	**15**	**1**	**3**
	Raised	35	15	33
Brain Swelling	**None**	**33**	**6**	**9**
	Present	19	9	27
Hypoxic Brain Damage	**None**	**5**	-	**5**
	Minimal	25	9	15
	Moderate	**11**	**5**	**8**
	Severe	9	2	1
Pathological Cause of Death	**P.B.D.**	**23**	**6**	**14**
	E.I.L.	16	7	16
	O.I.C.	**5**	**1**	**1**
	E.C.	1	-	1

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

5.9.3 CONTUSION ON CT SCAN

Correlation between observation of Contusion on CT scan and Primary Pathological Lesions seen at PM.

Table 21, Shows the cross matching of the assessment of contusions on the worst CT scans and PM signs of primary pathological lesions

Contusions at PM

Of the 50 patients with no contusions seen on the CT scan, 41 showed contusions at PM, (14 minimal, 26 moderate and one severe). Of the 16 patients who showed contusion with no mass effect on the worst CT scan, in 2 contusions were missed at PM, in 14 contusions were seen at PM (3 minimal, 10 moderate and one severe). Of the 36 patients who showed contusions with mass effect on the worst CT scan, the contusion was missed at PM in one, and 35 showed contusion at PM (13 moderate and one severe).

Diffuse Axonal Injury

Of the 50 patients with no contusion on the CT scan, 27 had no evidence of DAI and 12 showed DAI (2 mild, 5 moderate and 5 severe). Of the 37 patients with contusion without mass effect, 20 had no signs of DAI and 8 showed DAI (2 mild, 1 moderate and 5 severe). Of the 15 patients who had contusions with mass effect, 9 showed no evidence of DAI and 3 showed mild DAI.

The correlation between observation of contusions on the worst CT scan and post-mortem features of dynamic pathological processes

Table 21 Shows the cross matching of contusion on the worst CT scan with post-mortem features of dynamic pathological processes:

Signs of Raised Intracranial Pressure

Of the 50 patients with no evidence of contusion on the CT scan, 35 showed signs of raised ICP, of the 37 patients with contusion without mass effect, 35 showed signs of raised ICP and of the 15 patients with contusion with mass effect, 13 showed signs of raised ICP at PM.

Brain Swelling at P.M.

Of the 50 patients with no evidence of contusion on the CT scan, 19 showed brain swelling, of the 37 patients with contusion without mass effect, 26 had brain swelling and 10 showed no brain swelling. Of the 15 patients with contusions with mass effect, 10 had brain swelling at PM.

Hypoxic Brain Damage

Of the 50 patients with no contusion on the CT scan, 45 showed HBD (25minimal, 11 moderate, and 9 severe), of the 37 patients with contusion without mass effect, 34 showed HBD (19 minimal, 11 moderate, and 4 severe), and of the 15 patients with contusion with mass effect, 13 showed HBD (9 minimal and 4 moderate).

Pathologist's opinion of the Cause of Death

Of the 50 patients with no signs of contusion on the CT scan, 23 were considered to have died from primary brain damage, 16 from an expanding intracranial lesion, 5 from other intracranial complications, and one from extracranial complications. Of 37 patients with contusion without mass effect, 14 died from primary brain damage, 16 from an expanding intracranial lesion, and 1 from other Intracranial complications. Of the 15 patients who had contusions with mass effect, 6 died from primary brain damage, 7 from an expanding intracranial lesion, 1 from other intracranial complications, and one from extracranial complications.

Comments

CT scanning underestimates the occurrence of Contusion is a well known, but, in some cases even moderate and severe contusions identified at PM were not seen with the CT scan. Conversely and surprising, in 3 cases contusions seen on CT were missed at post-mortem. Diffuse Axonal Injury was somewhat less often found in cases with contusion with mass effect. Patients with contusions seen on CT more often had raised ICP and brain swelling, whether or not the contusions had mass effect. The cause of death was somewhat more often due to primary brain damage in the patients with contusions seen on CT .

Table 22

INTRAPARENCHYMAL LESIONS

PM Resuts		No Lesion n= 57	Subcortical Lesion n= 11	Deep White Matter n= 9	Basal Ganglia n= 12	Brainstem Cerebellum n= 13
Contusion	**None**	**5**	**1**	**2**	**1**	**3**
	Minimal	4	2	3	3	5
	Moderate	**45**	**7**	**4**	**6**	**5**
	Severe	3	1	-	2	-
Diffuse Axonal Injury	**None**	**35**	**5**	**3**	**8**	**5**
	Mild	5	1	-	1	-
	Moderate	**2**	**1**	**1**	-	**2**
	Severe	2	2	2	1	3
Intracranial pressure	**Normal**	**8**	**2**	**3**	**4**	**2**
	Raised	49	9	6	8	11
Brain Swelling	**None**	**22**	**3**	**6**	7	7
	Present	34	7	3	5	6
Hypoxic Brain Damage	**None**	7	-	**2**	-	**1**
	Minimal	29	6	4	6	8
	Moderate	**16**	**4**	**1**	**2**	**3**
	Severe	5	1	2	4	1
Pathological Cause of Death	**P.B.D.**	**19**	**6**	**4**	**5**	**9**
	E.I.L.	27	3	3	3	3
	O.I.C.	**3**	-	**1**	**3**	-
	E.C.	2	-	-	-	-

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

5.9.4 INTRAPARENCHYMAL LESIONS

Correlation between observation of the interparenchymal lesions on the worst CT scan and Primary Pathological Lesions seen at PM.

Table 22, shows the cross matching of the Intraparenchymal lesions on the worst CT scans of the 102 patients with the evidence of primary pathological lesions at PM of the same patients :

Diffuse Axonal Injury

Of 44 patients with no deep lesion on the worst CT scan, 9 showed DAI (5 mild, 2 moderate and 2 severe). Of 9 patients with subcortical lesion on the worst CT scan, 4 showed DAI (1 minimal, 1 moderate and 2 severe). Of the 6 patients studied with a deep white matter lesion on the worst CT scan, and 3 showed DAI (1 moderate and 2 severe). Of the 10 patients studied with basal ganglia lesions, DAI was seen only in 2 (1 mild and 1 severe), and of the 10 patients with a brainstem and/or cerebellum lesions, only 5 showed DAI (2 moderate and 3 severe).

Correlation between Intraparenchymal Lesions observed on CT scan and post-mortem evidence of dynamic processes

Table 22, Shows the cross matching of the intraparenchymal lesions seen on the worst CT scan with post-mortem features of dynamic pathological processes:

Raised Intracranial Pressure

Of the 11 patients with a subcortical lesion, 9 showed signs of raised ICP. Of 9 patients with a deep white matter lesion, 6 showed signs of raised ICP. Of the 12

patients with basal ganglia lesions, 8 showed signs of raised ICP. Of the 13 patients with Brainstem and/or cerebellum lesions on the worst CT scan, 11 showed signs of raised ICP at PM.

Brain Swelling

Of the 11 patients with a subcortical lesion, 7 showed brain swelling. Of the 9 patients with a deep white matter lesion, 3 showed brain swelling. Of the 12 patients with basal ganglia lesions, 5 showed brain swelling, and of the 13 patients with brainstem and/or cerebellum lesions 6 showed brain swelling at PM.

Hypoxic Brain Damage

Each of the 11 patients with a subcortical lesion showed HBD on PM (6 minimal, 4 moderate, and 1 severe). Of 9 patients with a deep white matter lesion, 7 showed HBD (4 minimal, 1 moderate, and 2 severe). Each of the 12 patients with basal ganglia lesions, showed HBD (6 minimal, 2 moderate, and 4 severe), and of the 13 patients with Brainstem and/or cerebellum lesions, 12 showed HBD (8 minimal, 3 moderate, and 1 severe).

Pathologist's opinion on Cause of Death

Of the 57 patients with no deep lesion seen on the worst CT scan, 19 were considered to have died from primary brain damage, 27 from an expanding intracranial lesion, 3 from other Intracranial complications, and 2 from Extracranial complications. Of the 11 patients with a subcortical lesion, 6 died from primary brain damage, 3 from an expanding intracranial lesion. of the 9 patients with a deep white matter lesion, 4

died from primary brain damage, 3 from an expanding intracranial lesion, and one from other intracranial complications. Of the 12 patients with a basal ganglia lesion, 5 died from primary brain damage, 3 from an expanding intracranial lesion, and 3 from other Intracranial complications. Of the 13 patients with Brainstem and/or cerebellum lesions, 9 died from primary brain damage and 3 from an expanding intracranial lesion.

Comments

There was less correlation than expected between the presence of intraparenchymal lesion on CT and evidence of DAI at post-mortem. Thus DAI was found in only a half of patients with parenchymal lesions on CT, even when these lesions were in the brain stem or cerebellum, and was found in only 2 of 10 patients with lesions in the Basal ganglia. Nevertheless, patients with intraparenchymal lesions were more often considered to have died from primary brain damage than those without.

Table 23

INTRACRANIAL HAEMATOMA

PM Results		No Intracranial Haematoma n= 31	Intracranial Haematoma Of Surgical Significant n= 49	Intracranial Haematoma Not Surgical Significant n= 22
Contusion	**None**	**4**	**7**	**1**
	Minimal	6	7	4
	Moderate	**19**	**31**	**17**
	Severe	2	4	
Diffuse Axonal Injury	**None**	**16**	**30**	**10**
	Mild	3	1	3
	Moderate	**2**	**1**	**3**
	Severe	4	5	1
Intracranial pressure	**Normal**	**11**	7	**1**
	Raised	20	42	21
Brain Swelling	**None**	**14**	**21**	**10**
	Present	16	27	12
Hypoxic Brain Damage	**None**	**2**	**6**	**2**
	Minimal	18	23	12
	Moderate	**7**	**14**	**5**
	Severe	4	6	3
Pathological Cause of Death	**P.B.D.**	**18**	**12**	**13**
	E.I.L.	8	25	6
	O.I.C.	**2**	**3**	**2**
	E.C.	-	2	-

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

5.9.5 INTRACRANIAL HAEMATOMA ON CT

Correlation between observation of intracranial haematoma on CT and Primary Pathological Lesions at PM.

Table 23, Shows the cross matching of the Intracranial haematoma on the worst CT scans and the PM signs of primary pathological lesions.

Contusions

Of the 22 patients with an ICH considered not to be surgically significant, 21 showed contusions (4 minimal, and 17 moderate). Of the 49 patients with a surgically significant ICH, 42 showed contusion (7 minimal, 31 moderate and 4 severe).

Diffuse Axonal Injury

Of the 17 patients studied with ICH considered not to be surgically significant, 7 showed DAI (3 mild, 3 moderate and 1 severe), and the 37 patients studied with a surgically significant ICH, 7 showed DAI (1 mild, 1 moderate and 5 severe).

PM.

Correlation between an Intracranial Haematoma (ICH) on the worst CT scan and post-mortem features of pathological dynamic processes

Table 23, Shows the cross matching of the Intracranial haematoma seen on the worst CT scan with post-mortem features of dynamic pathological processes:

Raised intracranial Pressure

Of the 22 patients with ICH considered to be not surgically significant, 21 showed signs of raised ICP, and of the 49 patients with a surgically significant ICH, 42 showed signs of raised ICP.

Brain Swelling

Of the 22 patients with an ICH considered to be not surgically significant, 12 showed brain swelling, and of the 49 patients with a surgically significant ICH, 27 showed brain swelling.

Hypoxic Brain Damage

Of 22 patients with ICH considered to be not surgically significant, 20 showed HBD (12 minimal, 5 moderate, and 3 severe), and of the 49 patients with a surgically significant ICH, 43 showed HBD (23 minimal, 14 moderate, and 6 severe).

Pathologist's opinion on Cause of Death

Of the 31 patients who showed no ICH on the worst CT scan, 18 were considered to have died from primary brain damage, 8 died from an expanding intracranial lesion, 2 from other Intracranial complications. Of the 22 patients with ICH considered to be not surgically significant, 13 were considered to have died from primary brain damage, 6 from an expanding intracranial lesion and 2 from other Intracranial complications. Of the 49 patients with a surgically significant ICH, 12 were considered to have died from primary brain damage, 25 from an expanding intracerebral lesion, 3 from other Intracranial complications, 2 from Extracranial complications and 7 the cause was unknown.

Comments

Patients with an intracranial haematoma on CT more often showed signs of raised ICP at PM, but this did not apply to all cases. Most deaths were considered to be due to expanding intracranial lesions, but even with a surgically significant intracranial haematoma, 40% of deaths were not due to expanding intracranial lesions and twelve of the 42 were considered to have died from primary brain damage. This was confirmed in 7 cases by post-mortem evidence of diffuse axonal injury.

Table 24

CEREBROSPINAL FLUID SPACES

PM Results		Cerebrospinal fluid spaces							
		Basal Cisterns				Third Ventricle			
		Normal n=10	Compressed n=28	Obliterated n=63	Dilated n=1	Normal n=9	Compressed n=21	Obliterated n=71	Dilated n=1
Contusion	None	1	3	8	-	1	3	8	-
	Minimal	4	7	6	-	3	7	7	-
	Moderate	5	16	45	1	5	9	52	1
	Severe	-	2	4	-	-	2	4	-
Diffuse Axonal Injury	None	5	18	32	1	4	15	36	1
	Mild	1	3	3	-	1	1	5	-
	Moderate	3	1	2	-	2	2	2	-
	Severe	1	1	8	-	2	-	8	-
Intra-cerebral pressure	Normal	6	6	7	-	6	4	9	-
	Raised	4	22	56	1	3	17	62	1
Brain Swelling	None	7	13	25	-	7	10	28	-
	Present	3	15	36	1	2	11	41	1
Hypoxic Brain Damage	None	-	4	6	-	-	3	7	-
	Minimal	5	14	34	-	6	9	38	-
	Moderate	3	7	16	-	1	8	16	1
	Severe	2	3	7	1	2	1	10	-
Patho-logical Cause of Death	P.B.D.	6	7	30	-	5	5	33	-
	E.I.L.	2	13	24	-	3	13	23	-
	O.I.C.	1	3	3	-	-	2	5	-
	E.C.	1	1	-	-	1	-	1	-

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

5.9.6 BASAL CISTERNS

Correlation between observation of the state of on the basal cisterns on worst CT scan and Primary Pathological Lesions at PM.

Table 23, Shows the cross matching of the state of Basal Cisterns on the worst CT scans and the PM signs of primary pathological lesions.

Contusions

Of the 10 patients whose basal cisterns appeared normal on the worst CT, 9 were found at PM to have contusions (4 minimal, 5 moderate). Of the 28 patients with compressed basal cisterns, 25 patients were found at PM to have contusions (7 Minimal, 16 moderate, 2 severe). Of the 63 patients with obliterated basal cisterns, 55 patients were found at PM to have contusions, (6 minimal, 45 moderate, 4 severe). and the one with dilated basal cisterns showed moderate contusion

Diffuse Axonal Injury

Of the 10 patients whose basal cisterns appeared normal on the worst CT scan, half showed DAI at PM (1 mild, 3 moderate and 1 severe). Of the 28 patients with compressed basal cisterns, only 5 showed DAI (3 mild, 1 moderate and 1 severe). Of the 45 patients with obliterated basal cisterns, 13 showed DAI (3 mild, 2 moderate and 8 severe).

Correlation between the state of Basal Cisterns on the worst CT scan and post-mortem features of pathological dynamic processes

Table 24, Shows the cross matching of the state of Basal Cisterns on the worst CT scan with post-mortem features of dynamic pathological processes:

Raised Intra-Cranial Pressure

Of the 10 patients whose with basal cisterns appeared normal on the worst CT scan, 4 showed signs of raised ICP at PM. Of the 28 patients with compressed basal cisterns, 22 showed signs of raised ICP, and of the 63 patients with obliterated basal cisterns, 56 showed signs of raised ICP and a single patient with dilated basal cisterns showed signs of raised ICP

Brain Swelling

Of the 10 patients whose with basal cisterns appeared normal on the worst CT scan, 7 showed brain swelling. Of the 28 patients with compressed basal cisterns, 13 showed brain swelling. Of the 63 patients with obliterated basal cisterns, 25 showed brain swelling and the patient with dilated basal cisterns showed brain swelling at PM.

Hypoxic Brain Damage

All the 10 patients whose with basal cisterns appeared normal on the worst CT scan, showed HBD (5 minimal, 3 moderate, and 2 severe). Of the 28 patients with compressed basal cisterns, 24 showed HBD (14 minimal, 7 moderate, and 3 severe). Of the 63 patients with obliterated basal cisterns, 57 showed HBD (34 minimal, 16

moderate, and 7 severe), and the single patient with dilated basal cisterns showed severe HBD.

Pathologist's opinion on Cause of Death

Of the 10 patients whose with basal cisterns appeared normal on the worst CT scan, 6 were considered to have died from primary brain damage, 2 from an expanding intracranial lesion one from other Intracranial complications and one from extracranial complications. Of the 28 patients with compressed basal cisterns, 7 were considered to have died from a primary brain damage, 13 from an expanding intracranial lesion, 3 from other Intracranial complications and one from extracranial complications. Of the 63 patients with obliterated basal cisterns, 30 were considered to have died from a primary brain damage, 24 from an expanding intracranial lesion and 3 from other Intracranial complications.

5.9.7 THIRD VENTRICLE

Correlation between the state of on the third ventricle on the worst CT scan and Primary Pathological Lesions seen at PM.

Table 24, Shows the cross matching of the state of III ventricle on the worst CT scans and PM signs of primary pathological lesions

Contusions

Of the 9 patients with normal III Ventricle on the worst CT scan, 8 showed contusion (3 minimal and 5 moderate). Of the 21 patients with compressed III Ventricle, 18 showed contusion (7 minimal, 9 moderate and 2 severe). Of the 71 patients with obliterated III Ventricle, 63 showed contusion (7 minimal, 52 moderate and 4 severe), and the single patient with A dilated III Ventricle showed moderate contusion at PM.

Diffuse Axonal Injury

Of the 9 patients with normal III Ventricle on the worst CT scan, 5 had DAI (1 mild, 2 moderate and 2 severe). Of the 18 patients with compressed III Ventricle, 3 showed DAI (1 mild, and 2 moderate). Of the 51 patients with obliterated III Ventricle, 15 showed DAI (5 mild, 2 moderate and 8 severe), and the single patient with a dilated III Ventricle showed no DAI at PM.

Correlation between the III Ventricle on the worst CT scan and post-mortem features of pathological dynamic processes

Table 24, Shows the cross matching of the state of III Ventricle on the worst CT scan with post-mortem features of dynamic pathological processes:

Raised Intra-Cranial Pressure

Of the 9 patients with normal III Ventricle on the worst CT scan, only 3 showed signs of raised ICP. Of the 21 patients with compressed III Ventricle, 17 showed signs of raised ICP, and of the 71 patients with obliterated III Ventricle, 62 showed signs of raised ICP at PM. A single patient with dilated III Ventricle showed signs of raised ICP.

Brain Swelling

Of the 9 patients with normal III Ventricle on the worst CT scan, only 2 showed brain swelling at PM, of the 21 patients with compressed III Ventricle, 11 showed brain swelling at PM and of the 71 patients with obliterated III Ventricle, 41 showed brain swelling and 2 patients could not be classified.

Hypoxic Brain damage (HBD)

All the 9 patients with normal III Ventricle showed HBD (6 minimal, 1 moderate, and 2 severe). Of the 21 patients with compressed III Ventricle, 18 showed HBD (9 minimal, 8 moderate, and 1 severe). Of the 71 patients with obliterated III Ventricle, 64 showed HBD (38 minimal, 16 moderate, and 10 severe) A single patient with dilated III Ventricle showed moderate HBD at PM.

Pathologist's opinion on Cause of Death

Of the 9 patients with a normal III Ventricle, 5 were considered to have died from a primary brain damage, 3 from an expanding intracranial lesion and one from extracranial complications. Of the 21 patients with compressed III Ventricle, 5 were considered to have died from a primary brain damage, 13 from an expanding intracranial lesion and 2 from other intracranial complications. Of the 71 patients with an obliterated III Ventricle, 33 were considered to have died from a primary brain damage and 23 from an expanding intracranial lesion

Comments

Abnormalities of the basal cisterns or III Ventricle either compression or obliteration were closely associated with signs of raised intracranial pressure at post-mortem and to a lesser extent with brain swelling. In contrast, hypoxic brain damage was not clearly associated with, basal cisterns and III ventricle appearances. When, there was an abnormality of either basal cisterns or third ventricle the cause of death was more often due to expanding intracranial lesion. However, even with obliterated basal cisterns or III ventricle half of the deaths were due to primary brain damage.

Table 25

INTRAVENTRICULAR HAEMORRHAGE

PM Results		No Intraventricular Haemorrhage n= 54	Blood Limited to One Ventricle n= 19	Blood in > One Ventricle n= 29
Contusion	**None**	**5**	**2**	**5**
	Minimal	7	3	7
	Moderate	**37**	**14**	**16**
	Severe	5	-	1
Diffuse Axonal Injury	**None**	**32**	**9**	**15**
	Mild	4	2	1
	Moderate	**3**	**2**	**1**
	Severe	5	2	3
ICP	**Normal**	**8**	**2**	**9**
	Raised	46	17	20
Brain Swelling	**None**	**22**	**6**	**17**
	Present	31	12	12
Hypoxic Brain Damage	**None**	**4**	**1**	**5**
	Minimal	25	13	15
	Moderate	**14**	**4**	**8**
	Severe	11	1	1
Pathological Cause of Death	**P.B.D.**	**20**	**10**	**13**
	E.I.L.	20	8	11
	O.I.C.	**4**	**1**	**2**
	E.C.	1	-	1

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

5.9.8 INTRAVENTRICULAR HAEMORRHAGE

Correlation between the observation of the intraventricular haemorrhage on the worst CT scan and Primary Pathological Lesions at PM.

Table 25, Shows the cross matching of the Intraventricular haemorrhage on the worst CT scans and the PM signs of primary pathological lesions.

Contusions

Of the 54 patients with no Intraventricular blood on the worst CT scan, 49 showed contusions (7 minimal, 37 moderate and 5 severe). Of the 19 patients with blood in only one ventricle, 17 showed contusions (3 minimal and 14 moderate). Of the 29 patients with blood in more than one ventricle, 24 showed contusions (7 minimal, 16 moderate and 1 severe)

Diffuse Axonal Injury

Of the 54 patients with no Intraventricular blood on the worst CT scan, 12 showed DAI (4 mild, 3 moderate and 5 severe) and 10 could not be classified. Of the 19 patients with blood in only one ventricle, 6 showed DAI (2 minimal, 2 moderate and 2 severe) and 4 were unclassified. Of the 29 patients with blood in more than one ventricle, 5 showed DAI (1 mild, 1 moderate and 3 severe), and 9 were unclassified.

Correlation between the observation of the Intraventricular on the worst CT scan and post-mortem features of pathological dynamic processes

Table 25, Shows the cross matching of the Intraventricular blood on the worst CT scan with post-mortem features of dynamic pathological processes:

Raised Intra-Cranial Pressure

Of the 54 patients with no Intraventricular blood on the worst CT scan, 46 showed signs of raised ICP. Of the 19 patients with blood in only one ventricle, 17 showed signs of raised ICP, and of the 29 patients with blood in more than one ventricle, 20 showed signs of raised ICP at PM

Brain Swelling

Of the 54 patients with no Intraventricular blood on the worst CT scan, 31 showed brain swelling. Of the 19 patients with blood in only one ventricle on worst CT scan, 12 showed brain swelling, and of the 29 patients with blood in more than one ventricle, 12 showed brain swelling.

Hypoxic Brain Damage

Of the 54 patients with no Intraventricular blood on the worst CT scan, 50 showed HBD (25 minimal, 14 moderate, and 11 severe). Of the 19 patients with blood in only one ventricle, 18 showed HBD (13 minimal, 4 moderate, and 1 severe), and of the 29 patients with blood in more than one ventricle, 24 showed HBD (15 minimal, 8 moderate, and 1 severe) at PM.

Pathologist's opinion on Cause of Death

Of the 54 patients with no Intraventricular blood on the worst CT scan, 20 were considered to have died from primary brain damage, 20 from an expanding intracranial lesion, 14 from other intracranial complications, and one from an extracranial complications. Of the 19 patients with blood in only one ventricle, 10 were considered to have died from primary brain damage, 8 from an expanding intracranial lesion and one from other Intracranial complications. And of the 29 patients with blood in more than one ventricle, 13 were considered to have died from primary brain damage, 11 from an expanding intracranial lesion, 2 from other Intracranial complications and one from extracranial complications.

Comments

There was little correlation between intraventricular haemorrhage and post-mortem findings. Diffuse Axonal Injury was present in slightly more than 1/4 of cases without IVH and in 1/3 of cases with IVH. Also, the pathologists opinion on the cause of death showed no correlation with CT signs of intraventricular haemorrhage.

Table 26

SUBARCHNOID

PM Results		No Subarchnoid haemorrhage n= 35	Focal Subarchnoid haemorrhage n= 15	Diffuse Subarchnoid haemorrhage n= 44	Tentorial Blood n= 8
Contusion	**None**	**5**	**3**	**4**	-
	Minimal	9	2	5	1
	Moderate	**18**	**10**	**33**	**6**
	Severe	3	--	2	1
Diffuse Axonal Injury	**None**	**18**	**9**	**24**	**5**
	Mild	2	2	2	1
	Moderate	**3**	-	**3**	-
	Severe	3	2	5	-
Intracranial pressure	**Normal**	**8**	**2**	7	**2**
	Raised	27	13	37	6
Brain Swelling	**None**	**19**	7	**17**	**2**
	Present	17	8	26	9
Hypoxic Brain Damage	**None**	**5**	**1**	**3**	**1**
	Minimal	12	10	28	3
	Moderate	**12**	**3**	**8**	**3**
	Severe	6	1	5	1
Pathological Cause of Death	**P.B.D.**	**12**	**3**	**26**	**2**
	E.I.L.	14	9	12	4
	O.I.C.	**3**	**2**	**2**	-
	E.C.	2	-	-	-

Pathological cause of death

P.B.D. Primary brain damage
E.I.L. Expanding intracranial lesion
O.I.C. Other intracranial complications
E.C. Extracranial complocations

5.9.9 CT SIGNS OF SUBARCHNOID HAEMORRHAGE

Correlation between the presence of Subarchnoid haemorrhage (using HIT II) on the worst CT scan and Primary Pathological Lesions at PM.

Table 26, Shows the cross matching of the presence of Subarchnoid Haemorrhage on the worst CT scans and the PM signs of primary pathological lesions.

Contusions

Of the 35 patients with no SAH, 30 showed contusions (9 minimal, 18 moderate and 3 severe). Each of the 8 patients with tentorial SAH, showed contusions at PM (1 minimal, 6 moderate and 1 severe). Of the 15 patients with focal SAH, 12 showed contusions (2 minimal and 10 moderate), and of the 44 patients with diffuse SAH, 40 showed contusions (5 minimal, 33 moderate and 2 severe).

Diffuse Axonal Injury

Of the 26 patients studied with no SAH on the worst CT scan, , 8 showed DAI (2 mild, 3 moderate and 3 severe). Of the 6 patients studied with tentorial SAH, one showed mild DAI. Of the 13 patients studied with focal SAH, 4 showed DAI (2 mild and 2 severe), and of the 34 patients with diffuse SAH, 10 showed DAI (2 mild, 3 moderate and 5 severe).

Correlation between the presence of Subarchnoid haemorrhage (using HIT II) on the worst CT scan and post-mortem features of pathological dynamic processes

Table 26, Shows the cross matching of the presence of Subarchnoid Haemorrhage on the worst CT scan with post-mortem features of dynamic pathological processes:

Raised Intra-Cranial Pressure

Of the 35 patients with no SAH on the worst CT scan, 27 showed signs of raised ICP. Of the 8 patients with tentorial SAH, 6 showed signs of raised ICP. Of the 15 patients with focal SAH, 13 showed signs of raised ICP, and of the 44 patients with diffuse SAH, 37 showed signs of raised ICP.

Brain Swelling

Of the 35 patients with no SAH, 15 showed brain swelling, of the 8 patients with tentorial SAH, 6 showed brain swelling, of the 15 patients with focal SAH, 8 showed brain swelling, and of the 44 patients with diffuse SAH, 26 showed brain swelling.

Hypoxic Brain Damage

Of the 35 patients with no SAH on the worst CT scan, 30 showed HBD (12 minimal, 12 moderate, and 6 severe), of the 8 patients with tentorial SAH, 7 showed HBD (3 minimal, 3 moderate, and one severe), of the 15 patients with focal SAH, 14 showed HBD (10 minimal, 3 moderate, and one severe). and the 44 patients with diffuse SAH, 41 showed HBD (28 minimal, 8 moderate, and 5 severe).

Pathologist's opinion on Cause of Death

Of the 35 patients with no SAH on the worst CT scan, 12 were considered to have died from a primary brain damage, 14 from an expanding intracranial lesion, 3 from other Intracranial complications, and 2 from extracranial complications, of the 8 patients with tentorial SAH on the worst CT scan, 2 were considered to have died from a primary brain damage, and 4 from expanding intracranial lesion, of the 15 patients with focal SAH, 3 were considered to have died from a primary brain damage, 9 from an expanding intracranial lesion and 2 from other intracranial complications, and of the 44 patients with diffuse SAH , 26 were considered to have died from a primary brain damage, 12 from expanding intracranial lesion and 2 from other intracranial complications.

Comments

Subarchnoid haemorrhage was seen in the CT scans 66% of this series of fatal cases 67/102 , in accord with the adverse prognosis associated with this finding. However there were no clear post-mortem findings that would explain either the origin of traumatic SAH or its adverse effects. Subarchnoid haemorrhage present without contusion was 11% of cases, and was not related to the occurrence of DAI, raised ICP or Hypoxic brain damage. Brain swelling was present in 47% of cases with no SAH, but in 62% of cases with SAH. No correlation existed with the pathologists opinion on the cause of death.

5.10 DISCUSSION

The aim in this study was to identify the correlation between abnormalities seen on the CT scan and the micro and macro-pathology found at post-mortem in head injured patients

Although it is the patients initial CT scan that usually provides the basis for classification of injury and for management decision, additional abnormalities develop in many severely injured patients and are particularly likely in fatal cases. Therefore, when a patient had two or more CT scans, we have analysed the worst finding. Thus in one study of the early scanning of head injured patients, virtually 100% of patients who deteriorated had an abnormal CT scan (108), however in another study the CT scan done at the time of deterioration was normal in 33% (109). Certainly the initial CT scan is no assurance that a haemorrhagic lesion will not develop, and 25% of delayed haemorrhages develop in patients whose first scan after injury is normal (110-111). An early repeat of the CT scan is valuable in detecting the progression of small lesions.

In this study in all the patients with who initially had a normal CT scan, the scan appearances deteriorated before death. No patient died with a normal CT scan. The initial CT scan seems to be less sensitive to detect the dynamic pathological process of the head injury, on the other hand CT scans may show transient abnormalities not giving rise to a fixed abnormality at the post-mortem.

There are several pathological lesions characteristic of closed head injury and most are usually detected by CT scan. Contusions usually appear on the surface of the anterior and inferior frontal and temporal lobes, they can take two forms haemorrhagic and non-haemorrhagic, but the later may be impossible to distinguish from the focal cerebral oedema. In this study contusions were manifested on the worst CT scan in about half of the patients 51%, but were found the contusion appeared in 87.3% at post-mortem. Some patients may die as a result of head injury without a contusion seen on the post-mortem, nevertheless the post-mortem pathological study is more sensitive to the presence of contusion than CT. The Contusion Index that was developed allows the extent of contusion in various part of the brain to be expressed quantitatively (45). The relative insensitivity of CT to contusion is well recognised and may reflect small size, partial volume with base of the skull, yet even moderate and severe lesion at post-mortem were not seen on CT scan, 79% of patients without contusion seen on the CT scan, showed moderate contusion on PM and 5.3% showed severe contusion
The observation of contusion on the worst CT scan was found to be associated with signs of raised ICP at the post-mortem in 92% of patients.

Surprisingly when there were appearances of SAH on the CT scan only, 77.6% showed contusion at PM, and 60% of patients with contusion at PM had no SAH on the CT scan. Contusion have been postulated to be the most likely source of traumatic SAH (75,76)

The patients with DAI pathologically confirmed have been reported to show different lesions on the CT scan. There may be single or multiple small haemorrhages in the

corpus callosum and or posterolateral quadrant of the brain stem (112,113). The CT scan can not directly detect white matter injury, but lesions typically associated with DAI were seen in 44.1% of patients, this finding is similar to that in other studies in which CT scan detected lesions in 40% causes with DAI (8). The sensitivity of PM to DAI depends directly upon the length of survival, at least 12-24 hours are needed for first abnormalities (axonal bulbs) to be detected by silver stain. After few days small clusters of microgalia are seen and Wallerian degeneration occurs in patients who survive for several weeks or months,

In this study DAI was found in only 29% of cases that underwent microscopical examinations and only 40% of patients who showed intraparenchymal lesions on the CT scan exhibited DAI on PM. Also a third of the patients with non surgically significant ICH showed DAI on PM. Conversely 60% of patients with parenchymal lesion thought to be markers for DAI showed no evidence at PM of axonal injury. In some this may have been because of the insensitivity of conventional to diffuse axonal injury at post-mortem. In some cases the CT lesions are presumably vascular disruption injuries occurring as a direct result of the initial injury and their presence can no longer be used to imply DAI.

Abnormalities on the CT scan are extremely useful in predicting raised ICP . Any abnormality in the symmetry of the ventricular system, basal cisterns and third ventricle is associated with increased probability of raised ICP in life (37,82,114). The raised ICP can be detected indirectly at PM by the signs of herniation of one or both parahippocampal gyri and Brainstem damage in the form of midline haemorrhage

and/or infarction. In this study the incidence of raised ICP at PM was 81.4%. similar to previous reports (113).

Nearly half of the patients with normal basal cisterns on the CT scan died with pathological evidence of raised ICP, this discrepancy may be because ICP is a dynamic processes, and not all patients were rescanned immediately before death. 85.7% of the patients with abnormal basal cisterns on CT showed signs at PM of raised ICP. This increased to 88.9% for patients with completely obliterated basal cisterns. Almost identical correlations were found with obliteration of the third ventricle. This means that the often simpler and more reliable observation of III ventricular obliteration has the same clinical significance as obliteration or compression of the basal cisterns.

A good correlation also found between ICH on CT scan and raised ICP at PM. 88.7% of patients with ICH manifested raised ICP at PM. however 95.5% of ICH considered not to be surgically significant showed raised ICP at PM. This led to the question of why these lesions were not operated upon, however such cases the raised ICP was due to an associated diffuse abnormality.

Hypoxic brain damage is a common pathological lesion in patients dying as a result of blunt head injury (115). Ischaemia on CT scan appears as an area of decreased density, but the overall accuracy of CT in identification of hypoxic brain damage is very limited (116). In this study the pathological incidence of the HBD was 90.2%, similar to other studies (107, 116). However there was no correlation between HBD at PM, and signs of raised ICP on CT. 50% of the patients with normal basal cisterns showed HBD at PM and 37% of those with basal cisterns compression had HBD at PM. This is

surprising because raised ICP adversely affects the cerebral blood flow by decreasing the cerebral perfusion, and pressure would be expected to lead to HBD. Furthermore there was no correlation between HBD and SAH. 51.4% of patients with no SAH on the CT had HBD at PM and 31.3% of patients with SAH on CT scan had HBD at PM. This is also surprising as experimental work indicates that traumatic SAH causes vasospasm which can cause ischaemia, and there is some evidence that Nimodipine improves the outcome in patients with traumatic SAH (71), as well as those with spontaneous rupture of aneurysms.

Brain swelling is defined as an increase in the volume of part or all the brain. Brain swelling may be severe enough to rise the ICP. The incidence of brain swelling at post-mortem was 53.9% in this study were 70.6% of patients with contusion on CT had brain swelling at PM. Brain swelling is an integral part of maturation of a contusion, due to physical disruption of the tissue with damage to the blood-brain barrier and loss of normal physiological regulation of the arterioles (117). Surprisingly only slightly more than half of the patients with signs of raised ICP showed brain swelling at PM, there was not a close correlation.

The TCDB classification, is increasingly widely used (37), in this study all the patients with DI IV on TCDB exhibited signs of raised ICP on PM , and more than 3/4 with DI III exhibited signs of raised ICP at PM. There was therefore a good correlation between the CT scan appearance of the midline shift and basal cisterns obliteration and the signs of raised ICP at PM. Surprisingly no subgroup in the TCDB correlated with the occurrence of HBD at PM.

This study has raised issues which need to be farther explained or re-examined. The divergence in some cases between CT signs and pathological evidence of DAI was intriguing. Previous studies have shown this in patients with basal ganglia lesions (112) and it is clear from mechanical work that a rotational force may be sufficient to cause vascular injury but insufficient to cause DAI , however this was not anticipation for "tissue tear" haemorrhages in corpus callosum and brain stem.

Over all this study indicates that there is :
No unique characteristic CT-pathological pattern, some expected associations were found, some surprisingly were not present.

The results do support a strong association between abnormality of the basal cisterns and third ventricle with signs of raised ICP at pathology, and also, between CT signs of a contusion with mass effect with the signs of raised ICP and contusion as identified at PM, between the subcortical lesion on CT and DAI at PM and, between the intracranial haematoma and the signs of raised ICP at PM.
Conversely association appeared to be lacking between, SAH and HBD, contusion or DAI at PM and abnormalities of the basal cisterns and third ventricle with HBD at PM.

What this study has shown is that the previously held idea that CT simply and directly shows living pathology must be in doubt. Conversely there are certain features on CT which cannot be explained by current pathological concept and finding and pathology must not necessarily be assumed to be a "gold standard".

CHAPTER VI

6.1 Conclusion

CT scanning has been employed widely in the management of head injured patients for nearly a 20 years. Therefore , enough time has elapsed to test its reliability and sensitivity; and as mentioned before, the reliability and the validity of the CT scan are of utmost value to the diagnosis, treatment and prognosis of head injured patients. From the study of the reliability of the CT scan in head injury which is contained in chapter IV in this thesis, we found :

1- Agreement about the presence of post-traumatic abnormalities on CT scan is significant but for most observation, of only a moderate degree.

2- Relatively high level of agreement for assignment of a scan as being either normal or showing a surgically significant mass lesion, this is particularly crucial to clinical decision making.

3- Abnormalities of CSF spaces are important in indicating brain swelling and raised intracranial pressure; assessments based upon the state of the III ventricle appeared to be more reliable than those based upon the cisterns.

4- Traumatic subarchnoid haemorrhage has an adverse prognosis and potentially implications for treatment but assessment of its presence and distribution is not reliable and a more precise definition is needed.

5-The greater consistency found amongst experienced observers indicates that training and education are able to improve the reliability of observations.

The question of how well CT scan depicts the pathology of head injury, that is the validity of CT scanning , was studied in thesis by comparison of the CT scan observation with the results of detailed neuropathology of fatal cases, contained in chapters V ,we found :

1- There should be caution about relating CT scan results to presence or absence of specific pathological features.

2- Strong support for a close, relation of CT scan results to dynamic events such as raised ICP sufficiently severe and persisting to leave a mark in the brain, but not to Hypoxic Brain Damage often assumed to be natural correlate of raised Intracranial Pressure

3- Classification of head injury based on CT scan may be useful, on empirical grounds rather than because it clearly separates different neuropathological entities.

4- CT scan is not a "living" post-mortem.

Post-mortem is not "gold standard". In research, use both CT and Pathology as often as possible.

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APPENDIX 1

THE PROFORMA USED BY THE OBSERVERS TO CODE THE CT SCAN FINDING IN THE OBSERVER VARIABILITY STUDY.

CT SCAN-HEAD INJURY INTERPRETATION

Pathology Study No. [ ][ ][ ][ ][ ][ ]

INS no. [ ][ ][ ]

Patient surname [ ][ ][ ][ ][ ][ ][ ][ ][ ][ ][ ][ ] Age: [ ][ ]

Date of Admission Scan [ ][ ][ ][ ][ ][ ]

X-ray Number [ ][ ][ ][ ][ ][ ]

Total No. of Scans [ ][ ]

Number in Sequence [ ][ ]

Date of Scan [ ][ ][ ][ ][ ][ ]

Type of Scan [ ]

1 = 10 10
2 = Tomscan
3 = Elscint

Major Diagnosis [ ]

1 = normal 2 = surgical (mass lesion)
3 = CT scan abnormal, no surgical mass lesion
4 = as 3 with CT signs of increased ICP

Assessment using Traumatic Coma Data Bank Classification

1 = Diffuse injury I
2 = Diffuse Injury II
3 = Diffuse Injury III
4 = Diffuse Injury IV
5 = Mass lesion for evacuation or evacuated
6 = Mass lesion not for evacuation

[ ]

Assessment using HIT II Classification of haematoma and contusion

1 = no 2 = yes - not surgically significant
3 = yes - surgically significant

	R	L	PF
Extradural haematoma	☐	☐	☐
Subdural haematoma	☐	☐	☐
Intracerebral haematoma	☐	☐	☐
Contusion* (no mass effect)	☐	☐	☐
Contusion* (mass effect)	☐	☐	☐

* Contusion = high or mixed density lesion involving cortex

Assessment of presence intraparcenchymal lesions
1 = no 2 = yes, single 3 = yes, multiple

	R	L	ML
Subcortical	☐	☐	☐
Deep white matter	☐	☐	☐
Basal ganglia	☐	☐	☐
Brainstem	☐	☐	☐
Cerebellum	☐	☐	☐

Assessment of CT Scans for signs of raised ICP ☐

Midline shift 1 = no 2 = yes < 5mm 3 = yes > 5mm

Measurement in mm ☐☐

Basal Cisterns
1 = normal 2 = compressed (partially obliterated)
3 = obliterated 4 = dilated
5 = not identified (due to technical factors)
(2-3-4 may have blood in cistern) ☐

Body of III Ventricle
1 = normal 2 = compressed (partially obliterated)
3 = obliterated (anterior recess may be visible)
4 = dilated ☐

Lateral Ventricles R L
1 = normal 2 = compressed relative to normal
3 = compressed relative to contralateral
4 = dilated relative to normal
5 = dilated relative to contralateral ☐ ☐

Intraventricular SAH
1 = no 2 = one ventricle 3 = more than one ventricle ☐

HIT II
1 = No SAH 2 = diffuse SAH
3 = focal SAH 4 = blood at tentorium ☐

GINS Classification
1 = No SAH 2 = Isolated tentorial blood
3 = focal basal SAH 4 = Diffue basal SAH
5 = Focal cortical SAH (Sylvian or sulcui)
6 = Diffuse cortical SAH 7 =(3 or 4) + (5 or 6) ☐

APPENDIX 2

THE OVERALL RAW DATA OF THE OBSERVER VARIABILITY

OVER ALL DISTRIBUTION OF OBSERVERS USING "MAJOR DIAGNOSIS"

CT scan No.	Normal CT Scan	Surgical Mass Lesion	Abnormal CT No Surgical Mass Lesion	Abn. CT ,No Surg. Mass Lesion, Sign of Raised ICP	Data Omitted
1	5	-	1	3	4
2	1	-	11	-	1
3	-	5	4	1	3
4	-	2	7	-	4
5	-	1	8	1	3
6	-	-	-	4	9
7	-	12	-	-	1
8	-	1	10	-	2
9	-	10	-	-	3
10	-	12	-	-	1
11	-	3	3	-	7
12	-	4	7	-	2
13	-	4	6	-	3
14	-	10	1	-	2
15	-	-	9	-	4
16	-	2	6	2	3
17	-	12	-	-	1
18	-	2	7	1	3
19	-	10	-	-	3
20	-	8	2	1	2
21	7	-	4	-	2
22	-	-	6	2	5
23	9	-	1	1	2
24	1	-	5	5	2
25	-	6	4	-	3
26	-	9	-	-	4
27	7	-	1	1	4
28	-	2	7	-	4

OVER ALL DISTRIBUTION OF OBSERVERS USING TRAUMATIC COMA DATA BANK CLASSIFICATION

CT scan No.	*DI I*	*DI II*	*DI III*	*DI IV*	*DI V*	*DI VI*	*Data Omitted*
1	-	6	-	-	-	3	4
2	-	7	1			2	3
3	-	-	-	-	12	-	1
4	-	-	-	-	-	12	1
5	7	1	1	-	-	-	4
6	-	1	1		8	1	2
7	1	-	10	-	-	-	2
8	-	7	-	-	7	1	3
9	-	-	4	-	-	-	9
10	-	-	-	-	11	1	1
11	-	6	1	-	-	2	4
12	6	4	-	-	-	-	3
13	1	4	2	-	-	1	5
14	-	-	-	-	2	4	7
15	-	-	-	-	4	1	8
16	-	-	-	-	10	-	3
17	-	1	-	-	-	8	4
18	10	-	1	-	-	-	2
19	1	8	-	-	-	3	1
20	-	3	-	-	4	3	3
21	-	-	2	-	7	2	2
22	-	10	-	-	-	1	2
23	-	6	1	-	-	2	4
24	-	9	-	-	-	-	4
25	-	-	-	-	10	-	3
26	-	6	-	-	-	3	4
27	10	1	-	-	-	-	2
28	-	7	-	-	-	4	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR EPIDURAL HAEMATOMA ON RIGHT SIDE

CT scan No.	*No EDH*	*EDH not Surgically Significant*	*EDH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	11	1	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	9	1	-	3
13	8	-	-	5
14	6	-	-	7
15	-	-	5	8
16	8	2	-	3
17	10	-	-	3
18	11	-	-	2
19	10	2	-	1
20	7	-	3	3
21	9	2	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	-	2	8	3
26	10	-	-	3
27	11	-	-	3
28	5	6	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR EPIDURAL HAEMATOMA ON LEFT SIDE

CT scan No.	*No EDH*	*EDH not Surgically Significant*	*EDH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	-	-	12	1
4	11	-	1	1
5	9	-	-	4
6	-	-	11	2
7	11	-	-	2
8	9	-	1	3
9	4	-	-	9
10	10	1	1	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	5	1	-	7
15	4	1	-	8
16	10	-	-	3
17	9	1	-	3
18	11	-	-	2
19	12	-	-	1
20	6	1	3	3
21	11	-	2	-
22	11	-	-	2
23	10	-	-	3
24	8	2	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR EPIDURAL HAEMATOMA ON POSTERIOR FOSSA

CT scan No.	*No EDH*	*EDH not Surgically Significant*	*EDH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	9	1	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	5	-	-	8
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	9	-	1	3
21	10	1	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR SUBDURAL HAEMATOMA ON RIGHT SIDE

CT scan No.	*No SDH*	*SDH not Surgically Significant*	*SDH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	7	4	1	2
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	9	1	-	3
9	4	-	-	9
10	9	1	2	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	5	1	-	6
15	5	-	-	8
16	-	-	20	3
17	10	-	-	3
18	11	-	-	2
19	11	1	-	1
20	9	-	1	3
21	8	1	2	2
22	11	-	-	2
23	10	-	-	3
24	8	2	-	3
25	7	1	2	3
26	8	2	-	3
27	11	-	-	2
28	10	1	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR SUBDURAL HAEMATOMA ON LEFT SIDE

CT scan No.	*No SDH*	*SDH not Surgically Significant*	*SDH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	-	1	11	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	6	4	-	3
9	4	-	-	9
10	2	2	8	1
11	10	-	-	3
12	10	-	-	3
13	7	1	-	5
14	3	3	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	11	1	-	1
20	8	1	1	3
21	10	1	-	2
22	11	-	-	2
23	10	-	-	3
24	2	8	-	3
25	10	-	-	3
26	8	2	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR SUBDURAL HAEMATOMA ON POSTERIOR FOSSA

CT scan No.	*No SDH*	*SDH not Surgically Significant*	*SDH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	9	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	10	1	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRACEREBRAL HAEMATOMA ON RIGHT SIDE

CT scan No.	*No ICH*	*ICH not Surgically Significant*	*ICH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	5	5	-	3
3	10	2	-	1
4	12	-	-	1
5	9	-	-	4
6	9	2	-	2
7	8	3	-	2
8	10	-	-	3
9	1	3	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	4	1	2	6
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	8	-	2	3
21	4	3	4	2
22	3	8	-	2
23	7	3	-	3
24	7	3	-	3
25	9	1	-	3
26	8	2	-	3
27	9	2	-	2
28	1	10	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRACEREBRAL HAEMATOMA ON LEFT SIDE

CT scan No.	*No ICH*	*ICH not Surgically Significant*	*ICH Surgically Significant*	*Data Omitted*
1	3	6	-	4
2	6	4	-	3
3	10	2	-	1
4	11	-	1	1
5	9	-	-	4
6	10	1	-	2
7	9	2	-	2
8	6	3	1	3
9	4	-	-	9
10	11	1	-	1
11	9	1	-	3
12	10	-	-	3
13	6	2	-	5
14	2	3	1	7
15	5	-	-	8
16	10	-	-	3
17	5	4	1	3
18	11	-	-	2
19	12	-	-	1
20	7	1	2	3
21	11	-	-	2
22	7	4	-	2
23	10	-	-	3
24	9	1	-	3
25	10	-	-	3
26	6	4	-	3
27	10	1	-	-
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRACEREBRAL HAEMATOMA ON POSTERIOR FOSSA

CT scan No.	*No ICH*	*ICH not Surgically Significant*	*ICH Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	2	2	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	10	1	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	10	1	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR CONTUSIONS WITH NO MASS EFFECT ON RIGHT SIDE

CT scan No.	*No Contusion*	*Contusion not Surgically Significant*	*Contusion Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	9	1	-	3
3	-	12	-	1
4	12	-	-	1
5	9	-	-	4
6	2	9	-	2
7	7	4	-	2
8	10	-	-	3
9	3	1	-	9
10	10	2	-	1
11	8	2	-	3
12	10	-	-	3
13	8	-	-	5
14	2	4	-	7
15	5	-	-	8
16	9	1	-	3
17	10	-	-	3
18	11	-	-	2
19	1	11	-	1
20	5	4	1	3
21	4	5	2	2
22	11	-	-	2
23	4	6	-	3
24	-	10	-	3
25	7	2	-	3
26	4	6	-	3
27	11	-	-	2
28	3	8	-	1

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR CONTUSIONS WITH NO MASS EFFECT ON LEFT SIDE

CT scan No.	*No Contusion*	*Contusion not Surgically Significant*	*Contusion Surgically Significant*	*Data Omitted*
1	6	3	-	4
2	9	1	-	3
3	7	5	-	1
4	12	-	-	1
5	8	1	-	4
6	8	1	-	2
7	6	5	-	2
8	7	3	-	3
9	4	-	-	9
10	6	5	1	1
11	9	1	-	3
12	10	-	-	3
13	4	4	-	5
14	3	3	-	7
15	5	-	-	8
16	10	-	-	3
17	7	3	-	3
18	11	-	-	2
19	12	-	-	1
20	7	2	1	3
21	9	2	-	2
22	9	1	-	3
23	10	-	-	3
24	1	9	-	3
25	10	-	-	3
26	5	5	-	3
27	11	-	-	2
28	10	1	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR CONTUSIONS WITH NO MASS EFFECT ON POSTERIOR FOSSA

CT scan No.	*No Contusion*	*Contusion not Surgically Significant*	*Contusion Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	11	1	-	1
4	12	-	-	1
5	9	-	-	4
6	12	-	-	2
7	12	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	7	1	-	5
14	5	1	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	9	1	-	3
21	9	2	-	2
22	10	1	-	2
23	9	1	-	3
24	10	-	-	3
25	10	-	-	3
26	11	-	-	2
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR CONTUSIONS WITH MASS EFFECT ON RIGHT SIDE

CT scan No.	*No Contusion*	*Contusion not Surgically Significant*	*Contusion Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	9	-	1	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	3	2	1	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	5	1	4	3
21	6	1	4	2
22	10	1	-	2
23	8	1	1	3
24	10	-	-	3
25	9	-	1	3
26	10	-	-	3
27	11	-	-	2
28	9	1	1	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR CONTUSIONS WITH MASS EFFECT ON LEFT SIDE

CT scan No.	*No Contusion*	*Contusion not Surgically Significant*	*Contusion Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	12	-	-	2
8	6	1	3	3
9	4	-	-	9
10	12	-	1	1
11	7	3	-	3
12	10	-	-	3
13	7	1	-	5
14	4	-	2	7
15	5	-	-	5
16	10	-	-	3
17	3	5	2	3
18	11	-	-	2
19	12	-	-	1
20	5	1	4	3
21	10	1	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	9	1	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR CONTUSIONS WITH MASS EFFECT ON POSTERIOR FOSSA

CT scan No.	*No Contusion*	*Contusion not Surgically Significant*	*Contusion Surgically Significant*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	3
6	12	-	-	2
7	12	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	9	1	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON RIGHT SUBCORTICAL

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	10	-	-	3
2	10	-	-	3
3	4	8	-	1
4	12	-	-	1
5	10	-	-	3
6	3	8	-	2
7	4	6	-	3
8	9	1	-	3
9	1	3	-	9
10	11	1	-	1
11	9	1	-	3
12	9	1	-	3
13	7	1	-	5
14	3	3	-	7
15	5	-	-	8
16	9	1	-	3
17	10	-	-	3
18	11	-	-	2
19	8	4	-	1
20	4	6	-	3
21	4	7	-	2
22	10	1	-	2
23	4	6	-	3
24	3	7	-	3
25	2	8	-	3
26	6	4	-	3
27	10	1	-	2
28	6	5	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON LEFT SUBCORTICAL

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	7	2	-	4
2	1	9	-	3
3	4	8	-	1
4	12	-	-	1
5	8	1	-	4
6	10	1	-	2
7	6	5	-	2
8	6	4	-	3
9	4	-	-	9
10	7	5	-	1
11	3	7	-	3
12	10	-	-	3
13	3	5	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	5	5	-	3
18	11	-	-	2
19	12	-	-	1
20	5	5	-	3
21	5	6	-	2
22	1	10	-	2
23	8	2	-	3
24	4	6	-	3
25	10	-	-	3
26	4	6	-	3
27	11	-	-	2
28	10	1	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON MIDLINE SUBCORTICAL

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	8	1	-	4
6	11	-	-	2
7	10	-	-	3
8	9	1	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	8	2	-	3
21	11	-	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON RIGHT DEEP WHITE MATTER

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	9	1	-	3
3	12	-	-	1
4	11	1	-	1
5	8	1	-	4
6	11	-	-	2
7	10	2	-	1
8	10	-	-	3
9	2	2	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	3	2	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	5	5	-	3
21	7	4	-	2
22	6	5	-	2
23	8	2	-	3
24	9	1	-	3
25	8	2	-	3
26	10	-	-	3
27	9	2	-	2
28	4	7	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON LEFT DEEP WHITE MATTER

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	4	5	-	4
2	7	3	-	3
3	12	-	-	1
4	9	3	-	1
5	8	1	-	4
6	11	-	-	2
7	9	2	-	2
8	6	4	-	3
9	4	-	-	9
10	9	3	-	1
11	5	5	-	3
12	8	2	-	3
13	8	-	-	5
14	2	4	-	7
15	5	-	-	8
16	10	-	-	3
17	8	2	-	3
18	11	-	-	2
19	11	1	-	1
20	5	5	-	3
21	10	1	-	2
22	5	6	-	2
23	8	2	-	3
24	9	1	-	3
25	10	-	-	3
26	9	1	-	3
27	9	2	-	9
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON MID-LINE LEFT DEEP WHITE MATTER

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	9	1	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	9	1	-	3
21	10	1	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON RIGHT BASAL GANGLIA

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	-	10	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	10	1	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	5	1	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	-	11	-	2
23	7	3	-	3
24	10	-	-	3
25	10	-	-	3
26	9	1	-	3
27	11	1	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON LEFT BASAL GANGLIA

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	9	3	-	1
5	10	-	-	3
6	11	-	-	2
7	9	2	-	2
8	9	1	-	3
9	4	-	-	9
10	9	3	-	1
11	1	9	-	3
12	10	-	-	3
13	8	-	-	5
14	3	3	-	7
15	5	-	-	5
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	5	6	-	2
23	7	3	-	3
24	10	-	-	3
25	10	-	-	3
26	9	1	-	3
27	10	1	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON MID-LINE BASAL GANGLIA

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON RIGHT BRAINSTEM

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	8	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON LEFT BRAINSTEM

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	2	2	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON MIDLINE BRAINSTEM

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	10	2	-	1
11	10	-	-	3
12	10	-	-	3
13	5	3	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	9	1	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "IIIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON RIGHT CEREBELLUM

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	8	2	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	10	1	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON LEFT CEREBELLUM

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	11	1	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	10	-	-	3
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	9	1	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT II CLASSIFICATION" FOR INTRAPARENCHYMAL LSEIONS ON MIDLINE CEREBELLUM

CT scan No.	*No Intraparenchymal Lesions*	*Single Intraparenchymal Lesions*	*Multiple Intraparenchymal Lesions*	*Data Omitted*
1	9	-	-	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	11	-	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	12	-	-	1
11	10	-	-	3
12	9	1	-	3
13	8	-	-	5
14	6	-	2	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	10	-	-	3
26	10	-	-	3
27	9	2	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS ON MIDLINE SHIFT

CT scan No.	*No Midline shift*	*Midline shift <5mm*	*Midline shift >5mm*	*Data Omitted*
1	9	-	-	4
2	7	4	-	2
3	-	9	3	1
4	-	-	12	1
5	9	-	-	4
6	7	4	-	2
7	11	-	-	2
8	4	2	4	3
9	1	3	-	9
10	-	-	12	1
11	7	3	-	3
12	10	-	-	3
13	6	2	-	5
14	3	1	2	7
15	-	1	4	8
16	-	-	10	3
17	9	1	-	3
18	10	1	-	2
19	12	-	-	1
20	5	4	1	3
21	2	8	1	2
22	11	-	-	2
23	10	-	-	3
24	10	-	-	3
25	-	2	8	3
26	10	-	-	3
27	11	-	-	2
28	9	2	-	2

OVER ALL DISTRIBUTION OF OBSERVERS ON THE STATE OF BASAL CISTERNS

CT scan No.	*Normal*	*Compressed*	*Obliterated*	*Dilated*	*Not Ident.*	*Data Omitted*
1	9	-	-	-	-	4
2	6	4	-	-	-	3
3	5	6	1	-	-	1
4	-	2	9	-	-	2
5	8	1	-	-	-	4
6	9	2	-	-	-	2
7	1	-	10	-	-	2
8	4	4	2	-	-	3
9	-	-	4	-	-	9
10	-	-	12	-	-	1
11	8	2	-	-	-	3
12	6	4	-	-	-	3
13	2	5	1	-	-	5
14	6	-	-	-	-	7
15	-	3	2	-	-	8
16	-	4	6	-	-	3
17	9	1	-	-	-	3
18	11	-	-	-	-	2
19	12	-	-	-	-	1
20	6	2	2	-	-	3
21	-	6	5	-	-	2
22	11	-	-	-	-	2
23	9	1	-	-	-	3
24	10	-	-	-	-	3
25	7	2	1	-	-	3
26	10	-	-	-	-	3
27	11	-	-	-	-	2
28	9	2	-	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS ON THE BODY OF THIRD VENTRICLE

CT scan No.	Normal	Compressed	Obliterated	Dilated	Data Omitted
1	9	-	-	-	4
2	2	8	-	-	3
3	4	8	-	-	1
4	-	2	10	-	1
5	8	1	-	-	4
6	3	8	-	-	2
7	1	1	9	-	2
8	6	3	1	-	3
9	-	-	3	-	10
10	-	-	12	-	1
11	9	1	-	-	3
12	6	4	-	-	3
13	2	5	1	-	5
14	6	-	-	-	7
15	-	-	5	-	8
16	-	1	9	-	3
17	10	-	-	-	3
18	11	-	-	-	2
19	12	-	-	-	1
20	5	5	-	-	3
21	-	2	9	-	2
22	11	-	-	-	2
23	10	-	-	-	3
24	10	-	-	-	3
25	1	9	-	-	3
26	10	-	-	-	3
27	11	-	-	-	2
28	11	-	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS ON THE STATE OF THE RIGHT LATERAL VENTRICLE

CT scan No.	*Normal*	*Compressed relative to Normal*	*Compresed relative to Contralateral*	*Dilated relative to Normal*	*Dilated relative to Contralateral*	*Data Omitted*
1	8	1	-	-	-	4
2	2	8	-	-	-	3
3	8	-	-	1	3	1
4	-	-	-	9	3	1
5	8	-	1	-	-	4
6	8	3	-	-	-	2
7	2	9	-	-	-	2
8	7	1	-	-	2	3
9	3	1	-	-	-	10
10	-	-	-	10	2	1
11	10	-	-	-	-	3
12	10	-	-	-	-	3
13	5	3	-	-	-	6
14	6	-	-	1	-	7
15	-	6	2	-	-	5
16	-	6	1	-	-	6
17	9	-	-	1	-	3
18	9	-	-	2	-	2
19	11	1	-	-	-	1
20	7	3	-	-	-	3
21	1	10	-	-	-	2
22	6	2	1	1	-	3
23	9	-	-	-	1	3
24	8	-	-	2	-	3
25	-	7	2	1	-	3
26	9	-	-	1	-	3
27	10	1	-	-	-	2
28	9	-	-	-	2	2

OVER ALL DISTRIBUTION OF OBSERVERS ON THE STATE OF THE LEFT LATERAL VENTRICLE

CT scan No.	*Normal*	*Compressed relative to Normal*	*Compresed relative to Contralateral*	*Dilated relative to Normal*	*Dilated relative to Contralateral*	*Data Omitted*
1	9	-	-	-	-	4
2	5	5	-	-	-	3
3	-	8	4	-	-	1
4	-	11	1	-	-	1
5	9	-	-	-	-	4
6	1	9	1	-	-	2
7	2	9	-	-	-	2
8	2	6	1	1	-	3
9	3	1	-	-	-	9
10	-	11	1	-	-	1
11	-	10	-	-	-	3
12	10	-	-	-	-	3
13	7	2	-	-	-	4
14	4	2	-	-	-	7
15	-	-	-	3	2	8
16	-	1	-	9	-	3
17	1	3	5	1	-	3
18	8	-	2	1	-	2
19	11	1	-	-	-	1
20	5	5	-	-	-	3
21	1	10	-	-	-	2
22	8	-	-	2	-	3
23	10	-	-	-	-	3
24	8	-	-	2	-	3
25	5	1	-	1	3	3
26	9	-	-	1	-	3
27	10	1	-	-	-	2
28	10	-	1	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS ON THE INTRAVENTRICULAR HAEMORRHAGE

CT scan No.	*No IVH*	*Blood in one Ventricle*	*Blood in more than one Ventricle*	*Data Omitted*
1	-	2	7	4
2	10	-	-	3
3	12	-	-	1
4	12	-	-	1
5	9	-	-	4
6	5	6	-	2
7	11	-	-	2
8	10	-	-	3
9	4	-	-	9
10	7	4	1	1
11	9	1	-	3
12	11	-	-	2
13	8	-	-	5
14	6	-	-	7
15	5	-	-	8
16	10	-	-	3
17	10	-	-	3
18	11	-	-	2
19	12	-	-	1
20	10	-	-	3
21	11	-	-	2
22	1	2	8	2
23	10	-	-	3
24	3	5	2	3
25	10	-	-	3
26	11	-	-	2
27	11	-	-	2
28	11	-	-	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "HIT H"ON THE DISTRIBUTION OF SUBARCHNOID HAEMORRHAGE

CT scan No.	*No SAH*	*Diffuse SAH*	*Focal SAH*	*Blood at Tentorium*	*Data Omitted*
1	4	-	4	1	4
2	8	-	2	-	3
3	10	-	2	-	1
4	1	8	3	-	1
5	8	1	-	-	4
6	4	5	2	-	2
7	1	5	1	4	2
8	5	2	3	-	3
9	3	1	-	-	9
10	1	5	3	3	1
11	7	-	3	-	3
12	9	1	-	-	3
13	5	1	-	2	5
14	4	1	1	-	7
15	3	1	1	-	8
16	10	-	-	-	3
17	10	-	-	-	3
18	11	-	-	-	2
19	12	-	-	-	1
20	6	1	-	3	3
21	5	3	-	3	2
22	8	-	3	-	2
23	2	3	4	1	3
24	-	9	1	-	3
25	4	-	5	1	3
26	10	-	-	-	3
27	11	-	-	-	2
28	8	-	2	1	2

OVER ALL DISTRIBUTION OF OBSERVERS USING "FISHER CLASSIFICATION"ON THE DISTRIBUTION OF SUBARCHNOID HAEMORRHAGE

CT scav No.	*No SAH*	*Diffuse or thin, no vertical layer more than 1mm.*	*Clote or vertical layer more than 1mm.*	*Intracerebral or Intraventricul ar clots*	*Data Omitted*
1	2	-	3	4	4
2	9	-	-	1	3
3	11	1	-	-	1
4	1	9	1	-	2
5	8	1	-	-	4
6	4	5	-	2	2
7	2	8	1	-	2
8	3	2	2	-	4
9	3	1	-	-	9
10	2	6	2	2	1
11	8	1	-	1	3
12	9	1	-	-	3
13	5	1	1	-	6
14	4	1	1	-	7
15	3	2	-	-	8
16	10	-	-	-	3
17	10	-	-	-	3
18	11	-	-	-	2
19	12	-	-	-	1
20	5	4	-	1	3
21	5	2	3	-	3
22	7	-	-	5	1
23	4	4	2	-	3
24	-	7	1	2	3
25	4	5	-	-	4
26	10	-	-	-	3
27	12	-	-	-	1
28	8	1	-	1	3

OVER ALL DISTRIBUTION OF OBSERVERS USING "GINS CLASSIFICATION"ON THE DISTRIBUTION OF SUBARCHNOID HAEMORRHAGE

CT scan No.	*No SAH*	*Isolated Tentorial Blood*	*Focal Basal SAH*	*Diffuse Basal SAH*	*Focal Cortical SAH (Sylvian or Sulcal)*	*Diffuse Cortical SAH*	*(3 or 4) + (5 or 6)*	*Data Omitted*
1	7	1	-	1	-	-	-	4
2	8	-	-	-	2	-	-	3
3	10	-	-	-	2	-	-	1
4	1	-	-	4	1	-	6	1
5	8	-	-	-	-	1	-	4
6	4	-	-	-	2	2	3	2
7	1	4	-	2	-	1	3	2
8	3	-	-	-	2	2	1	3
9	3	-	-	-	-	1	-	9
10	1	-	3	3	1	-	4	1
11	7	-	-	-	3	-	-	3
12	9	-	-	-	-	1	-	3
13	5	2	-	-	-	-	1	5
14	4	-	-	-	1	1	-	7
15	3	-	-	-	1	1	-	8
16	10	-	-	-	-	-	-	3
17	9	1	-	-	-	-	-	3
18	11	-	-	-	-	-	-	2
19	12	-	-	-	-	-	-	1
20	5	3	-	1	1	-	-	3
21	5	3	-	2	-	1	-	2
22	8	-	2	-	-	-	-	3
23	1	1	4	1	1	-	2	3
24	-	-	-	-	-	9	1	3
25	4	1	-	-	5	-	-	3
26	10	-	-	-	-	-	-	3
27	11	-	-	-	-	-	-	2
28	8	1	-	-	2	-	-	2

APPENDIX 3

THE PROFORMA USED FOR SCORING CT SCAN FILMS IN CHAPTER V

CT SCAN-HEAD INJURY INTERPRETATION

Pathology Study No. [| | | | | |]

INS no. [| | |]

Patient surname [| | | | | | | | | | | |] Age: [| |]

Date of Admission Scan [| | | | | |]

X-ray Number [| | | | | |]

Total No. of Scans [| |]

Number in Sequence [| |]

Date of Scan [| | | | | |]

Type of Scan [|]

1 = 10 10
2 = Tomscan
3 = Elscint

Major Diagnosis [|]

1 = normal 2 = surgical (mass lesion)
3 = CT scan abnormal, no surgical mass lesion
4 = as 3 with CT signs of increased ICP

Assessment using Traumatic Coma Data Bank Classification

1 = Diffuse injury I
2 = Diffuse Injury II
3 = Diffuse Injury III
4 = Diffuse Injury IV
5 = Mass lesion for evacuation or evacuated
6 = Mass lesion not for evacuation

[|]

Assessment of CT Scans for signs of raised ICP ☐

Midline shift 1 = no 2 = yes < 5mm 3 = yes > 5mm

Measurement in mm ☐☐

Basal Cisterns
1 = normal 2 = compressed (partially obliterated)
3 = obliterated 4 = dilated
5 = not identified (due to technical factors)
(2-3-4 may have blood in cistern) ☐

Body of III Ventricle
1 = normal 2 = compressed (partially obliterated)
3 = obliterated (anterior recess may be visible)
4 = dilated ☐

Lateral Ventricles R L
1 = normal 2 = compressed relative to normal
3 = compressed relative to contralateral
4 = dilated relative to normal
5 = dilated relative to contralateral ☐ ☐

Intraventricular SAH
1 = no 2 = one ventricle 3 = more than one ventricle ☐

HIT II
1 = No SAH 2 = diffuse SAH
3 = focal SAH 4 = blood at tentorium ☐

GINS Classification
1 = No SAH 2 = Isolated tentorial blood
3 = focal basal SAH 4 = Diffue basal SAH
5 = Focal cortical SAH (Sylvian or sulcui)
6 = Diffuse cortical SAH 7 =(3 or 4) + (5 or 6) ☐

Mass effect after surgery

No = 1 Same as Pre-op = 2 <Pre-op = 3 >Pre-op = 4 ☐

Arterial Territory Reduced Attenuation

Yes = 2 No = 1

	R	L
ACA	☐	☐
MCA	☐	☐
PCA	☐	☐
Watershed	☐	☐
Basilar	☐	☐

Diffuse Reduced Attenuation

Yes = 2 No = 1

R	L	PF
☐	☐	☐

Distribution of SAH

Basal
No = 1
R = 2
L = 3
Both = 4
☐

Cortical
No = 1
R = 2
L = 3
Both = 4
☐

APPENDIX 4

THE PROFORMA USED FOR CODING THE POSTMORTEM RESULTS IN CHAPTER V

Study Number: ___ ___ ___

PM Case Number: ___ ___ ___

INS Unit Number: ___ ___ ___ ___ ___ ___

HIRS: ___ ___ ___ ___

TALK AND DIE PATHOLOGY

1. Extracranial injuries.
   No : 1
   Yes : 2
   Unknown : 9

Soft Tissue: - Neck ___

Related to cervical vertebrae ___

Thorax ___

Abdomen ___

Spinal Cord ___

Fracture: - Facial ___

Vertebra(e) ___

Ribs ___

Single Long Bone ___

Multiple Long Bone ___

Pelvis ___

Other ___

2. Fracture of skull.

No : 1
Yes : 2
Unknown : 9

	L	R	M
Frontal			
Temporal			
Parietal			
Occipital			
Basal			

3. Haemorrhage.

None: 1 External: 5 Unknown: 9
Thoracic: 2 Associated with
Abdominal: 3 Long bone fracture 6
Gastro-intestinal: 4 Combination: 7

4. Contusions.

Absent or Mild: 1 Severe: 3
Moderate: 2 Unknown: 9

5. Intracranial Haematoma.

Absent or < 2 cm diameter/ < 35 ml: 1
> 2 cm / > 35 ml, not evacuated: 2
> 2 cm / > 35 ml, evacuated: 3
Unknown: 4

	L	R
Supratentorial		
Extradural		
Subdural (isolated)		
Intracerebral (isolated)		
Frontal		
Temporal		
Parietal		
Occipital		

	L	R
Basal Ganglia		
Burst Lobe	L	R
Frontal		
Temporal		
Parietal		
Occipital		
Intratentorial		
Extradural		
Subdural (isolated)		
(isolated)		
Intracerebellar		
Burst Lobe		

6. Raised Intracranial Pressure.

No: 1
Yes: 2
Unknown: 9

7. Hypoxic Brain Damage.

Absent or minimal: 1
Moderate: 2
Severe: 3
Unknown: 9

8. Pattern of Hypoxic damage.

No hypoxia: 1
Arterial territory infarction: 2
Boundary zone infarction: 3
Diffuse of multifocal: 4
2 + 3: 5
3 + 4: 6
2 + 3 + 4: 7
Unknown: 9

9. Diffuse axonal injury. ___

None: 1
Mild - Grade 1: 2
Moderate - Grade 2: 3
Severe - Grade 3: 4
Unknown: 9

10. Brain swelling ___

Absent: 1
Related to contusions: 2
Related to intracranial haematoma: 3
Related to hypoxic brain damage: 4
Related to combination: 5
Other: 6
Unknown: 7

11. Internal carotid arteries: ___

Normal: 1
Thrombosis: 2
Dissection: 3
Unknown: 9

12. Cerebral fat embolism. ___

No: 1
Yes: 2
Unknown: 9

13. Intracranial infection. ___

No: 1
Yes: 2
Unknown: 9

14. Pathologist's assessment of cause of death. ___

(Allocate total of 5 points between causes)

Primary Brain Damage ___

Expanding Intracranial Lesion ___

Other Intracranial Complications ___

Extracranial Complications ___

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