A Study of spontaneous intracerebral haemorrhage-clinical profile and outcome

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CERTIFICATE

This is to certify that this dissertation entitled **"A study of Spontaneous intracerebral haemorrhage-clinical profile and outcome"** submitted by **Dr.S.CHITRAMBALAM** appearing for **D.M.,Branch-I Neurology** Degree examination in August 2009 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled "**A study of spontaneous intracerebral haemorrhage-clinical profile and outcome"** is done by me at Institute Of Neurology, Madras Medical College & Govt. General Hospital, Chennai, during 2006-2009 under the guidance& supervision of **Prof. V.NATARAJAN, M.D.D.M.**

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of D.M. Degree in Neurology.

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CONTENTS

INTRODUCTION

The word *stroke* is used to refer to a clinical syndrome, of presumed vascular origin, typified by rapidly developing signs of focal or global disturbance of cerebral functions lasting more than 24 hours or leading to death (World Health Organisation 1978)[33].

It affects between 174 and 216 people per 100,000 population in the UK each year (Mant *et al* 2004), and accounts for 11% of all deaths in England and Wales. Cerebral infarction accounts for 69% of strokes, primary haemorrhage for 13%, subarachnoid haemorrhage for 6%, and 12% are of uncertain type (Wolfe *et al* 2002). The risk of recurrent stroke within five years of a first stroke is between 30% and 43% (Mant *et al* 2004).

Extravasation of blood into the brain parenchyma was recognized as early as 1658 by Wepfer[61], although he saw the clot as an obstruction of 'vital spirits' rather than as the disease in itself, and subsequently by Morgagni[62]. The cause remained obscure, and to a large extent it still is.[77]

We have come a long way since the time when Charcot (1881) felt "that if apoplexy was not immediately fatal, most survivors only retained life at the expense of deplorable infirmities and perpetual confinement to bed."

With the arrival of computed tomography of the brain, diagnosis of intra-cerebral haemorrhage has been taken from the age of calculated speculation with details of clinical features, angiograms and 'bloody taps' to the present day of arrival of a definitive diagnosis in a matter of minutes. A significant reason for the decreasing trend of mortality in the intra-cerebral haemorrhage patients in the industrialized countries have also been due to the identification of the factors which might adversely affect the outcome, stratifying patients and instituting prompt acute stroke care.

Though medical management of intra-cerebral haemorrhage has been the way, surgical management has been known since the times of McEwen, who performed the first successful operation for intra-cerebral haematoma in 1883. Though no definite guidelines exist to decide between the two lines of management, the increasing availability of minimally invasive techniques like stereo-tactic aspiration may make this modality indispensable in the days to come.

But all is not well for a country like India, where there still exist pockets where CT brain is unheard of, stroke care centers unimaginable, and supply almost always short of demand. Thereby, identifying definite risk factors, designing and implementing policies to contain them, making prompt and accurate diagnosis, stratifying patients according to outcome predictors, and thus ensuring prompt referral of deserving critical patients to tertiary centers for intensive management, may be the need of the hour as we

wait to embrace the newer advances into our management protocols. Intracerebral hemorrhage accounts for approximately 10% of strokes and its clinical importance derives from its frequency and accompanying high mortality. Although the latter is strongly dependent on HEMATOMA SIZE and to a lesser extent, LOCATION, the overall mortality rate varies between 25% and 60%.[93] There has been a general decline since the 1980s in the incidence of stroke, including ICH, as a result of improved detection and treatment of hypertension. However, ICH continues to be a major health problem especially in those who lack hypertension treatment and the genetically predisposed.

A growing body of evidence suggests that genetic factors, such as the possession of the E2 and E4 alleles of the apolipoprotein E, play an important role in the occurrence of certain forms of ICH, such as lobar hemorrhages(O'Donnell et al., 2000).[98] Novel potential genetic factors predisposing to ICH continue to be added by experimental and clinical studies (Gould et al., 2006).[97]

Although it would appear intuitively that *evacuation of a hematoma might be beneficial,* surgical results have not been superior to those with medical measures alone (Waga and Yamamoto; Batjer et al; Juvela et al; Rabinstein et al).

A large, multicenter, randomized study involving 1,033 patients with supratentorial hemorrhage, under the auspices of the Surgical Trial in Intracerebral Haemorrhage (STICH) study reported by Mendelow and colleagues, has failed to show a benefit from early surgery on survival or neurologic functioning at 6 months. This

negative result extended to almost all levels of neurologic deficit and all age groups. In a post hoc analysis, clots that were small and close to the surface of the brain may have benefited from evacuation.

As a result, this approach has been virtually abandoned, but in a few instances with ongoing deterioration in young patients with hematomas that are easily accessible from the cortical surface, evacuation of the clot has been undertaken.[3] In contrast, the *surgical evacuation of cerebellar hematomas* is a generally accepted treatment and is a more urgent matter because of the proximity of the mass to the brainstem and the risk of abrupt progression to coma and respiratory failure.[3]

Mayer and coworkers studied the promising approach of administering clotting factor VII within 4 h of hemorrhage. In a preliminary study(phase I&II), survival was improved and there was a reduction in enlargement of the hematoma, but their subsequent series(phase III FAST trial) has failed to confirm the benefit on survival so that infusion of factor VII is not currently part of routine practice .[93] Therefore, the surgical management of ICH is debatable because the assessment of potential interventions awaits the completion of properly designed prospective clinical trials.

AIMS AND OBJECTIVES

1. To assess clinical profile of patients with Spontaneous Intracerebral Haemorrhage.

2. To identify factors which correlate with outcome of patients with Intracerebral Haemorrhage including the adverse influence of alcohol and especially alcoholic binge within 48 hours of ictus on the mortality of patients with Intra Cerebral Haemorrhage.

3. To assess the utility of Intra Cerebral Haemorrhage Score (ICHS) and National Institute of Health Stroke Scale (NIHSS) in evaluating patients with Intra Cerebral Haemorrhage and prognoticating.

4. To assess the significance of calculating the **volume** of Intra Cerebral Haemorrhage at various **locations** and their impact on the outcome and to assess the impact of Intra Ventricular Haemorrhage at various loations of ICH on the outcome.

REVIEW OF LITERATURE

Intracerebral haemorrhage accounts for 10 to 15% of first time strokes world over[1]. Incidences are predicted to be higher in Asian population[2]. The 30 day mortality ranges from 32% to 55 %[2]. Of the total patients with ICH only 20 % of patients are expected to be functionally independent at the end of 6 months[2].

Primary (Hypertensive) Intracerebral Hemorrhage

This is the mundane but often devastating "spontaneous" brain hemorrhage. It is predominantly a result of chronic hypertension and degenerative changes in cerebral arteries. In order of frequency, the most common sites of a cerebral hemorrhage are (1) the putamen and adjacent internal capsule (50 percent); (2) the central white matter of the temporal, parietal, or frontal lobes (lobar hemorrhages, not strictly associated with hypertension); (3) the thalamus; (4) one or the other cerebellar hemisphere; and (5) the pons. The vessel involved is usually a small penetrating artery that originates from a larger trunk vessel. Approximately 2 percent of primary hemorrhages are multiple. Multiple nearly simultaneous intracerebral hemorrhages raise the possibility of amyloid angiopathy or a bleeding diathesis but may occur when one conventional hypertensive intracerebral hemorrhage causes hypertension, which in turn leads to one or more

additional hemorrhages.[3]

The extravasation of blood into the substance of the brain forms a roughly circular or oval mass that disrupts the tissue and can grow in volume if the bleeding continues .Adjacent brain tissue is distorted and compressed. If the hemorrhage is large, midline structures are displaced to the opposite side of the cranium and the reticular activating and respiratory centers are compromised, leading to coma and death. It has been long known that both the size and the location of the clot determine the degree of secondary brainstem compression and this was confirmed by Andrew and associates. Rupture or seepage of blood into the ventricular system or rarely to the surface subarachnoid space may occur, and the CSF becomes bloody in these cases. When the hemorrhage is small and located at a distance from the ventricles, the CSF may remain clear even on repeated examinations. In the first hours and days following the hemorrhage, varying degrees of edema accumulates around the clot and adds to the mass effect. Hydrocephalus may occur as a result of bleeding into the ventricular system or from compression of the third ventricle. The extravasated blood undergoes a predictable series of changes. At first fluid, the collection becomes a clot within hours. Before the clot forms, red cells settle in the dependent part of the hematoma and forms a meniscus with the plasma above; this is particularly prone to occur in cases of anticoagulant-induced hemorrhage. The resultant fluid level can be observed on CT scan and MRI ("hematocrit effect"). Hematomas, when examined in autopsy material, contain

only masses of red blood cells and proteins; rarely one sees a few remnants of destroyed brain tissue. The hematoma is often surrounded by petechial hemorrhages from torn arterioles and venules.

Within a few days, hemoglobin products, mainly hemosiderin and hematoidin, begin to appear. The hemosiderin forms within histiocytes that have phagocytized red blood cells (RBCs) and takes the form of ferritin granules that stain positively for iron. As oxyhemoglobin is liberated from the RBCs and becomes deoxygenated, methemoglobin appears. This begins within a few days and imparts a brownish hue to the periphery of the clot. Phagocytosis of red cells begins within 24 h, and hemosiderin is first observed around the margins of the clot in 5 to 6 days. The clot changes color gradually over a few weeks from dark red to pale red, and the border of golden-brown hemosiderin widens. The edema disappears over many days or weeks. In 2 to 3 months, larger clots are filled with a chrome-colored thick fluid, which is slowly absorbed, leaving a smooth-walled cavity or a yellow-brown scar. The iron pigment (hematin) becomes dispersed and studs adjacent astrocytes and neurons and may persist well beyond the border of the hemorrhage for years.[3]

Imaging techniques demonstrate a predictable sequence of changes. In CT scans, fresh blood is visualized as a white mass as soon as it is shed. The mass effect and the surrounding extruded serum and edema are hypodense. After 2

to 3 weeks, the surrounding edema begins to recede and the density of the hematoma decreases, first at the periphery. Gradually the clot becomes isodense with brain. There may be a ring of enhancement from the hemosiderin-filled macrophages and the reacting cells that form a capsule for the hemorrhage.

At one point several weeks after the bleed, the appearance may transiently simulate a tumor or abscess. By MRI, either in conventional T1- or T2-weighted images, the hemorrhage is not easily visible in the 2 or 3 days after bleeding, as oxyhemoglobin is diamagnetic or, at most, is slightly hypointense, so that only the mass effect is evident. MR gradient-echo images that display areas of magnetic susceptibility will show hemorrhages earlier and detect remnants of deposited hemosiderin even years afterwards.[3]

After several days the surrounding edema is hyperintense in T2-weighted images. As deoxyhemoglobin and methemoglobin form, the hematoma signal becomes bright on T1-weighted images and dark on T2. As the hematoma becomes subacute, the dark images gradually brighten. When methemoglobin disappears and only hemosiderin remains, the entire remaining mass is hypodense on T2-weighted images, as are the surrounding deposits of iron. The sizes of cerebral hemorrhages vary widely. *Massive* refers to hemorrhages several centimeters in diameter; *small* applies to those 1 to 2 cm in diameter and less than 20 mL in volume. The volume and location relate to outcome

and the nature of the initial neurologic deficit[3].

Risk Factors

There has been a rapid and remarkable 60% decline in death rates from stroke in the United States, and in most of the industrialized countries since 1972[5].

In addition, there is a striking decline in the incidence of intra-cerebral haemorrhage in Sweden[8]. This declining trend in case fatality rate of stroke in most industrialized countries may be explained by 3 mechanisms.

1.Improved acute stroke care which has prolonged survival.

2.Stroke cases in these places are less severe3. Stroke cases that were mild in severity and previously undetected are being detected now[9].

This data serves to highlight the operation of environmental influences that are amenable to modification, and the intensity of effort and optimal utilization of public health resources to implement preventive measures might be the way for developing countries like India to deal with stroke. This journey begins with assessing the changing

risk factors which might predispose our population to the dise **Untreated or under-treated hypertension** is the single most important risk factor for spontaneous intracerebral haemorrhage (ICH)[3].

Spontaneous intracerebral haemorrhage is considered to be predominantly a direct effect of chronic hypertension and the degenerative changes in the cerebral arteries[3] Advanced **age** and heavy **alcohol** consumption are also known to be risk factors for ICH[2].

Pathogenesis

Sustained hypertension induces smooth muscle cell proliferation in the arterioles. This process is termed hyper plastic arteriolo sclerosis[42]. Over time, smooth muscle cells die and the tunica media is replaced by collagen, resulting in vessels with decreased tone and poor compliance. The arterioles ultimately undergo ectasia and aneurysmal dilation[42]. These micro aneurysms, called Charcot-Bouchard aneurysms, are susceptible to rupture leading to cerebral hemorrhage and were proposed by Charcot and Bouchard in 1868 as a key element of deep ICH[43,44.45]. These aneurysms, occur in vessels that are less than 300 micro meter in diameter, most commonly in the basal ganglia, thereby making this the most common location of bleed[4]. However in few of the haemorrhages examined at autopsy with serial sections by C M fisher, the bleeding

could not be traced to the aforementioned aneurysms[3].

Takebayaschi and co workers, in an electron microscopy study, found breaks in the elastic lamina at multiple sites, almost always at the bifurcation of the small vessels. Possibly these represent sites of secondary rupture from tearing of small vessels by the expanding hematoma.[3] The extravasated blood forms an oval mass that disrupts and compresses the adjacent tissue as it grows in volume.

If the haemorrhage is large, midline structures are displaced to the opposite side and the reticular activating and respiratory centers can be compromised, leading to coma and death.

In the first hours and days following the haemorrhage, a limited amount of oedema accumulates around the clot and adds to the mass effect. Hydrocephalus may occur as a result of bleeding into the ventricular system or from compression of the third ventricle[3].

Clinical features

Of all the cerebrovascular diseases, brain hemorrhage is the most dramatic and from ancient times has been given its own name, "apoplexy." The

prototype is an obese, plethoric, hypertensive male who falls senseless to the ground—impervious to shouts, shaking, and pinching—breathes stertorously, and dies in a few hours. A massive blood clot escapes from the brain as it is removed postmortem. With smaller hemorrhages, the clinical picture conforms more closely to the usual temporal profile of a stroke, i.e., an abrupt onset of symptoms that evolve gradually and steadily over minutes or hours, depending on the size of the ruptured artery and the speed and expansion of bleeding[3].

Perihematomal edema volume increases by approximately 75% during the first 24 hours after spontaneous ICH and has been implicated in the delayed mass effect that occurs in the second and third weeks after ICH[55,56].

The clinical presentation of ICH had two main elements : symptoms that reflect the effects of intracranial hypertension and those that are specific for the location of the haematoma.The general clinical manifestations related to ICP are headache, vomiting, depressed level of consciousness, which vary in their frequency.[93]

A few clinical features which occur with a greater frequency in patients with intra-cerebral haemorrhage need special mention·

Acute reactive hypertension, far exceeding the patient's chronic hypertensive level, is

a feature that should always suggest haemorrhage; it is seen with moderate and large clots situated in deep regions. **Vomiting** at the onset of intra-cerebral haemorrhage occurs much more frequently than with infarction and should always suggest haemorrhage as the cause of an acute hemiparesis.

Severe headache is generally considered to be an accompaniment of intra-cerebral haemorrhage, but in almost 50 percent of cases headache has been absent or mild in degree **Loss of consciousness** is more often a feature of haemorrhage than infarct, though a significant number of haemorrhagic patients are alert at presentation. **Seizures**, usually focal, occur in the first few days in some 10 percent of cases of supratentorial haemorrhage, but more commonly as a delayed event, months or even years after the event.

Most of the haemorrhages develop over 30 – 90 minutes.[2] Thereby the patient might develop headache or vomiting, followed by sagging of the face in the next few minutes, followed by slurring of speech and then notice the arms and legs gradually weakening[3]. In the majority of cases, the haemorrhage has its onset while the patient is up and active; onset during sleep is a rarity. However, haemorrhages may occur when the patient is calm and unstressed (Caplan, 1993).

Headache, acute hypertension, and vomiting with a focal neurologic deficit are therefore the cardinal features and serve most dependably to distinguish haemorrhage from ischemic stroke. Having said that, it must be noted that these prodromal symptoms do not occur with any consistency[3].

Types of intracerebral haemorrhage:

Putaminal haemorrhage

This is the most common site of hypertensive bleed, and the adjacent internal capsule is usually damaged. Contralateral hemiparesis is therefore the sentinel sign. When haemorrhage is large, drowsiness gives way to coma, a dilated pupil and fixed ipsilateral pupil, and decerebrate rigidity.

Thalamic haemorrhages

They produce a contralateral hemiparesis due to extension into the adjacent internal capsule. A prominent sensory deficit involving all the sensory modalities is usually present. Thalamic haemorrhage, by virtue of its extension into the

subthalamus and high midbrain, may cause a series of ocular disturbances.

Pontine haemorrhages

Here deep coma usually ensues in a few minutes, and the clinical picture is dominated by total paralysis, decerebrate rigidity, and small (1-mm) pupils that react to light. Lateral eye movements, evoked by head turning or caloric testing, are impaired or absent. Death usually occurs within a few hours, but there are rare exceptions in which consciousness is retained and the clinical manifestations indicate a smaller lesion in the tegmentum of the pons.

Cerebellar Haemorrhage

Repeated vomiting is a prominent feature, along with occipital headache, vertigo, and inability to sit, stand, or walk. In the early phase of the illness, other clinical signs of cerebellar disease may be minimal or lacking; only a minority of cases show nystagmus or cerebellar ataxia of the limbs, although these signs must always be sought. Dysarthria and dysphagia may be prominent in some cases but usually absent. In the series collected by St. Louis and colleagues, patients with vermian clots and hydrocephalus were at the highest risk for rapid deterioration. As the hours pass, and occasionally with unanticipated suddenness, the patient becomes stuporous and then comatose or suddenly apneic as a result of brainstem compression, at which point

reversal of the syndrome, even by surgical therapy, is seldom successful.[3]

Caudate haemorrhage

The most common clinical presentations are severe headache, nausea, vomiting, and signs of marked meningeal irritation, mimicking a SAH or primary IVH.These patients show no prominent motor, sensory, or visual abnormalities (Pedrazzi et al 1990).The parenchymal haemaatoma itself does not require surgical intervention but emergency extraventricular drainage may be required when acute hydrocephalus develops.However, a permanent VPShunt is necessary only rarely.[76]

Lobar Hemorrhage

Bleeding in areas other than those listed above, specifically in the subcortical white matter of one of the lobes of the cerebral hemisphere, is not associated strictly with hypertension. Any number of other causes are usually responsible, the main ones being anticoagulation or thrombolytic therapy, acquired coagulopathies, cranial trauma, arteriovenous malformation , trauma, and, in the elderly, amyloidosis of the cerebral vessels.In one series of 26 patients with lobar ICH, 14 had

normal blood pressure, and in several of the fatal cases there was amyloidosis of the affected vessels; 2 patients were receiving anticoagulants, 2 had an arteriovenous malformation, and 1 had a metastatic tumor. Similarly, in the series of 22 patients with lobar clots reported by Kase and colleagues, 55 percent were normotensive; metastatic tumors, arteriovenous malformations, and blood dyscrasias were found in 14, 9, and 5 percent of the patients, respectively. Amyloid angiopathy is an important cause of lobar hemorrhage in the elderly patients

Course and Prognosis

The immediate prognosis for large and medium-sized cerebral clots is grave; some 30 to 35 percent of patients die in 1 to 30 days. In these cases, either the hemorrhage has extended into the ventricular system or intracranial pressure becomes elevated to levels that preclude normal perfusion of the brain. Or the hemorrhage seeps into vital centers such as the hypothalamus or midbrain.[3]

A formula that predicts outcome of hemorrhage based on clot size was devised by Broderick and coworkers; it is mainly applicable to putaminal and thalamic clots. A volume of 30 mL or less, calculated by various methods from the CT scan predicted a generally favorable outcome; only 1 of their 71 patients with clots larger than 30 mL had regained independent function by 1 month. By contrast, in

patients with clots of 60 mL or larger and an initial Glasgow Coma Scale score of 8 or less, the mortality was 90 percent .

As remarked earlier, **it is the location of the hematoma, not simply its size that determines the clinical effects**. A clot 60 mL in volume is almost uniformly fatal if situated in the basal ganglia but may allow reasonably good outcome if located in the frontal or occipital lobe. From the studies of Diringer and colleagues (1998), hydrocephalus is also an important predictor of poor outcome.[3]

Predictors of Outcome

Intra-cerebral haemorrhage has been known as the stroke subtype with the highest case fatality rates from time immemorial.[7]

To identify the patients with a potential worse outcome would enable the treating team to anticipate and triage patients for stratifying care and prognosticate for the sake of clinical research and explaining the situation to the family. Numerous parameters have been identified, some with consistent results in predicting outcomes, and others not so.

Age

Age has been reported to be a significant independent outcome predictor in some [13,14] but not the majority of previous studies. Age may appear important for several reasons. Younger patients tend to present to hospital sooner after ictus[13]; although no specific therapy has been demonstrated to have a significant effect on outcome in controlled trials, earlier treatment may reduce mortality.

Second, the elderly, may not receive life-sustaining treatment as aggressive as that given to younger patients. Finally, age may serve as a proxy for other inter-current illnesses that might complicate the clinical situation.

Glasgow Coma Scale

The Glasgow Coma Scale (GCS) measures the best eye, motor and verbal responses, and is a widely used and accepted prognostic score for both traumatic and non-traumatic altered consciousness levels[18]. The score has been validated for its inter-observer reliability.[19]

The assessment of consciousness level in acute stroke is important for clinical management and as an indicator of prognosis. As stroke may cause localized motor,

speech or language deficits, the accuracy of the GCS as a measure of consciousness level may be affected. In turn, its' prognostic value may be impaired.

Weir et al found that dysphasic subgroup showed that the verbal component provided additional prognostic information to the combined eye and motor scores[17]. When a language disorder is absent, the verbal score contributes prognostic information by measuring level of consciousness or by acting as a marker for confusion[17]. Thereby, the GCS score might retain independent outcome predictor value in stroke patients, its utility is increased if combined with the other stroke predictors[17].

The National Institutes of Health Stroke Scale (NIHSS)

The NIHSS is a 15-item neurologic examination stroke scale used to evaluate the effect of acute stroke on the levels of consciousness, language, neglect, visual-field loss, extraocular movement, motor strength, ataxia, dysarthria, and sensory loss and thereby quantitate accurately the neurological deficit. There is an allowance for untestable items [28]. The scale is widely used as a clinical assessment tool to evaluate acuity of stroke patients, determine appropriate treatment, and predict patient outcome[29].

Computed tomography findings

With the advent of CT scanning, estimations of the **volume** of intra-cerebral haemorrhages were possible which lead to numerous studies, which confirmed a positive correlation between increasing volume of bleed and worsening outcome[22,27]. Kothari et al then went to on devise a simple technique to estimate the volume of bleed by using the formula for volume estimation of an ellipsoid[24]. The volume as estimated by the above technique has been validated to be accurate and quick to use[23]. **Expansion of the haematoma** as a complication has been noted to have a graver prognosis[25]

Tuhrim S et al noted that **intra-ventricular extension** of a bleed in a supra-tentorial location was associated with worsening of prognosis[27]. This finding was confirmed by a few other studies as an independent predictor of outcome[15].

Infra-tentorial location of the bleed has been identified as a potential predictor of poor outcome, due to the increased chances of obstructive hydrocephalus and brainstem involvement[17,26].

Outcome predictor scores

Most of these independent variables which predicted outcome in ICH patients were noted to have greater significance of correlation on combination as assessed by multivariate analysis. This led to formulation and validation of a series of scores with varying permutations of the above mentioned variables. One of the earliest and one that has stood the test of time is the **Intra-Cerebral Haemorrhage(ICH) score** as conceived by J Claude Hemophill et al[15].

Based on the strength of independent association of the specified parameters, points were assigned and a stratification scale designed. The total ICH Score ranges from 0 to 5 and the 30 day mortality of each of the scores was found to have a linear correlation with the score[15].

A number of studies following this study have validated the score including the one by Fernandes[30] et al, Cheung et al[32] and Ruiz Sandovol et al[31].

MATERIALS AND METHODS

Setting ***:*** In-patients,

Government General Hospital,

Madras Medical College, Chennai.

Ethical committee : Obtained

Approval

Design of study ***:*** Single center, descriptive study

Period of study ***:*** August 2006 – March 2009.

Sample size ***:*** 231 patients.

Selection of study subjects

Inclusion criteria:

Patients with Spontaneous

Intracerebral Haemorrhage.

Exclusion criteria :

Traumatic ICH

Haemorrhagic infarcts

AVM and aneurysmal ICH

Primary IVH

Methodology :

The study was carried out in the wards of Government General Hospital. Patients with Spontaneous Intracerebral Haemorrhage as confirmed by computed tomography of the brain were selected. A total of 231 patients were included as per the selection criteria. They were enrolled into the study after informed consent was obtained from the patients and from the closest relative in case of patients in altered sensorium.

Patients' demographic, social, economic and medical details were recorded in the proforma sheet. Information regarding the symptoms preceding the present illness were recorded as well.

The initial assessment on admission included assessment of level of consciousness using the **Glasgow Coma Scale (GCS**), blood pressure measurement, neurological deficit assessment using **the National Institute of Health Stroke Scale (NIHSS)** .

The **Intra Cerebral Haemorrhage (ICH) Score** was calculated, and the **Guy Allen score** was calculated 24 hours after admission to differentiate patients into the subtypes of strokes.

Computed Tomography (CT) Scanning of the Brain was studied to record the location of the bleed, presence of intraventricular extension. The volume of the bleed was calculated using the ABC/2 technique.

In the **bedside ABC/2 method**, the CT slice with the largest area of haemorrhage was identified. The largest diameter (A) of the haemorrhage on this slice was measured. The largest diameter 90 degrees to A on the same slice was measured next (B). Finally, the approximate number of 10-mm slices on which the ICH were seen was calculated (C). C was calculated by a comparison of each CT slice with haemorrhage to the CT slice with the largest haemorrhage on that scan. If the haemorrhage area for a particular slice was greater than 75% of the area seen on the slice where the haemorrhage was largest, the slice was considered 1 haemorrhage slice for determining C. If the area was approximately 25% to 75% of the area, the slice was considered half a haemorrhage slice; and if the area was less than 25% of the largest haemorrhage, the slice was not considered a haemorrhage slice. These CT haemorrhage slice values were then added to determine the value for C. All measurements for A and B were made with the use of the centimeter scale on the CT scan to the nearest 0.5 cm. A, B, and C were then multiplied and the product divided by 2, which yielded the volume of haemorrhage in cubic centimeters (ml). Outcome was evaluated on the 30^{th} day of discharge (or earlier in case of death) and functional disability graded using the **Modified Rankin Scale (**mRS). This score assesses functional independence and impact on activities in daily living and grades patients from 0 (no symptoms) to 6 (death).

RESULTS

Total no. of patients -- 231

Sex :

Male -- 162 (70.1%)

Female -- 69 (29.8%)

Age group (years) :

20 – 40 -- 39 (16.8%)

40 – 60 -- 102 (44.1%)

60 – 80 -- 87 (37.6%)

> 80 -- 3 (1.2%)

Arrival time in EMD after ictus :

½ hr	--- 0	(0%)
1—2 hrs	--- 3	(1.2%)
2—3 hrs	--- 21	(9.09 %)
3—6 hrs	--- 57	(24.6%)
6—12hrs	--- 36	(15.5%)
12—24 hrs	--- 30	(12.9%)
24—48 hrs	--- 30	(12.95)
After 2 days	--- 54	(23.3%)

Educational status :

Primary school	-- 39	(16.8%)
Sec.school	-- 63	(27.2%)
U.G	-- 21	(9.09%)
P.G	-- 3	(1.29%)

Professional -- 0 (0%)

Uneducated -- 105 (45.4%)

Religion :

Hindu -- 195 (84.4%)

Muslim -- 18 (7.79%)

Christian -- 18 (7.79%)

Others -- 0 (0%)

H/O DM -- 36 (15.5%)

H/O HT -- 99 (42.8%)

Smoking -- 66 (28.5%)

Alcoholism -- 108 (46.7%)

Alcohol

consumption

within 48hrs -- 75 (32.4%)

symptoms at onset :

Headache	--	102	(44.1%)
Vomiting	--	129	(55.8%)
Headache & Vomiting	--	81	(35.1%)
LOC	--	120	(51.9%)
Seizure	--	30	(12.9%)
Limb weakness	--	186	(80.5%)
Speech disturb	--	132	(57.1%)
Sensory loss	--	27	(11.6%)
Visual disturb	--	30	(12.9%)

Activity when symptoms noticed first :

Sleep	--	18	(7.79%)

On awakening	--	9	(3.89%)
While rest	--	60	(25.9%0
Light activity	--	126	(54.5%)
Heavy exertion	--	18	(7.79%)

Diastolic BP(mm Hg) on admission :

< 80	--	6	(2.59%)
80 – 90	--	42	(18.1%)
91 – 100	--	69	(29.8%)
101 – 110	--	63	(27.2%)
111 – 120	--	27	(11.6%)
121 – 130	--	6	(2.59%)
>130	--	18	(7.79%)

Systolic BP(mm Hg) on admission :

< 120	--	12	(5.19%)
120 – 140	--	30	(12.9%)

141 – 160 -- 39 (16.8%)

161 – 180 -- 54 (23.4%)

181 –200 -- 45 (19.5%)

201 – 220 -- 21 (9.07%)

> 220 -- 30 (12.9%)

NIHSS :

Mild -- </= 6 -- 54 (23%)

Moderate -- 7—10 -- 21 (9.1%)

Moderately

Severe -- 11 – 15 -- 30 (12.9%)

Severe -- 16 – 22 -- 57 (25%)

Very severe -- >/= 23 -- 69 (29.9%)

mRS :

1 -- 15 (6.49%)

2 -- 3 (1.29%)

3 -- 12 (5.19%)

4 -- 48 (20.7%)

5 -- 153 (66.2%)

ICH location :

Putamen	-- 126	(54.5%)
Thalamus	-- 60	(25.9)
Pons	-- 12	(5.19%)
Cerebellum	-- 9	(3.89%)
Lobar	-- 18	(7.79%)
Caudate nucleus	-- 6	(2.59 %)

VOLUME OF ICH ;

Putamen

Total no.of pts :126

< 5ml	--	21(16.6%)
5-10ml	--	18(14.2%)
11-20 ml	--	15(11.9%)
21-30 ml	--	18(14.2%)
31-40 ml	--	6(4.76%)
41-50 ml	--	24(19.04%)
51-60 ml	--	6(4.76%)
61-70 ml	--	9(7.14%)
71-100 ml	--	3(2.38%)
>101 ml	--	6(4.76%)

Thalamus

Total no. of pts :60

<2 ml -- 15(25%)

2-5 ml -- 12(20%)

6-10 ml -- 12(20%)

11-20 ml -- 15(25%)

>20 ml -- 6(10%)

Pons

Total no. of pts :12

<2 ml -- 3(25%)

2-5 ml -- 3(25%)

6-20 ml -- 3(25%)

>20 ml -- 3(25%

Cerebellum

Total no. of pts : 9

<10 ml -- 3(34%)

10-20 ml -- 6(66%)

Lobar hge

Total no. of pts : 18

<10 ml -- 6(33.3%)

10-20 ml -- 6(33.3%)

>20 ml -- 6(33.3%)

Caudate hge

Total no. of pts : 6

<5 ml -- 3(50%)

5-10 ml -- 3(50%)

IVH		**Overall incidence**
Total no. of pts: 102	--	44.1%
IVH in putaminal hge -- 48(38.1%)	--	47.1%
IVH in thalamic hge -- 36(60%)	--	35.2%
IVH in pontine hge -- 3(25%)	--	2.9%
IVH in cerebellar ICH -- 6(66.7%)	--	5.8%
IVH in lobar hge -- 3(16.7%)	--	2.94%
IVH in caudate Hge -- 6(100%)	--	5.8%

Guy's Allen score

<15 -- 39(16.88%)

16.40 -- 75(32.46%)

41.60 -- 93(40.25%)

>60 -- 24(10.38%)

Outcome at 1 month (mRS)

0 -- 9(3.89%)

1 -- 15(6.49%)

2 -- 3(1.29%)

3 -- 42(18.8%)

4 -- 18(7.79%)

5 -- 87(37.6%)

6 -- 57(24.6%)

Alcohol consumption within 48hrs of Ictus and death

No. of of pts who consumed alcohol within

72 hrs preceding the ictus -- 75 (32.5%)

No. of pts who died in the study -- 57 (24.7%)

No. of pts who consumed alcohol within

72 hrs preceding the ictus among those

who died -- 27 (47.4%)

Alcohol consumption within 48hrs of Ictus and good outcome

Total no. of patients with

good outcome(mRS</= 3) -- 69 (30%)

Among the patients with good outcome,

those who consumed alcohol within

72 hrs of the ictus -- 21 (30%)

Among the patients with good outcome,

those who did not consume alcohol within

72 hrs of the ictus -- 48 (70%)

Table 1

Outcome of patients with putaminal ICH with respect to volume

N=126

volume		mRS					
	0	1	2	3	4	5	6
<5 ml	6	9		3	3	6	
5-10 ml				3	6	6	
11-20 ml		3		3		6	
21-30 ml						12	6
31-40 ml						3	3
41-50 ml						18	6
51-60 ml						3	3
61-70 ml						3	6
71-100 ml							3
>101 ml							6

Table 2

Outcome of patients with thalamic ICH with respect to volume

N=60

volume	mRS						
	0	1	2	3	4	5	6
<2 ml		3		9		3	

2-5 ml				9	3		
6-10 ml				3	3	6	
11-20 ml						9	6
>20 ml						6	

Table 3

Outcome of patients with pontine ICH with respect to volume

N=12

volume	mRS						
	0	1	2	3	4	5	6
<2 ml				3			
2-5 ml				3			
6-20 ml							3
>20 ml							3

Table 4

Outcome of patients with cerebellar ICH with respect to volume

N=9

volume	mRS						
	0	1	2	3	4	5	6
<10ml			3				
10 - 20 ml							6

Table 5

Outcome of patients with lobar ICH with respect to volume

N=18

volume	mRS						
	0	1	2	3	4	5	6
<10 ml				3			3
10-20 ml				3	3		
>20 ml						3	3

Table 6

Outcome of patients with caudate ICH volume with respect tovolume

N = 6

volume	mRS						
	0	1	2	3	4	5	6
<5ml	3						
5 – 10ml						3	

Table 7

Outcome of patients with putaminal hge with respect to IVH

N = 126

	mRs						
	0	1	2	3	4	5	6
pts with IVH						24	24
pts without IVH	6	12		9	9	33	9

Table 8

Outcome of patients with thalamic hge with respect to IVH

N = 60

	mRS						
	0	1	2	3	4	5	6
pts with IVH					6	24	6
pts with out IVH		3		18			3

Table 9

Outcome of patients with pontine hge with respect to IVH

N = 12

	mRS						
	0	1	2	3	4	5	6
pts with IVH							3
pts with out IVH				6			3

Table 10

Outcome of patients with cerebellar hge with respect to IVH

N = 9

	mRS						
	0	1	2	3	4	5	6
pts with IVH							6
pts without IVH			3				

Table 11

Outcome of patients with lobar hge with respect to IVH

N = 18

	mRS						
	0	**1**	**2**	**3**	**4**	**5**	**6**
pts with IVH				**3**			
pts without IVH				**3**	**3**	**3**	**6**

Table 12

Outcome of patients with caudate hge with respect to IVH

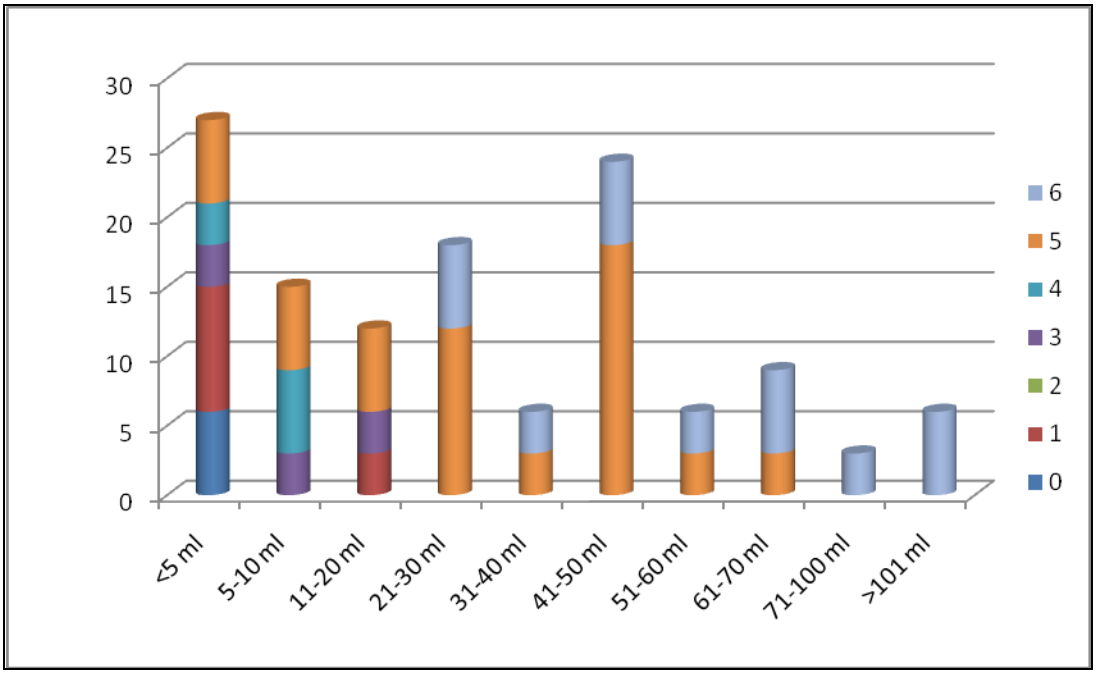
N = 6

	mRS						
	0	1	2	3	4	5	6
pts with IVH	3					3	
pts with out IVH							

TABLE 1

Outcome of pts with putaminal ICH with respect to volume

N=126



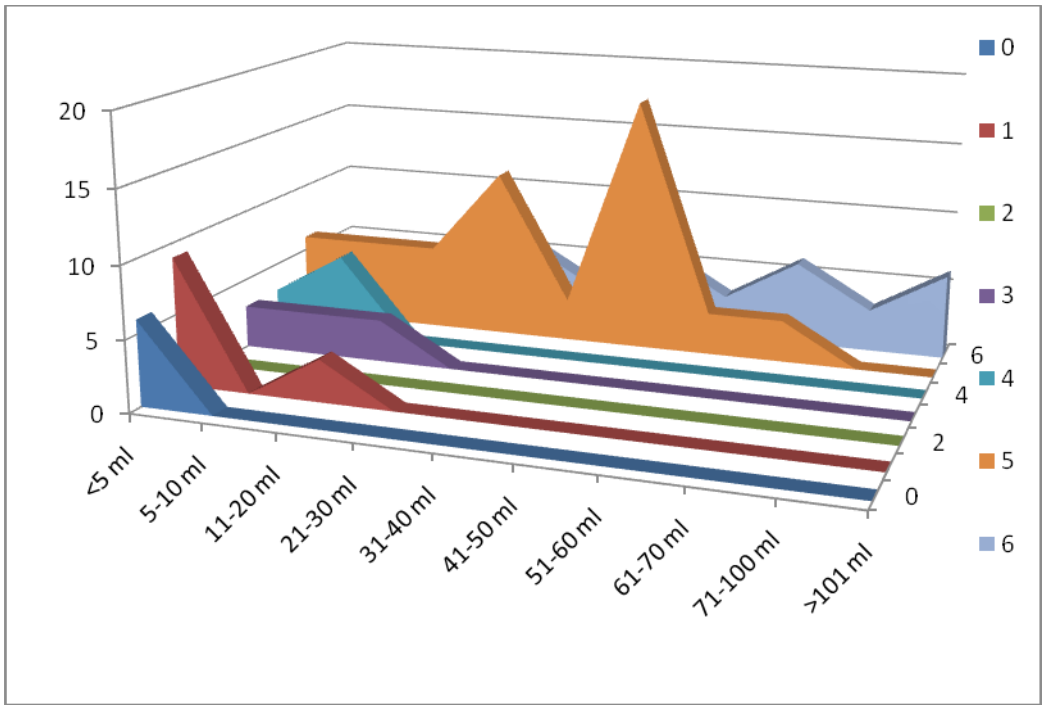
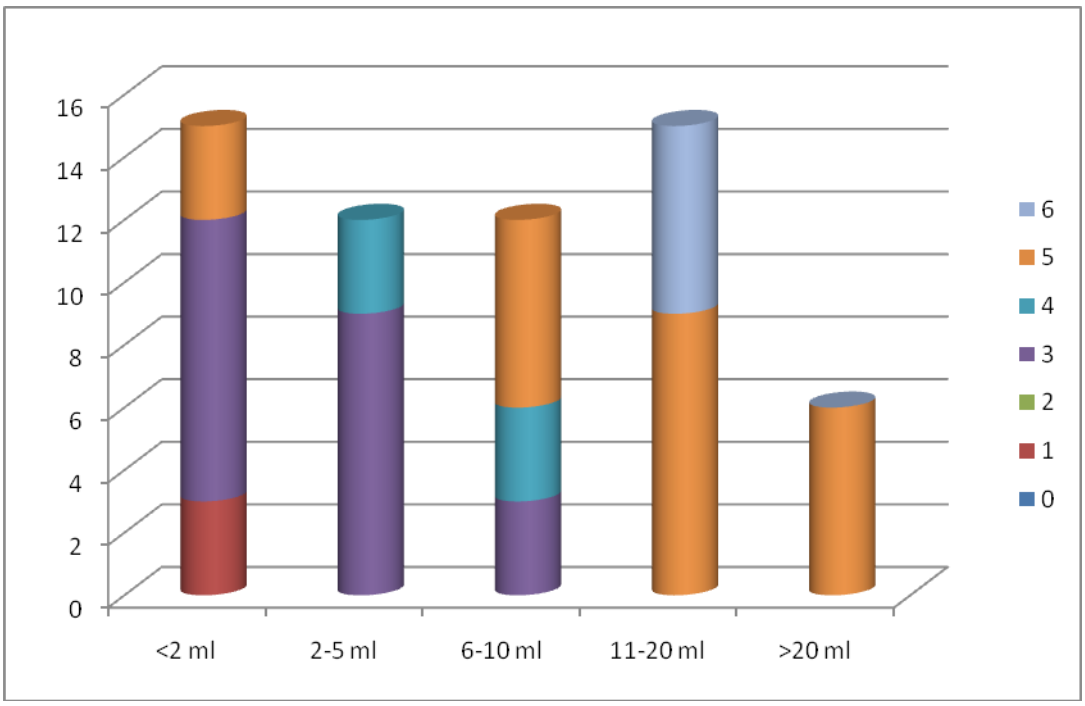


TABLE 2

Outcome of pts with thalamic ICH with respect to volume

N=60



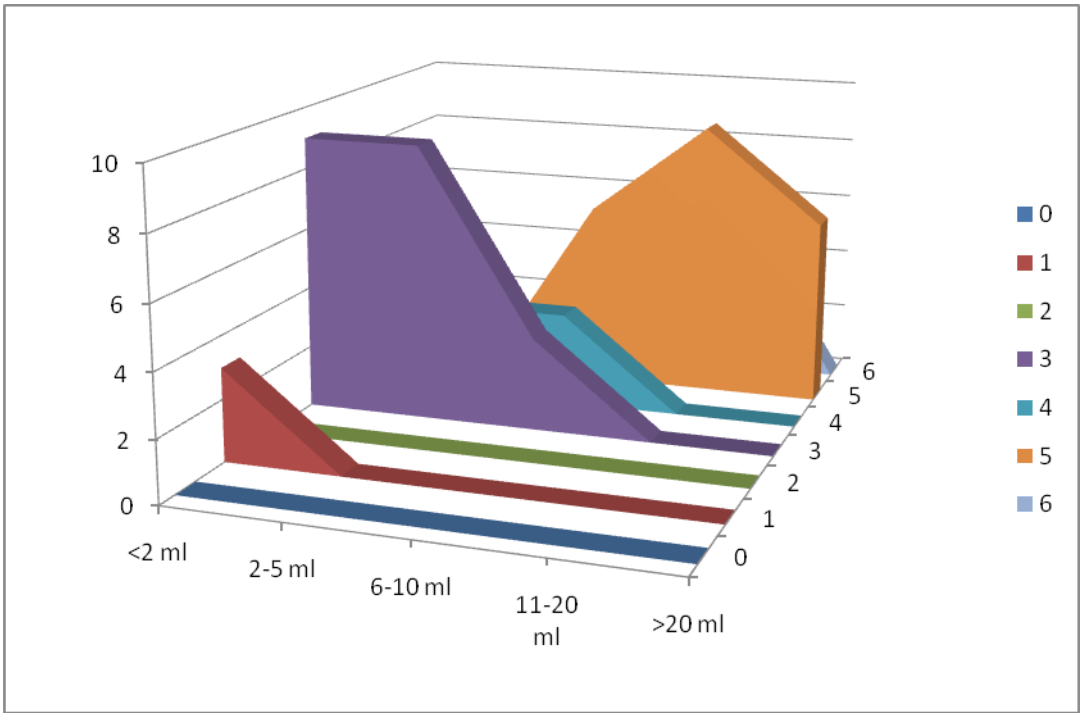
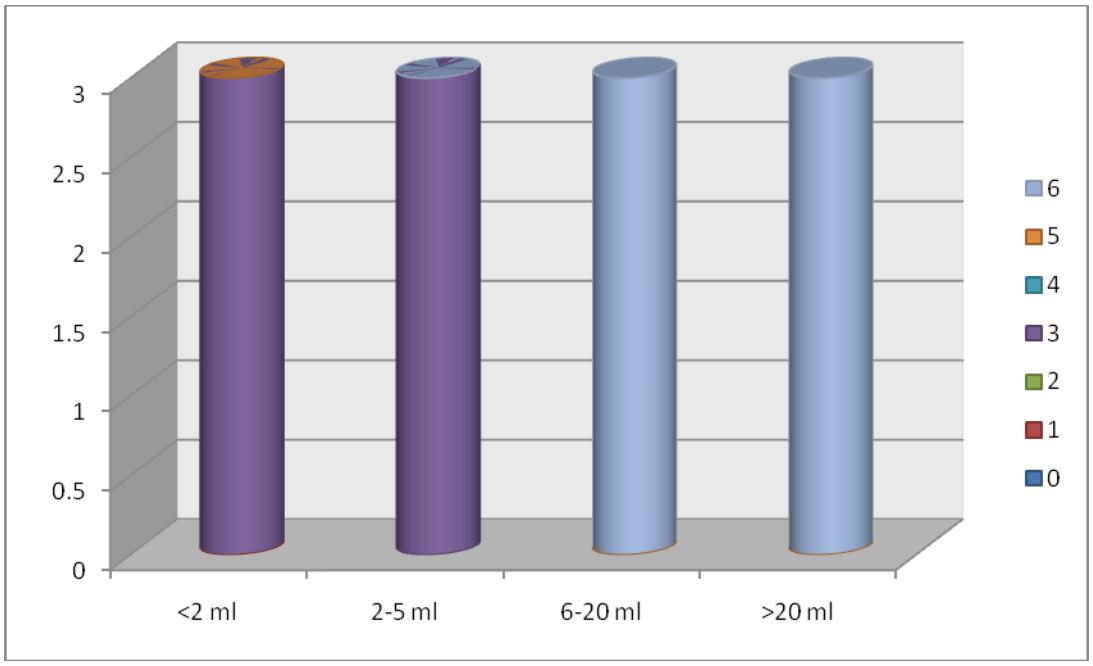


TABLE 3

Outcome of pts with pontine ICH with respect to volume

N=12


3
2.5
2
1.5
1
0.5
0
<2 ml
2-5 ml
6-20 ml
>20 ml
6
5
4
3
2
1
0

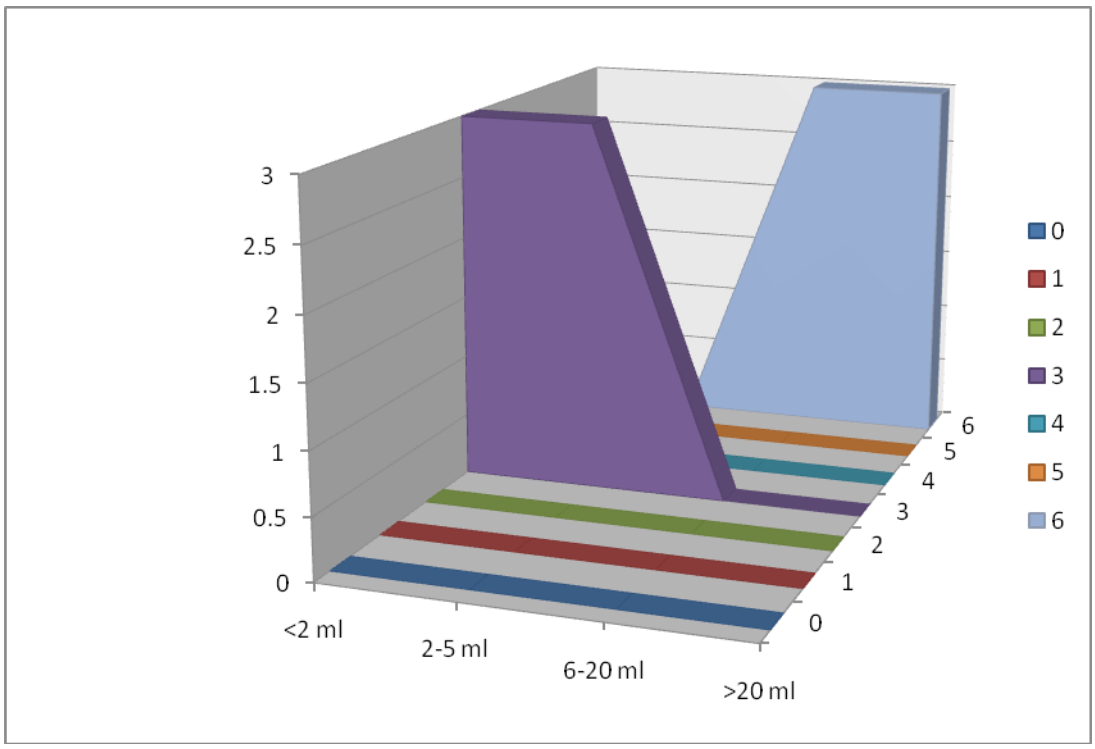
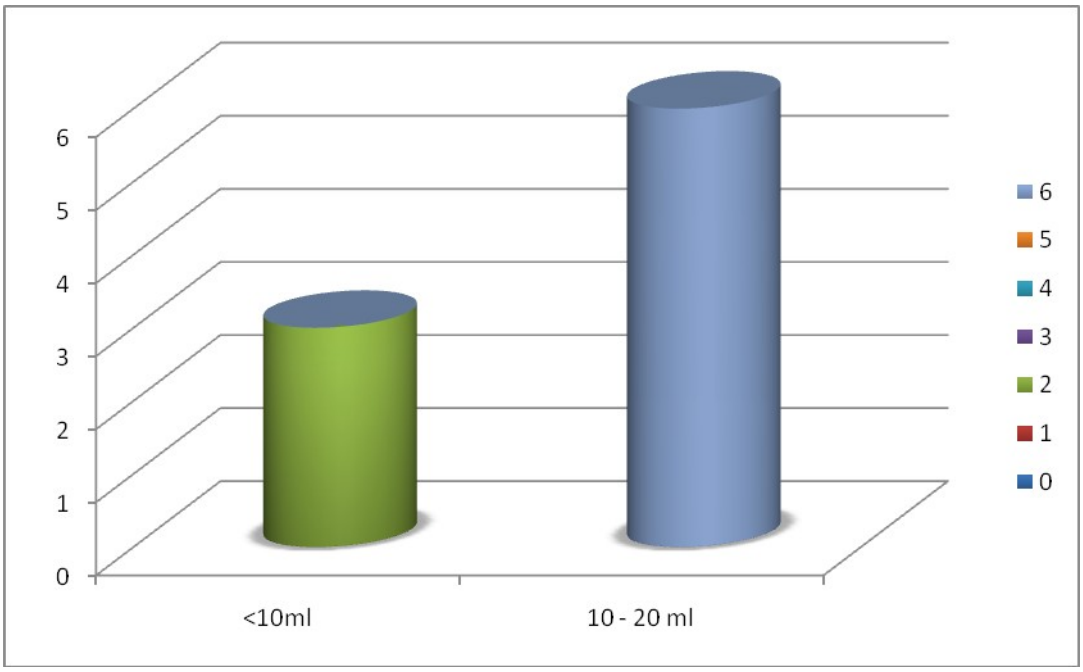

3
2.5
2
1.5
1
0.5
0
<2 ml
2-5 ml
6-20 ml
>20 ml
0
1
2
3
4
5
6

TABLE 4

Outcome of pts with cerebellar ICH with respect to volume

N=9



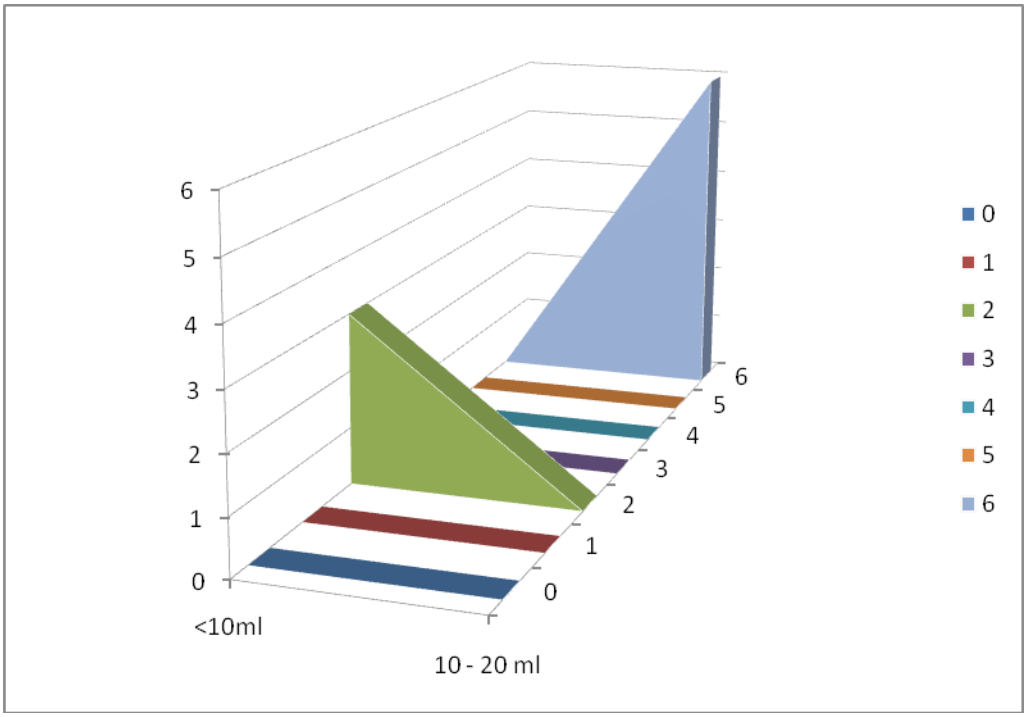
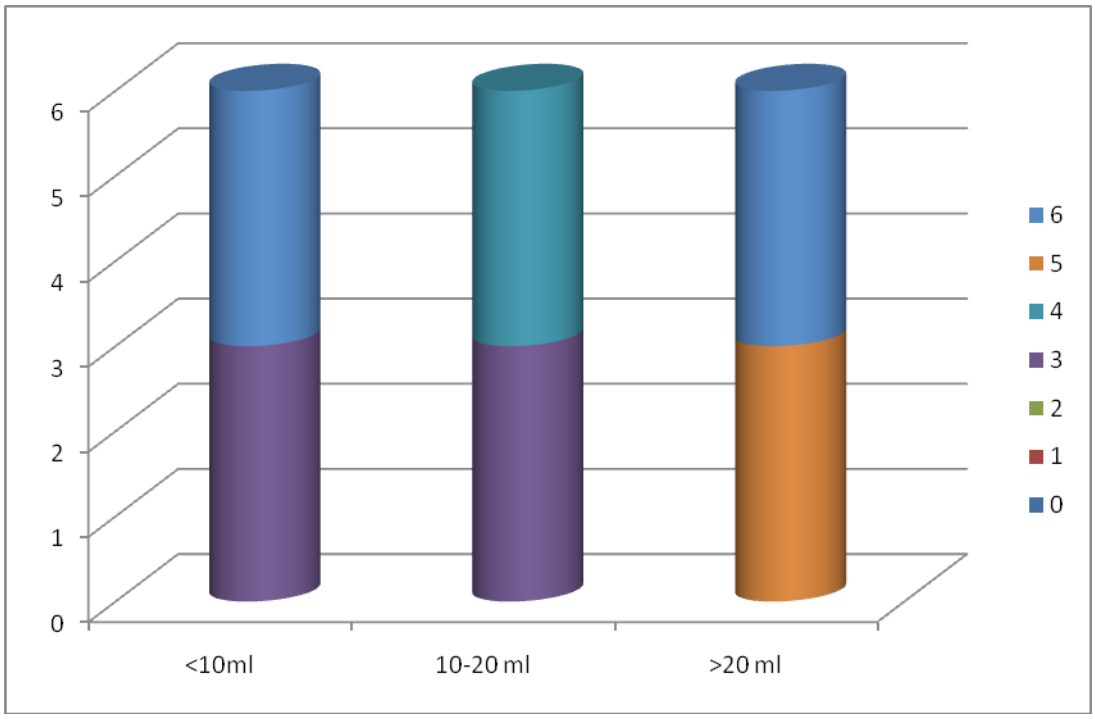


TABLE 5

Outcome of pts with lobar ICH with respect to volume

N=18



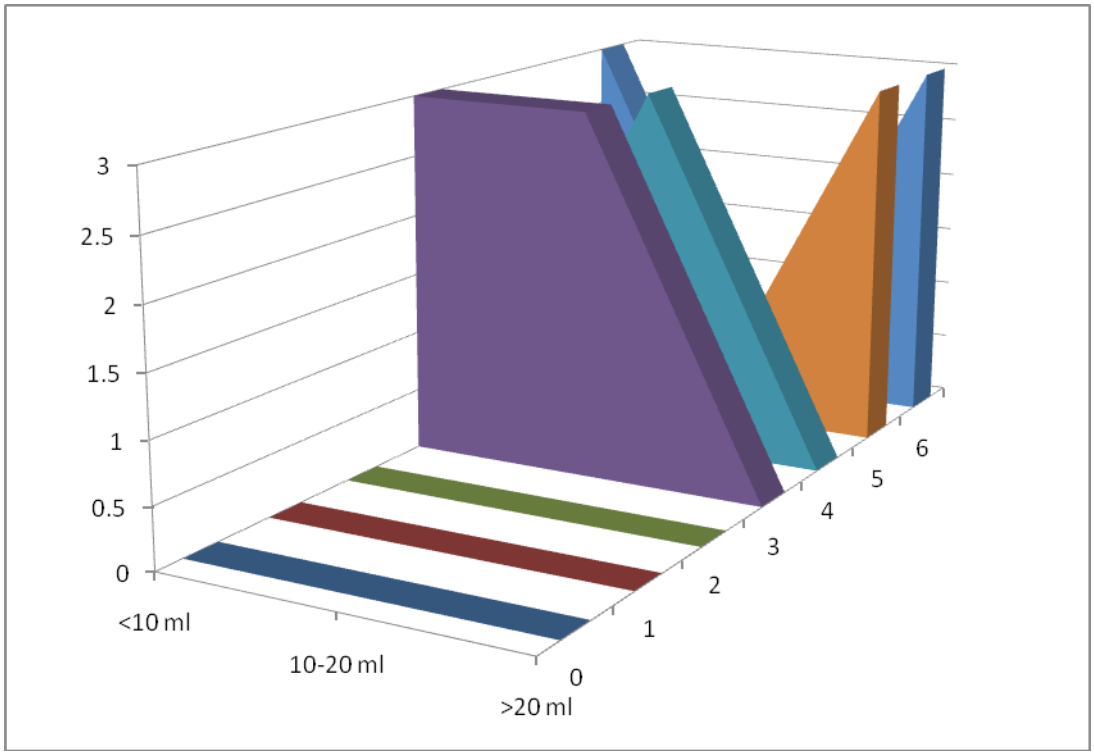
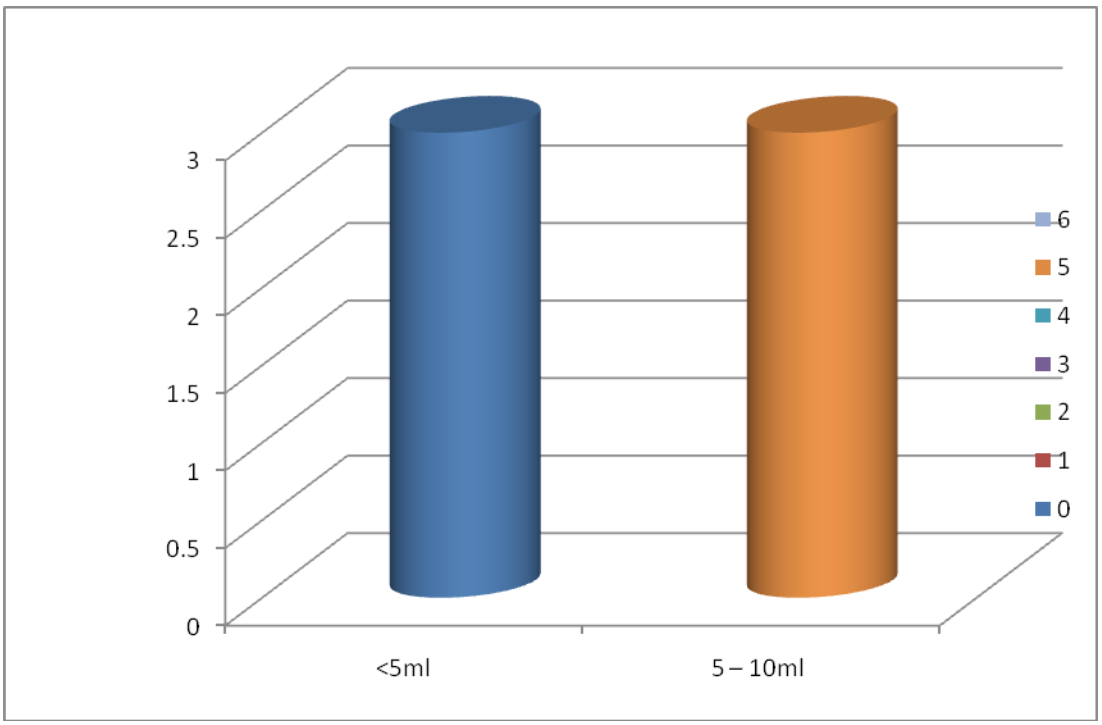


TABLE 6

Outcome of pts with caudate ICH with respect to volume

N=6



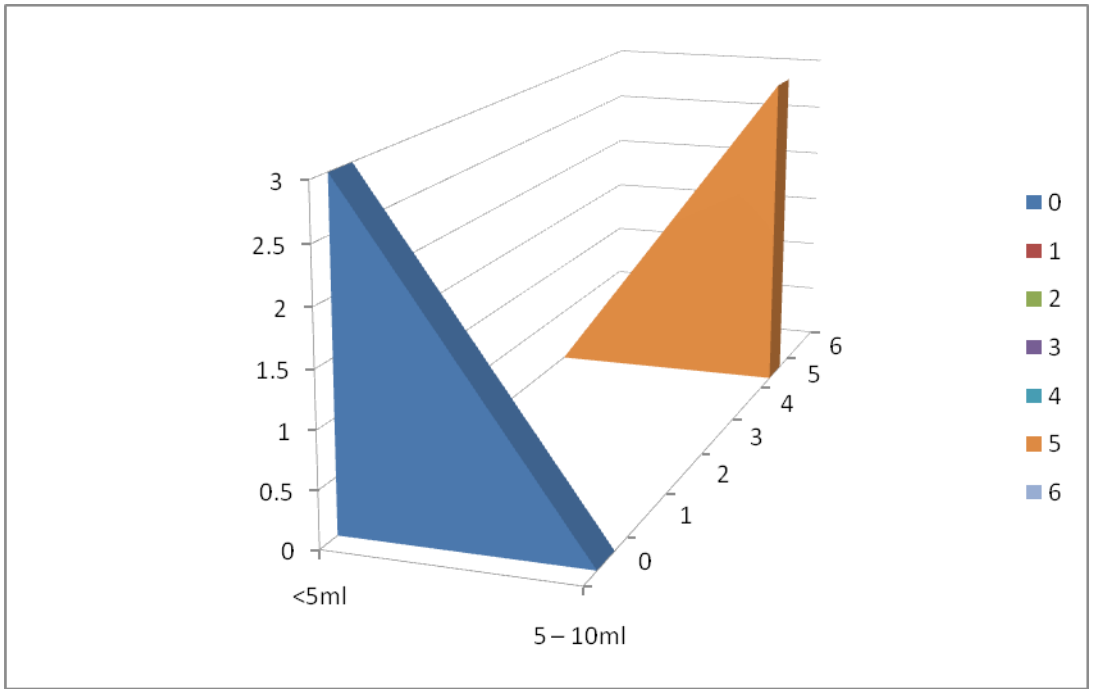
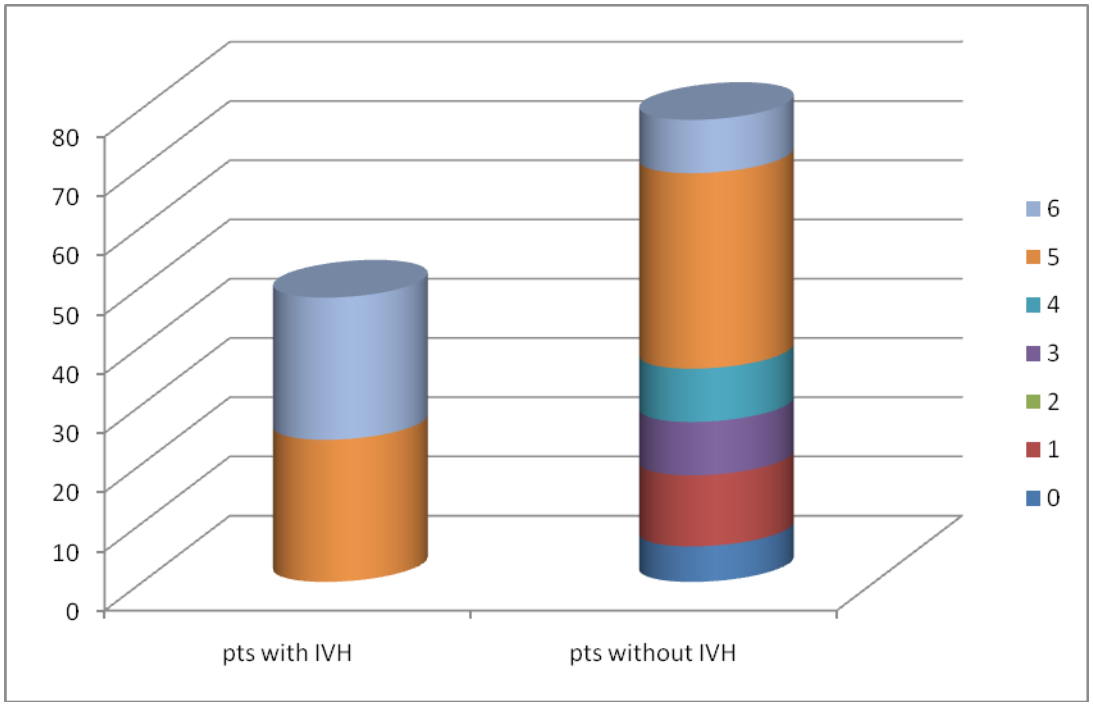


TABLE 7

Outcome of pts with putaminal ICH with respect to IVH

N=126



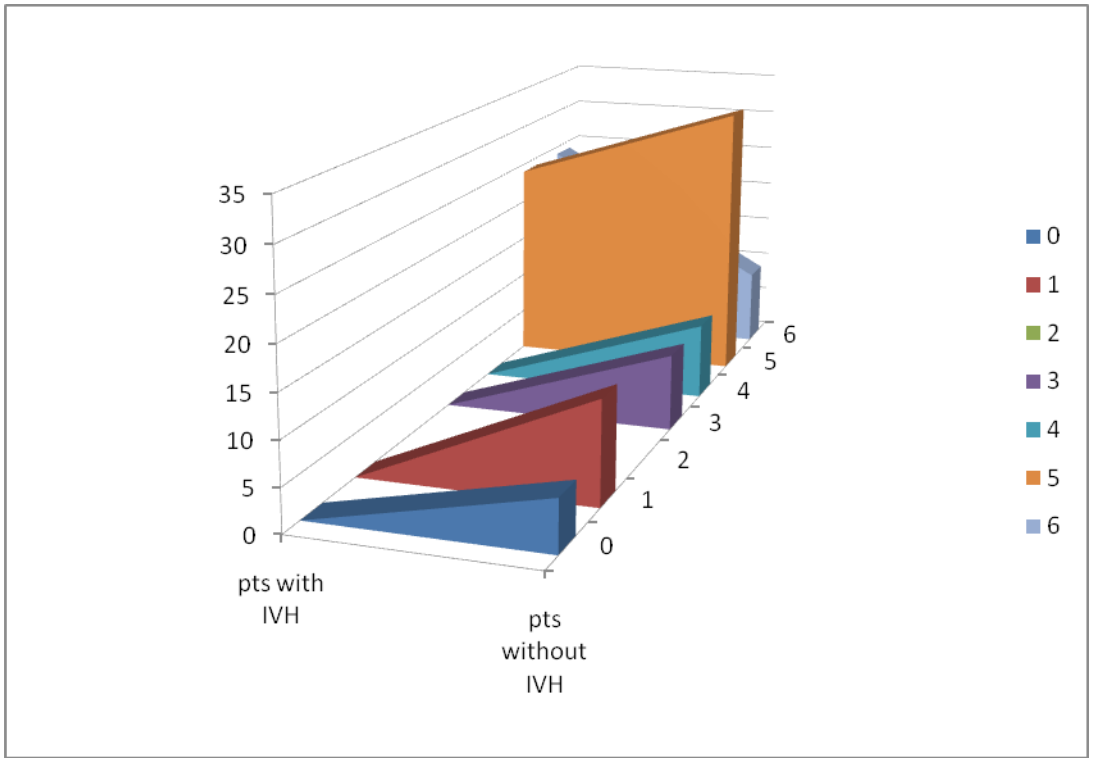
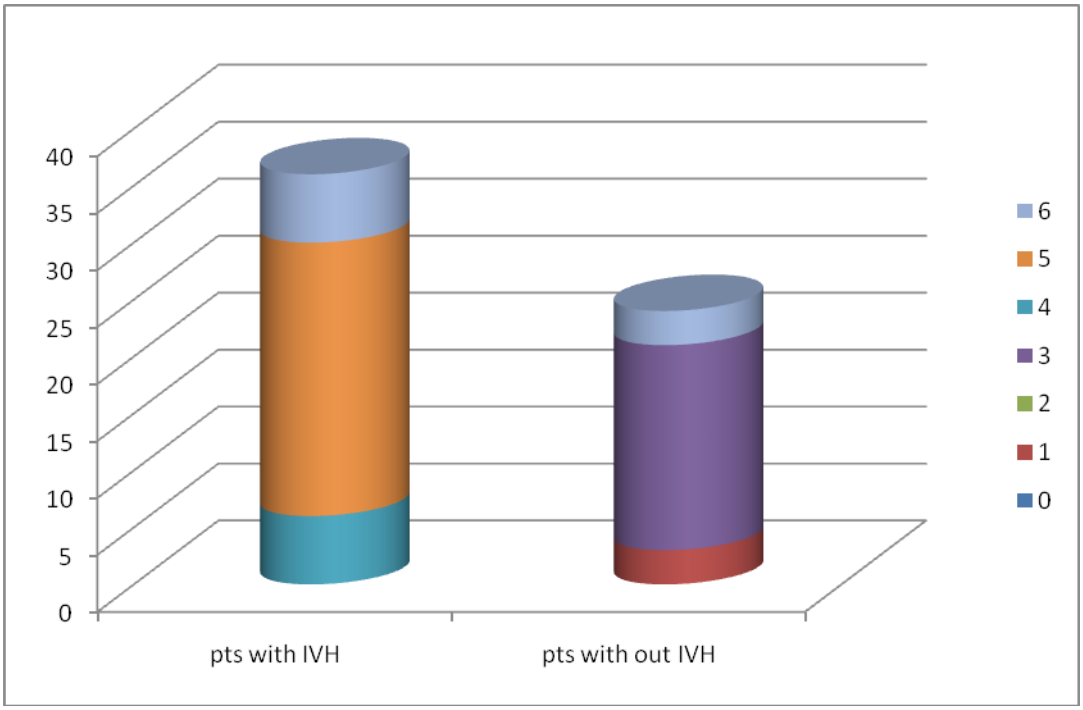


TABLE 8

Outcome of pts with thalamic ICH with respect to IVH

N=60



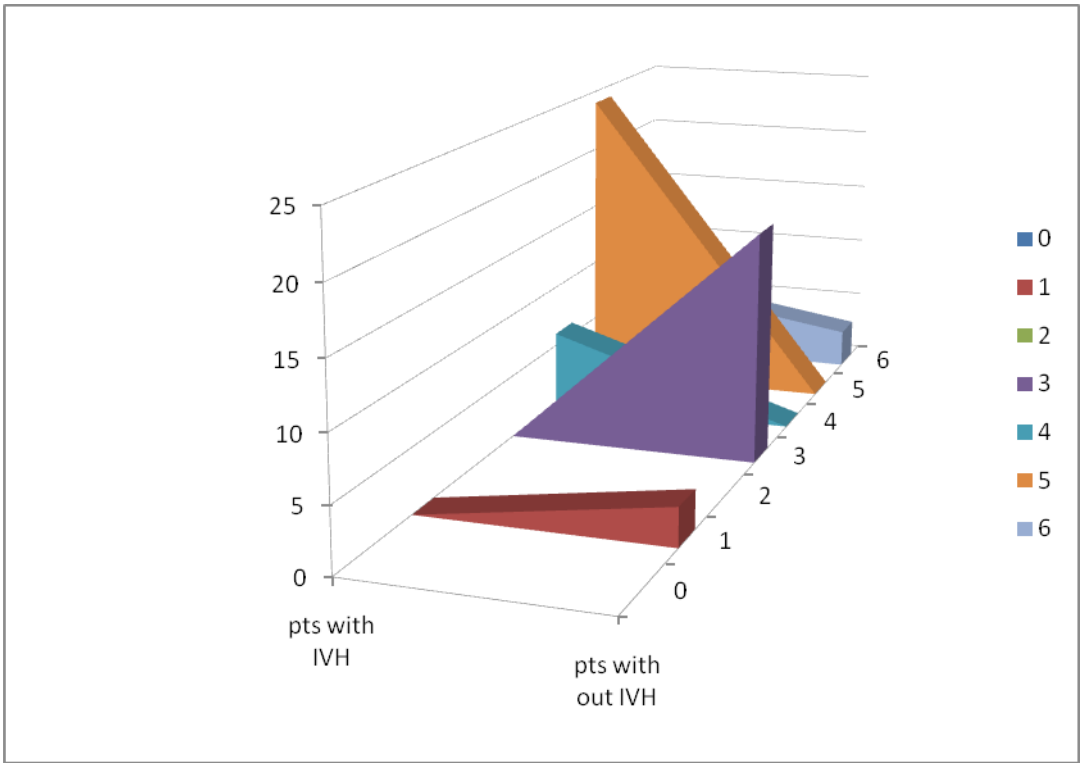
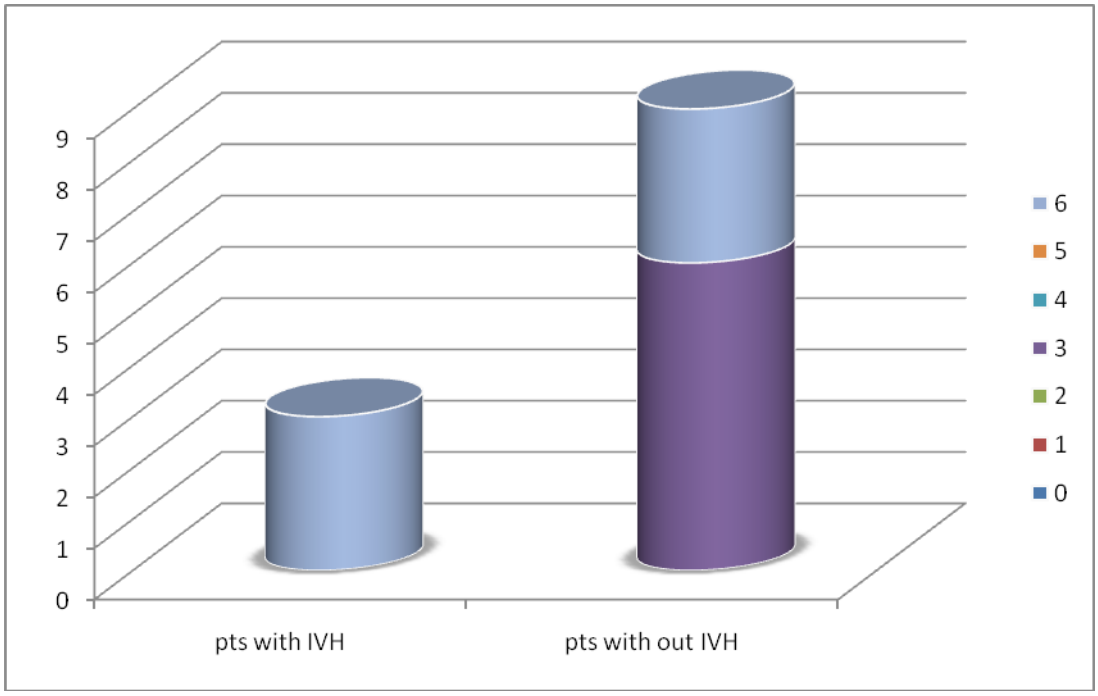


TABLE 9

Outcome of pts with pontine ICH with respect to IVH

N=12



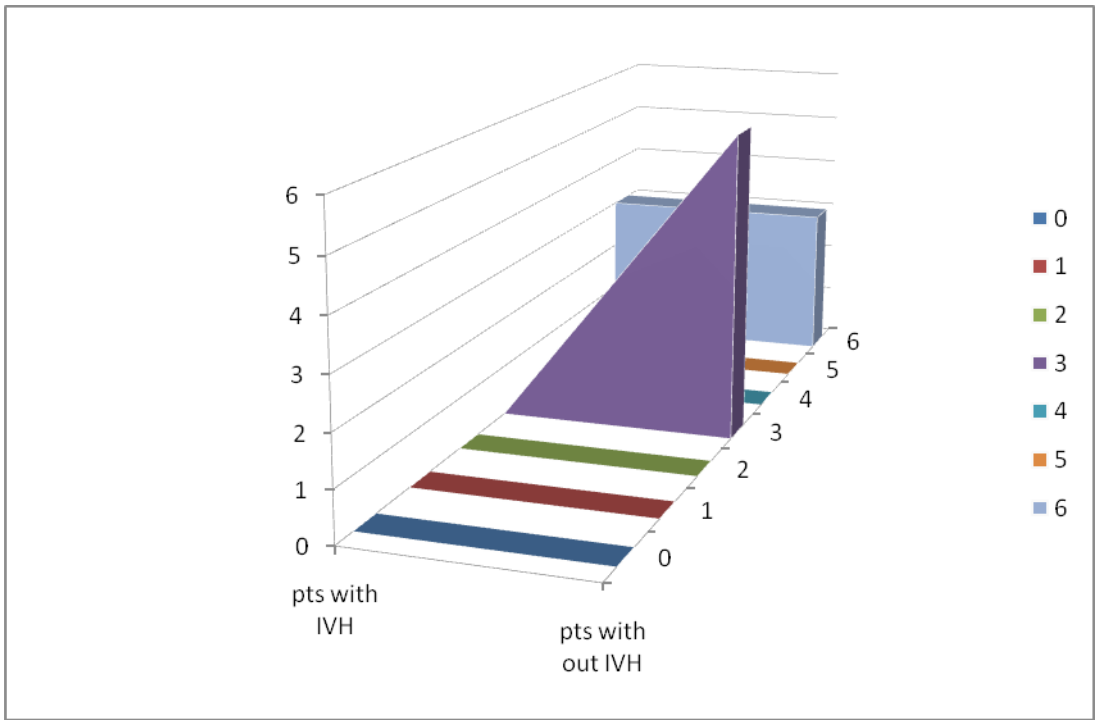
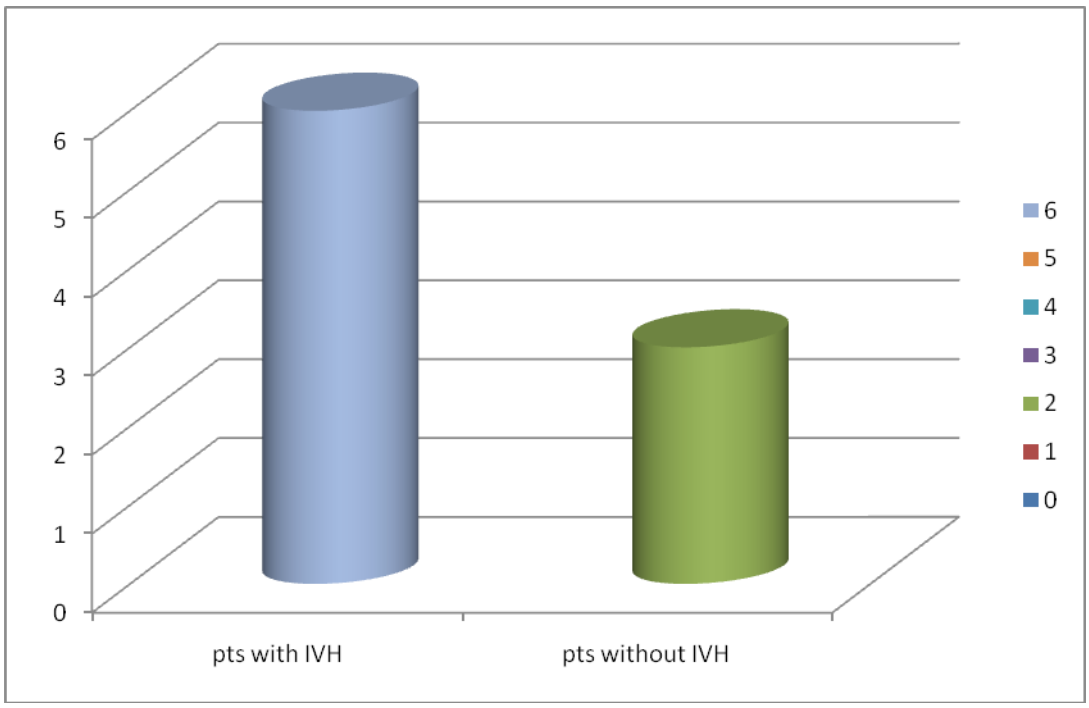


TABLE 10

Outcome of pts with cerebellar ICH with respect to IVH

N=9



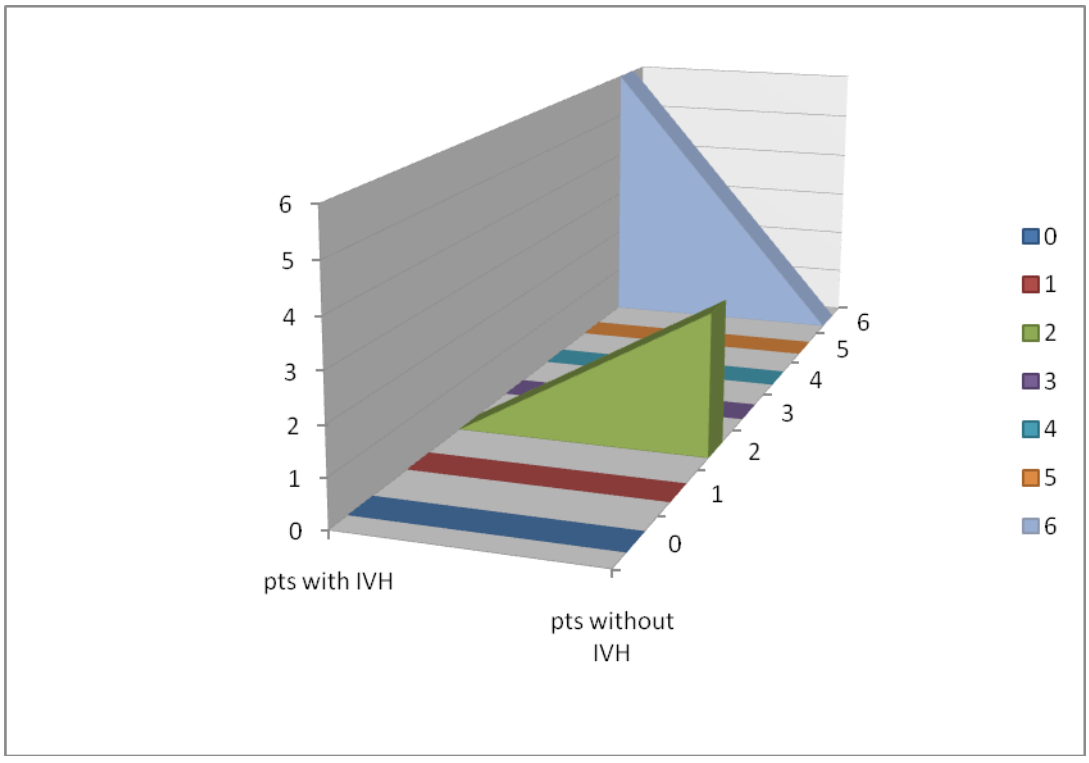
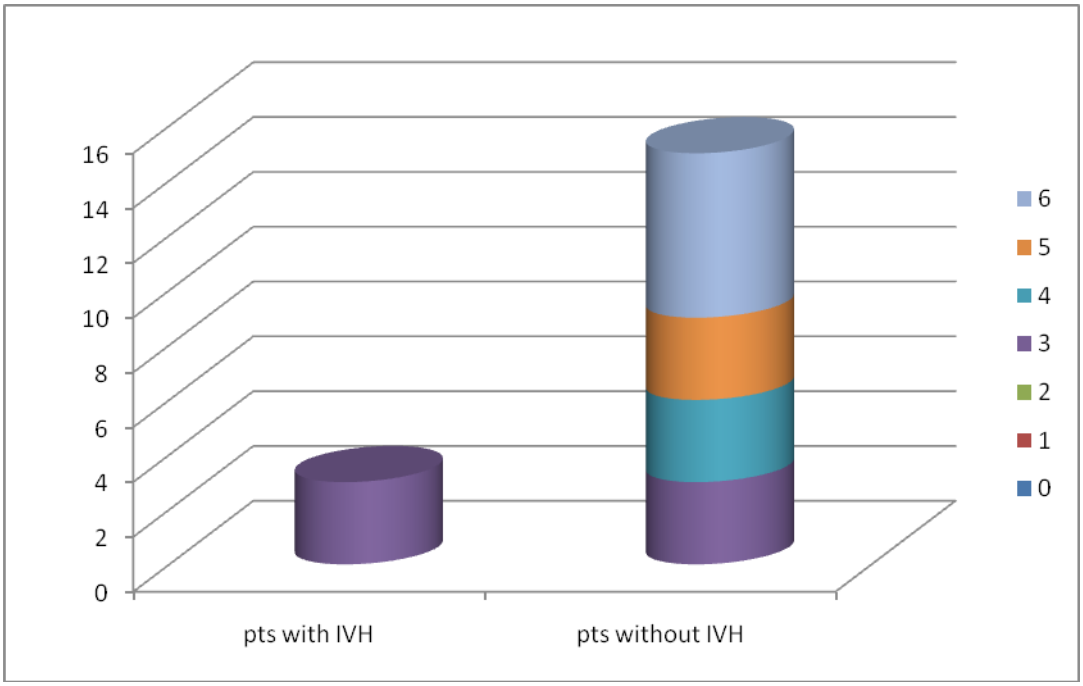


TABLE 11

Outcome of pts with lobar ICH with respect to IVH

N=18



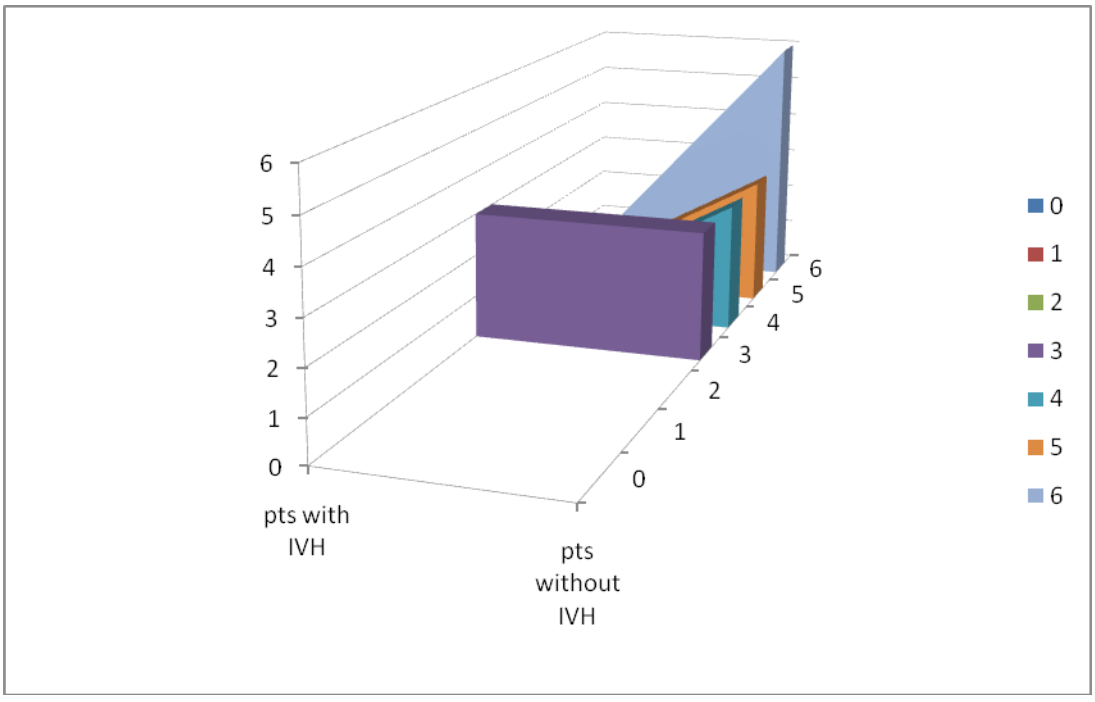
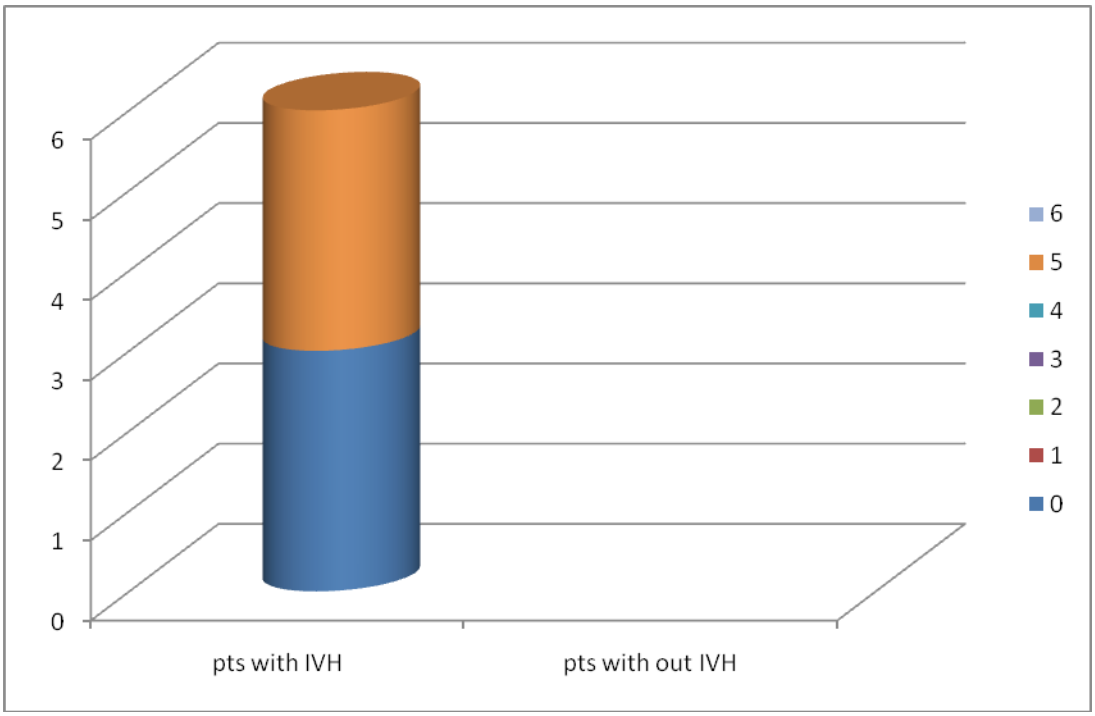
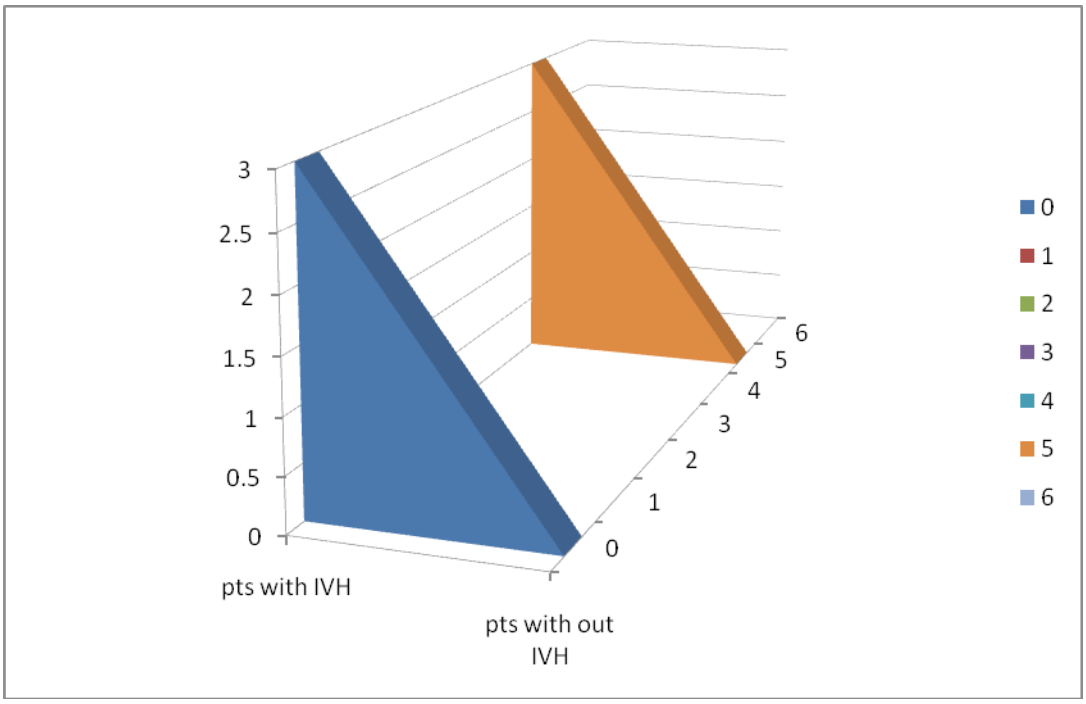


TABLE 12

Outcome of pts with caudate ICH with respect to IVH

N=6


6
5
4
3
2
1
0
pts with IVH
pts with out IVH
6
5
4
3
2
1
0


3
2.5
2
1.5
1
0.5
0
pts with IVH
0
1
2
3
4
5
6
pts with out IVH
0
1
2
3
4
5
6

DISCUSSION

Among the 231 patients enrolled, male patients constituted 162 (70.1%) and female patients 69 (29.8%) making up a male :female ratio of 5:2.Similarly, in the study of brain herniations in ICH,conducted at SGPGIMS,Lucknow,out of 24 patients, 18(75%) were men and only 6(25%) were women,indicating a similar male preponderence.[86] In general spontaneous intracerebral haemorrhage occurs more commonly in men than women(Fewel et al).[78]The influence of gender is unclear, with various investigators reporting a higher incidence in women (Matsumoto et al,AbuZied et al, Robins et al) or in men (Barucha et al, Waga et al, Kunitz et al).[89] If the reason for the significantly higher proportion of male patients with ICH in our study is analysed it can be understood that the most important contributing factor is alcoholism prevalent among males . The overall incidence of alcoholism is 46.7% and among the 162 males, 108 gave h/o alcoholism and of whom 75 had consumed alcohol within 48hrs of the ictus.

From previous studies (Gupta S.P. et al) it is obvious that in India the prevalence of hypertension in males and females respectively is 59.9 and 69.9 per 1000 in urban population and 35.5 and 35.9 per 1000 in rural population.[80] In the developed countries, the normal ratio of hypertension frequency in women versus men increases from 0.6 to 0.7 at age 30 to1.1 to 1.2 at age 65 and the environmental factors implicated in the development of hypertension with increasing age like salt intake, obesity,

occupation, family size, crowding and alcohol intake have importance only in more affluent societies. In our study most of the patients are from less affluent societies.

The study by Kearney et al suggested that men and women have similar overall prevalence of hypertension all over the world.[79] Therefore the prevalence of hypertension among either sex is not the reason for increased incidence of ICH in males but it is the prevalence of alcoholism among males that makes them more susceptible to ICH. This implies that statutory measures have to be taken to curb the habit of consuming alcoholic beverages by Indians.

ICH is rare before the age of 45 years and becomes increasingly more frequent with advancing age.[89] Age greater than 65 years is associated with an odds ratio of 2.8 for recurrence of ICH.Although age cannot be treated, this is useful prognostic information for patients and their health care providers.[74] On analysing the age group of patients it is seen that the incidence after 80 years is only about 1% which probably reflects the lower life expectancy of people in this part of the world. Among the rest of the patients only 16.8% of them are below the age of 40 yrs.These factors are concordant with the previous observation that advancing age and heavy alcohol consumption increase the risk of ICH.[64,65]

In 2005, Mayer and colleagues published the results of a phase 2 B trial using recombinant activated factor VII (rFVIIa) acutely to manage noncongulopathic ICH. This dose – finding study included 399 subjects allocated to 40,80, or 160 mg/kg

or rFVIIa within 4 hrs of symptom onset and within 1hr from the baseline CT scan. The primary outcome was haematoma growth at 24hrs and the reduction in volume was statistically significant only for the highest dosage. There was no effect on haematoma enlargement among those treated more than 3hrs after symptom onset. Mortality and the combined outcome of death or severe disability were significantly reduced in patients treated with rVIIa.[67] The current USFDA label indication for rFVIIa is haemophilia with factor inhibitions and bleeding emergencies. Though rFVIIa does not hold an FDA indication for use in management of ICH at present, the recommendations may likely change in the near future in which case the most important pre requisite will be the time of arrival of the patient to the hospital being less than 3hrs. In our study it can be seen only 10% of patients had arrived within 3hrs to the EMD, a figure which needs to be substantially improved through health education and creation of awareness among the public regarding the early arrival to the hospital. Even at 6hrs, only 35% of the patients had arrived which reflects the various factors associated with failure of patients to arrive earlier like lack of awareness, lack of access to transport facilities at the earliest, delay in local clinics, poverty and so on.

Analysing the educational status of the patient population it can be seen that about 45% of patients are uneducated with only additional 17% having completed primary school and professionals being 0%. This probably reflects the fact that more educated people in the higher socio economic stratum get their risk factors like hypertension well controlled and in case of emergency seek treatment at non

government institutions.

The analysis of religious background of patients shows that about 84% of them belong to Hindu community with 8% each from Muslim and Christian communities which probably is a reflection of the distribution of various religions in this part of the country and not the incidence of ICH in these communities.

The prevalence of hypertension has been found to rise in the recent years globally.Kearney et al., pooled data from different regions of the world to estimate the overall burden of hypertension.The survey showed that the prevalence of hypertension in 2000 was 26% of the adult population globally and that in 2025 would increase by 24% in developed countries and 80% in developing countries.[79] Despite the development of new medications to treat hypertension, adequate treatment continues to be a problem in major part of the globe. The prescribing pattern of drugs used to treat hypertension changes over time in response to changes in recommended guidelines and innovations in drug formulations. In addition, the classes of antihypertensive drugs used vary among the countries. The overall rate of successful blood pressure control is low among hypertensive patients receiving treatment, and despite the inadequacy of certain drugs , many of the patients continue the same treatment regimen.[83]

With repeated BP recordings during admission and followup and with investigations like ECG and Echo it has been confirm ed that all the patients in our study had hypertension. Nonetheless, only 43% of them had their hypertension detected

before the ictus and in them blood pressure control was inadequate in many instances. This clearly indicates that undetected and undertreated hypertension is the most important risk factor for spontaneous ICH. This is consistent in the previous studies which have concluded that Hypertension remains the single most important target for ICH prevention, the odds ratio for ICH is 3.5 for untreated hypertension but only 1.4 for treated hypertension and the treatment of hypertension can prevent ICH (class I).[68] Therefore it becomes imperative to detect hypertension in the asymptomatic population and devise pharmacologic and nonpharmocologic treatment modalities for long term management of hypertension.According to a study performed by Thakkappan and co workers the overall prevalence of hypertension was 36.7% in southern part of India.[81] Hypertension is directly responsible for 57% of all stroke deaths in India.[8o] A declining trend in the incidence of ICH has been reported from Hisayama, Japan where it was also related to a decrease in the frequency of hypertension.[69]

The autopsy study of McCormick and Rosenfield challenged the view that hypertension represents the main causative factor in ICH.[70] Their series included a large number of cases of ICH due to blood dyscrasias, vascular malformation and tumours and hypertension was regarded as the sole basis for bleeding in only 25% of the total. This discrepancy with some reported series of ICH may reflect in them part of referral pattern bias, as well as more stringent criteria in establishing a causal relationship between hypertension and ICH. However, with other popular studies, the role of hypertension as a leading risk factor is well established and its frequency has been

estimated to be between 72% and 81%.[71] The causative role of hypertension is supported by the high incidence of left ventricular hypertrophy in autopsy cases of ICH and the significantly higher admission blood pressure readings in ICH patients compared with those with other forms of stroke.[72]

The guidelines for the management of acute ICH published by the Special Writing Group of the Stroke Council and the American Heart Association, acknowledge the absence of systematic overviews and randomised interventional trials. They, however, recommended treatment of hypertension as the most effective means to prevent primary ICH and its associated morbidity and mortality. Many studies have identified hypertension as a major risk factor for the development of primary and recurrent ICH. Chronic hypertension (HTN) is the most important risk factor for ICH and is responsible for almost 60% of cases.[39,40,41] The strength of association between BP and haemorrhagic stroke appears to be greater than that observed for ischaemic stroke. A study on 17 cohorts from the People's Republic of China and Japan involving 115 757 participants showed a significantly stronger association of usual diastolic BP with haemorrhagic stroke than with nonhaemorrhagic stroke, and the odds ratio for 5 mmHg lower BP was 0.54 [95% CI, 0.50–0.58] vs 0.61 [95% CI, 0.57–0.66] for haemorrhagic and ischaemic stroke, respectively.

Elevated blood pressure is common acutely after ICH, even in patients without a prior history of HTN[57,58,59]. In most cases, blood pressure spontaneously declines over 7 to 10 days, and the maximal decline occurs over the first

24 hours[60].

In cohorts from Japan and the People's Republic of China according to usual diastolic BP (Eastern Stroke and Coronary Heart Disease Collaborative Research Group, 1998), logistic regression analyses showed a stronger association of usual diastolic BP with haemorrhagic stroke than with nonhaemorrhagic stroke (OR for 5 mmHg decrease 0.54 CI: 0.50–0.58, vs 0.61 CI: 0.57–0.66, P for heterogeneity 0.02). (The Lancet, 1998; 352: 1801–1807).

With regard to haemorrhagic stroke there is a mechanism of action plus evidence that blood pressure reduction can prevent the risk of stroke.[82] It is well established that hematoma expansion in spontaneous ICH occurs within the first 24 hours after ictus in about one third of patients[48.49]. Hematoma volume and hematoma expansion are predictors of 30-day functional outcome and mortality[48,50.51]. A direct relationship between blood pressure and risk of hematoma enlargement has been shown in some studies, but others do not support this association[52,53,54]. It is also unclear if high blood pressure is the cause or a hemodynamic response to the growing hematoma[54].

The Systolic Hypertension in the Elderly Program (SHEP) Study determined the effects, on stroke, of treating isolated systolic hypertension in 4736 elderly patients. Antihypertensive treatment appears to be more protective against haemorrhagic stroke than ischaemic stroke, the relative risk reduction for haemorrhagic stroke being 0.46 (95% CI: 0.21–1.02) compared to 0.63 (95% CI: 0.48–0.82) for

ischaemic stroke. Treatment benefits were also reported to appear earlier for haemorrhagic stroke compared to ischaemic stroke, observed within 1 year for haemorrhagic stroke but not until the second year for ischaemic stroke. However, the number of patients with haemorrhagic stroke was only 28.

High BP has also been shown to be an important risk factor for the recurrence of ICH. In a prospective study of 74 patients with hypertensive brain haemorrhage, higher diastolic BP was related to an increased rate of rebleeding. Recurrence rate was calculated to be 10.0% per patient-year among individuals with diastolic BP>90 mmHg and <1.5% in those with lower diastolic BP. A retrospective study of 51 patients with previous ICH also showed the stroke recurrence rate to be lower in a group with post-stroke BP<80 mmHg compared to those with post-stroke BP >90 mmHg.

The recently reported Perindopril Protection Against Recurrent Stroke Study (PROGRESS) investigated the effects of BP lowering with an angiotensin-converting enzyme (ACE) inhibitor-based regimen in patients with a history of cerebrovascular disease. Compared with placebo, treatment with the ACE inhibitor, perindopril, with the addition of a diuretic, indapamide, in 58% of subjects, resulted in a significant 28% (95% CI: 17–38) reduction in the occurrence of all stroke. Treatment appeared to offer greater protection against haemorrhagic stroke, reducing risk by 50% (95% CI: 26–67) compared to ischaemic stroke, which reduced risk by 24% (95% CI 10–35). The role of BP in the prognosis and major complications of acute ICH and

optimal management of BP in the early stages of acute ICH is still unclear.

Abott et al showed an increased risk of intracerebral haemorrhage in cigarette smoking Hawaiian men of Japanese ancestry. The risk of haemorragic stroke was 2.5 times higher in smokers, an effect that is independent of other risk factors. However, the diagnosis of ICH was often made on clinical grounds without verification by imaging or autopsy findings.In a recent study on computed tomography diagnosis of ICH in Finland, Juvela et al found that smoking was not independent for ICH. In our study too, only **28%** of the patient were found to be **smokers** and and in none of the patients with ICH, smoking was an isolated risk factor. Similarly the incidence of diabetes mellitus in our study was only **15%** indicating that **DM** is a less important risk factor for ICH compared to untreated hypertension.

Excessive alcohol consumption is associated with increased risk of ICH and should be discouraged (class II).[68] The series reported by Donaline et al and Juvela et al documented an increased risk of ICH in relation to alcohol ingestion, an effect that operated independently from other risk factors. Both studies showed a strong dose – response relationship between alcohol use and ICH.Alcohol induced hypertension predisposes to spontaneous intracerebral haemorrhage.[93] The series of Juvela et al documented a similar effect for alcohol ingestion within 24 hrs from ICH onset and within 1 week from onset.[90]

In our study, 47% of patients had given H/o regular alcohol

consumption, making alcohol ingestion a single predominant avoidable risk factor for ICH. In addition, those who consumed alcohol within 48hrs preceding the ictus was 32% which again is a significant figure statistically. Among the 69 patients who had a good outcome among the 231patients in our study,only 21(30%) had had an alcoholic binge in the preceding 48 hours and 48(70%) had been sober in that period.Most importantly, among the 57 patients who died in the study , 27 (47.4%) of them had consumed alcohol within 48hrs of the ictus making it clear that alcohol ingestion not only increases the incidence of ICH but also mortality, especially in those with a recent binge.

Clinical features of ICH associated with increased ICP are headache and vomiting. Although they vary greatly in their frequency, depending on the location of the haemorrhage, their overall importance at the onset of ICH is limited.[91] Of the 54 patients alert enough to report the symptom, only 36% reported headache in the series of Mohr et al. Aring's series disclosed a frequency of headache of 23%. The reporting of vomiting at onset follows similar frequencies of 44% and 22% in these two series. In our study, headache was reported by 44% of patients, vomiting by 56% and both headache and vomiting by 35%. These findings stress the important clinical point that absence of headache and vomiting does not rule out ICH.[89] On the other hand, when present, these signs suggest ICH as the most likely diagnosis, since occlusive strokes show them in less than 10% of cases.[88] This corresponds to the conclusion of Mohr, Caplan, and Melski (1978) regarding significance of headache and vomiting in the diagnosis of ICH.

Seizures at the onset of ICH is uncommon.[88] They have been reported at the rates as low as 7%, 11% and 14% in the studies of Mohr, Furlan, and Aring respectively, when all forms of ICH is considered together. In some groups such as in those with lobar haemorrhages, according to Kase, Willams, and Wyatt et al, seizures have been reported in as many as 32% of patients. In our study interestingly almost similar figures are seen i.e. the overall incidence is about 13% and the incidence of seizures in Lobar, putaminal, thalamic and infratentorial locations of haemorrhage, are 30%, 16%, 5%, and 0% respectively. Another interesting observations in our study is that almost all patients of putaminal haemorrhage who had seizures had fairly large haematomas with volume being in excess of 40ml. Among the focal symptoms, like most previous studies, limb weakness was the commonest being 81% followed by speech disturbances in 57% of patients in our study.

Patients with ICH frequentlyshow some degree of decreased alertness at the time of admission, as a consequence of increased ICP. According to Wiggins, Moody, Toole et al, the frequency and severity of this sign vary to some extent according to the location of the haemorrhage, but when all forms are considered, it is present in atleast 60% of the cases and in two thirds of them to the extent of being comatosed. According to Portenoy, Lipton and Berger et al, coma has correlated with ventricular extension, large size of haematoma and poor vital prognosis.[88]

The degree of impairment of consciousness depends on the location, size and extension of the haematoma (deep structures and ventricles) and

some degree of altered consciousness was present in 60% of the cases reported by Mohr and co-workers.[91] In our study, about 52% of patients presented with disturbed consciousness and among them all those with significant loss of consciousness had a poor outcome with an mRS of 5 or 6.

According to Fisher, Picard and Polak, ICH occurs characteristically during activity and onset during sleep is extremely rare.[88] It occurred in only one instance in Fisher's series, and in only 3% of ICH cases included in the NINCDS stroke data bank(Kunitz, Gross, Heyman et al — 1984) . In our study, 8% of patients gave history of onset during sleep and another 4% on awakening. In about 63% of patient the onset was during activity, most of them during light activity.

In hypertensive patients, cerebral infarction is secondary to increased atherosclerosis, whereas cerebral haemorrhage is the result of both elevated arterial pressure and the development of cerebral vascular microaneurysm . Only age and arterial pressure are known to influence the development of microaneuysms and rupture of the microaneurysms is the usual cause of hypertension associated intracerebral haemorrhae.[84]Higher levels of both systolic and diastolic blood pressure have been associated with an increased incidence of haemorrhagic strokes in patients of all ages.[85] Thus it is not surprising that arterial pressure shows a better association with intracerebral haemorrhage than with either cerebral or myocardial infarction. In 1855, before blood pressure could be measured, Kirkes observed hypertrophy of the heart in 17 of 22 patients with fatal brain haemorrhage.[63] In our study about 49% of

patients had their admission Diastolic BP more than 100mm of Hg and about 65% of the patients had their systolic BP more than 160mm of Hg.In one study of 188 patients with primary ICH, researchers determined the cause to be hypertension in 72% of patients.[93]

The National Institute of Health stroke scale(NIHSS) is the most commonly used scale for grading severity of stroke. It summarises all the impairments of stroke into one scale. It is simple and short. The generally accepted neurologic examination for stroke patients is the NIHSS, which assesses neurologic domains such as level of consciousness, motor, sensory, speech, and language. This standardized tool aids in localizing the stroke lesion and in quantifying the deficit.[92] Among the 57 patients who died in our study, none of them had a NIHSS score of less than 16, and only 21% of had a score of 16 to 22 and about 79% of had a score of more than 23 indicating a very severe impairment. Among the 72 patients with a very severe NIHSS score, 45 (62.5%) died in the first month. Thus it can be inferred that NIHSS score is an indication of severity of the ICH and those with very severe score have about 63% probability of mortality in the first month after the ictus.

Among those who died in the first month, none had an ICH score of 0, 6 (10%) had a score of 1, 12 (20%)had a score of 2 and the rest i.e. 39 (69%)had a score of 3 or 4. This indicates that the patients with the ICH score of 0 usually survive and the 30 day mortality increases steadily with ICH score. Thus it can be inferred that the ICH score is a simple clinical grading scale that allows the risk

stratification on presentation with ICH. The use of a scale such as the ICH score could improve standardization of clinical treatment protocols and clinical research studies in ICH.

Spontaneous ICH occurs predominantly in the deep portions of the cerebral hemispheres. The haemorrhages of putaminal, thalamic, and pontine location occur in the vascular distribution of small perforating intracerebral arteries, lenticulostriate, thalomaperforating and basilar paramedian groups respectively. Cerebellar haemorrage occurs in the area of dentate nucleus, which is supplied by small branches of both the superior and the anterior – inferior cerebellar arteries. Thus most ICHs originate from the rupture of deep arteries, of diameters between 50 and 200mm. These same arteries are recognized to be those occluded in cases of lacunar infarcts, a form of stroke correlated with chronic hypertension. Thus, it is apparent that those various groups of small arteries, located in well defined anatomic areas become the target of chronic hypertension and the result can be either occlusion or rupture, leading to lacunar infarcts or ICH, respectively.

The most common location of spontaneous intracerebral haemorrhage is the putamen, accounting for about 35% to 50% of the cases. In our study too, putamen is the commonest site of ICH, the incidence being 54.5%. A biracial population study of 1,038 ICH cases showed that 49% were located deep in hemisphere, 35% lobar, 10% cerebellar, and 6% in the brain stem[46]. The hematoma location may be suggestive of the underlying etiology. Among 100 cases of ICH at the University of

South Alabama Medical Centre, Kase and associates found putaminal haemorrhage in 34, lobar haemorrhage in 24, thalamic haemorrhage in 20, cerebellar haemorrhage in 7, pontine haemorrhage in 6, caudate haemorrhage in 5, and putaminothalamic in 4.When this is compared with the frequency of ICH location in our study, it can be seen that except for lobar haemorrhages which are substantially less common in our study,the incidence regarding other locations are fairly similar. The reason may be that cases of ICH due to AVM, aneurysmal rupture etc. were excluded from our study. This again reiterates the fact that cases of hypertensive ICH is common at deeper locations.

Spontaneous intracerebral hemorrhage (ICH) is a neurologic emergency that accounts for about 10% to 20% of all strokes and has a 30-day mortality rate of 35% to 52%[35,36]. Furthermore, only 21% of patients suffering ICH are expected to be independent at 6 months [37]. Despite advances in our understanding of the pathophysiology and complications associated with ICH, in-hospital mortality from ICH decreased by a mere 6% between 1990 and 2000, compared with 36% and 10% mortality reductions achieved for ischemic stroke and subarachnoid hemorrhage, respectively. [38]Bamford et al reported a 30 day mortality of 44 – 51% in paients with ICH, with the majority of early deaths occuring due to brain herniation.[87] In the study of brain herniations in patients with ICH, by Kalita et al, out of 24 patients, 13 patients died by 1 month, with a 30 day mortality of 54%.[86]

Various authors, including Bogousslavsky et al, Bozzola et al,

Portenoy et al, and Sacco et al reported a mortality rate varying from 20% to 70%.[89] Although the mortality is strongly dependent on haematoma size and to a lesser extent, location, the overall mortality rate varies between 25% and 60%.[93] Among the total number of 231 patients in our study, the number of patients who died (mRS of 6) in the first month is 57 (25%) and those who had severe disability (mRs of 5) is 87 (38%).

According to Flaherty et al, death at 1 year varies by location, being highest for brainstem at 65%, 57% for lobar, 51% for deep ganglionic, and 42% for cerebellar.[66] The mortality rates reported for lobar haemorrhage have been between the extremes of 11.5%(Ropper&Davis, 1980) and 32%(Kase et al., 1982), in comparison with 42% basal ganglionic and thalamic ICH, and 43% for posterior fossa haemorrhages(Steiner et al., 1984). However in large series of patients comparisons of mortality lobar and deep hemisheric ICH have shown no significant differences (Massaro et al., 1991).

These authors reported a 30-day fatality rate of 27.7% for patients with lobar haemorrhage, and 31.8% with deep haemorrrhages.These differences in mortality among series of lobar and deep ICH may reflect vaiation in haematoma size and mass effect more than superficial vs. deep location.[73] In our study, 30 day mortality is highest for cerebellum at 66%, 50% pons, 26% at putamen, 17% at lobar, 10% at

thalamus,and 0% at caudate nucleus,reflecting the fact that the prognosis is worse for infratentorial ICH compared to the supratentorial haematomas.

In 1961 McKissock and colleagues reported the first randomized, prospective, controlled trial which suggested that surgical evacuation was not beneficial for treating ICH. In total, 12 prospective randomized trials have investigated the role of surgical evacuation in the treatment of ICH. In summary, these results have shown that surgery does not significantly improve outcomes when compared with medical therapy alone. In 2005 the landmark Surgical Trial in Intracerebral Hemorrhage (STICH) study prospectively examined the role of surgery in 1033 patients. Surgical clot removal within 24 hours of spontaneous supratentorial ICH was of no significant benefit however, the STICH results must be interpreted with caution because high crossover from the medical group to surgery (140 out of 530 patients) and the lack of clinical equipoise regarding surgical evacuation of large lobar hematomas and nondominant hemisphere hematomas leading to nonrandomization into STICH may have resulted in bias favoring medical treatment . A meta-analysis examining mortality in these 12 studies reported 392 of 1182 (33%) deaths in the medical group versus 346 of 1183 (29%) in the surgical group. Although no statistically significant benefit can be attributed to surgical treatment, the trend favoring surgery is seen despite imminently herniating and deteriorating patients being excluded from the trials. Although the preponderance of evidence suggests that routine

surgical evacuation of supratentorial hematomas is not superior to medical treatment alone, surgery may be beneficial in a subset of patients.[78]

Putaminal haemorrhage is the most common form of ICH and can manifest in different ways, depending on the size and extent of the haematoma.[89] Dense neurological deficits associated with coma usually suggest large haematomas and are associated with a poor prognosis. Intraventricular extension also implies extensive parenchymal dissection or destruction.[94] Patients who present with partial motor deficits, an alert neurological status, normal extraocular movements, full visual fields, and no lateral or upward extension of the haematoma have a better prognosis on the basis of reversible compression of capsular fibres, as the internal capsule is either displaced medially or directly involved by the haematoma.Modern CT series of putaminal haemorrhage document a mortality rate of 37%, in contrast to 65% to 75% from pre CT era. This difference reflects the description of the full spectrum of haematoma size in recent reports, including benign haematomas with benign outcomes, which were misdiagnosed as infarcts in the pre CT era.[93] In our study, we have come across putaminal haemorrhages of various sizes ranging from 0.75ml to 144ml. On analyzing the outcome with respect to volume it is evident that the morbidity and mortality steadily increase with increasing volume of ICH, with all the 9 patients with a volume of more than 100ml dying in the first month. Among 126 patients with putaminal haemorrhage, 48 had ventricular extension, all with volumes more than those with no IVH,and with worse

outcome with 50% dying in the first month and another 50% being left with severe disability. These factors are consistent with the earlier observation of Hier, Davis, Richardson and Mohr et al that a putaminal haematoma that directly extends into the ventricle is usually of large size and is associated with high mortality.[94]

The thalamic form of ICH accounts for 10% to 15% of parenchymatous haemorrhage (Walshe, Davis and Fisher et al, 1977).[88] Compared to similar earlier studies, the incidence of thalamic haemorrhage is more in our study, being 26% versus 15%, the reason being the relative absence of nonhypertensive etiologies in our patients. Thalamic haemorrhages of various volumes have been encountered, with the smallest being 0.5ml and the largest 24ml. Compared to putaminal haemorrhages, thalamic haematomas are relatively smaller with 45% of them being smaller than 5ml and 90% of them smaller than 20ml, but more than 47% of putaminal haemorrhages are larger than 20ml. The reason for this difference may be that about 60% of the thalamic haemorrhages are associated with IVH with partial decompression of thalamic haematomas into the ventricles because of which the true intraparenchymal volume cannot be assessed easily.

Though larger haematomas are associated with poorer outcome, it is not strictly linear as can be seen from the fact that the 6 patients with thalamic haematoma who died, had a volume of 11 to 20ml and none with the volume larger than 20ml died in the first month.Similar observation was made by Piepgras and Rieger who described two patients in their series who survived with

haematomas of more than 4 cm diameter.[95]Small and moderate-sized hemorrhages that rupture into the third ventricle have been associated with fewer neurologic deficits and better outcomes.[3]

Similarly presence or absence of IVH in thalamic haematomas does not clearly predict the prognosis.According to Qureshi, Safdar, Weil et al who reviewed 182 African American patients in 1995, an ICH volume of greater than 30 ml and ventricular extension are independent predictors of early deterioration and death. This fact which holds good for our patients of putaminal ICH, interestingly can not be applied to our patients with thalamic ICH for reasons already quoted. This observation assumes significance when surgery is contemplated in a patient with thalamic ICH.

The pons is the most common location for nonvascular causes of ICH in the brainstem. Spontaneous nontraumatic midbrain and medullary haematomas are rare.[89] Fang and Foley and later Dinsdale reviewed the necropsies at Boston City Hospital and found 511 ICHs among 19,093 autopsies, of which 30 were pontine (6%).In our study ,similarly, pontine haemorrhage comprised 5.2 % of ICH cases. In the aforementioned review of 30 cases of pontine haemorrhages among 511 cases of ICH , two thirds of patients were comatose on presentation and had massive haemorrhages that extended into midbrain or fourth ventricle. Within 48hrs 78% of patients had died.

As per Steegmann and others, massive pontine

haemorrhages are always fatal and some patients with medium sized haematomas and most patients with small or segmental haematomas survive, with various degree of residual neurological deficits.[89]In a series of 60 patients with pontine hemorrhage reviewed by Nakajima, 19 survived (8 of whom had remained alert). Similarly, Wijdicks and St. Louis reported that 21 percent made a good recovery—mostly those who were awake on admission.[3] In our study, all the 6 patients with pontine haematomas of volume less than 5ml survived with an mRS of 3 and all those with a volume of more than 5ml died earlier, with 50% of them with and 50% of them without IVH. Therefore it can be inferred that with regard to pontine haematoma, it is the volume of haematoma and not the presence or absence of IVH that predicts the outcome.

Cerebellar haemorrhage appear with a frequency variously quoted as between 5% and 15% and average frequency is about 10% approximating the relative percentage of weight of the cerebellum in reference to the entire brain.[88] But according to Dinstale and Freeman, the frequency of cerbellar haemorrhage ranges between 5% and 10%. In our study, it is only 4%. In the series the Fisher et al , the left hemisphere was affected twice as often as the right. Mckissock et al also commented on a left cerebellar predominance. Most other series do not report haemorrhage laterality. Similarly, in our study the right cerebellar hemisphere has been involved twice as often as the left.

The clinical course in cerebellar haemorrhage is notoriously unpredictable; patients who are alert or drowsy on admission can deteriorate suddenly to

coma and death without warning, while others in a similar clinical status have an uneventful course with complete recovery of function. Of those patients who were not comatose on admission, only 20% had a smooth, uneventful recovery in the series of Ott et al, whereas 80% deteriorated to coma, one-fourth of them within 3 hours from the onset.[96] A similar frequency was observed in the series of Fisher et al, where only 2 of 18 patients had a benign course, the other 16 deterirating to coma at variable intervals, mostly within few hours after onset. Although most cases deteriorate early in the course, occasional patients have shown fatal decompensations at a later state, even a month later, although they were stable in the interim.

Since prediction of clinical course cannot be made based on clinical parameters on admission, the recommendation followed that surgical evacuation of the hematoma should be undertaken whenever the diagnosis is made within 48 hours from the onset. The need for prompt diagnosis and emergency surgery had its justification in the documented poor surgical outcome with worsening preoperative mental status, the surgical mortality being 17% for responsive and 75% for unresponsive patients. These figures have proved generally accurate, despite occasional reports of good surgical results in comatose patients. The use of CT scan in cerebellar haemorrhage has permitted the recognition of many different aspects of these lesions, some of which are useful early predictors of clinical course.[89]

Little and collegues reported two groups of patients with cerebellar

haemorrhage. The first group presented with an abrupt onset, progressive course and low level of consciousness. CT in this group of patients showed cerebellar haematomas 3cm or greater in diameter, obstructive hydrocephalus and extension of haemorrhage into the fourth ventricle. The second group of patients was awake and stable and had haematomas smaller than 3cm in diameter. They had good outcome. As a rule, a cerebellar hematoma less than 2 cm in diameter leaves most patients awake and infrequently leads to deterioration, therefore generally not requiring surgery. Hematomas that are 4 cm or more in largest diameter, especially if located in the vermis, pose the greatest risk, and some surgeons have recommended evacuation of lesions of this size no matter what the clinical status of the patient.[3] Similar results were obtained in our study too with 3 patients with volume of less than 10ml having a good outcome of mRS score of 2 and all the 6 patients of haematoma volume of 10 to 20ml dying in the first month. Interestingly, in our study all the 3 patients who survived did not have IVH and all the 6 patients who had larger volume and consequently ventricular extension died in the first month. Therefore it can be inferred from our study that a volume of more than 10ml and intraventricular extension are indicators of bad prognosis in cerebellar haemorhage.

According to Ropper et al, the prognosis of lobar ICH is relatively better than that of other forms of ICH and mortality ranges from 11% to 29%. According to Helweg et al, the functional outcome for survivors of lobar haemorrhage also tends to be better. In our study also, the 30 day mortality of Lobar haemorrhage is only 16.7% compared to

cerebellar, pontine and putaminal haemorrhages with 66%, 50% and 26% respectively.

In their series of 22 patients Kase and associates reported good outcomes in those with haematoma volumes less than 20 cm^3,70% survived after the surgical removal of haematomas that were 20 to 60 cm^3. No patient with a haematoma volume greater than 60cm^3 survived. Similar to these studies, among our patients too, all those who died in the first month had Lobar haematoma of volume more than 20ml and the outcome mRS was proportionate to the volume of haematoma.

With respect to ventricular extension, in contrast to other locations, patients with Lobar haematomas and IVH had a better outcome compared to those with no ventricular extension. The probable reason for this observation is that all the three patients with lobar ICH with IVH had small haematomas of volume less than 5ml but in close proximity to the lateral ventricle. This conforms to the observations of Kase et al according to whom ventricular extension in patients with lobar ICH was a factor that correlated with location (proximity to ventricular system) rather than size of the haematoma.[88]

According to Stein and associates caudate haematoma represents approximately 5% to 7% of cases of ICH, the bleeding vessels being the perforating branches of the anterior and middle cerebral arteries.[75] The outcome of patients with caudate haemorrhage is so excellent that more than 80% of patients return to normal activities and less than 20% of patients remain slightly hemiparetic with death being rare (Chung et al 2000).[76]

In our study, the incidence of caudate haematoma is as low as 3% only. With respect to volume, those with volume <5ml had complete recovery and those with larger haematoma of volume >5ml had a significantly worse outcome with an mRS of 5. Another very interesting observation in our study is that all the six patiets had ventricular extension and none died in the first month, probably indicating that ventricular extension serves as a natural decompressive mechanism with respect to caudate haematoma,making the prognosis excellent for caudate haemorrhage,especially with regard to mortatity.

CONCLUSION

1.Males predominate among patients of ICH with a ratio of 5:2.,the male predominance is probably attributable to the high prevalence of alcohol among males.

2.Hypertension is the most important causative factor for ICH.

3.Alcohol ingestion not only increases the incidence of ICH but also the mortality, especially in those with a recent binge.

4.Among patients of ICH, headache is reported by 44% of patients, vomiting by 56% and both headache and vomiting by 35%.

5.Patients presenting with significant loss of consciousness have a poor outcome with an mRS of 5 or 6.

6. NIHSS score is an indication of severity of the ICH and those with very severe score(>22) have about 63% probability of mortality in the first month after the ictus.

8. Patients with the ICH score of 0 usually survive and the 30 day mortality increases steadily with ICH score.

9.The frequency of ICH location is as follows : Putamen- 54.5% ; Thalamus-25.9% ; Lobar ICH- 7.8% ; Pons- 5.2% ; Cerebellum – 3.9% ; Caudate nucleus- 2.6% .

10.Almost all patients of putaminal haemorrhage who had seizures have fairly large haematomas with volume being in excess of 40ml and consequently a poorer outcome. Putaminal haemorrhage is the most common form of ICH and a putaminal haematoma that directly extends into the ventricle is usually of large size and is associated with high mortality.

11.Though larger thalamic haematomas are associated with poorer outcome, it is not strictly linear. Similarly presence or absence of IVH in thalamic haematomas does not clearly predict the prognosis.With regard to pontine haematoma,it is the volume and not the presence or absence of IVH that predicts the outcome but a volume more than 10 ml and intraventricular extension are indicators of bad prognosis in cerebellar haemorrhage.

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PROFORMA

NAME : AGE : SEX :

UNIT : IP NO : MIN NO :

ADDRESS/PHONE NO :

DATE OR ENTRY :

Date and time of ictus :

Date and time of admission :
In any centre:
In our centre:
Arrival time in EMD after ictus : ½ hr ( ); 1-2hrs ( ); 2-3 hrs ( ); 3-6 hrs ( ); 6-12 hrs ( ); 12-24 hrs ( ); 24-48 hrs ( ); after 2 days ( )
Educational status : pri sch ( ); sec sch ( ); U.G ( ); P.G ( ); Professional ( ); uneducated ( )

Religion :Hindu ( ); Muslim ( ); Christian ( ) others ( )

Income : Low ( ); Middle ( ) ; High ( )

Relevant medical conditions :

DM :

HT :

Habits : Ethonal :

Smoking :

Symptoms at onset : Headache ( ); vomiting ( ); LOC ( ); Seizures ( )
Limb weakness ( ); speech distur ( ); sensory loss ( );visual distur ( )

Activity when symptoms noticed first : sleep ( );on awakening ( );
While rest ( ); light activity ( );Heavy exertion ( );others ( )

H/O Bleeding Disorder/Anticoa/Antipl/Th.lytic :

Pulse/BP : RR: ;Temp: ;BMI:

CVS,RS,Abd,Others/bleeding :

GLASGOW COMA SCALE :

EYE OPENING :_____ 4 spontaneous; 3 To voice; 2 To pain; 1 None

VERBAL RESPONSE :_____ 5 Normal conversation; 4 Disoriented conversation 3 words coherent; 2 no words…only sounds; 1 none

MOTOR RESPONSE :_____ 6 Normal; 5 Localizes to pain; 4withdraws to pain; 3 Abnormal flexion; 2 Abnormal extension; 1 None

NIHSS :

_____

1a Level of conciousness :_____
0 Alert 1 Drowsy 2 stuporous 3 comotose

1b LOC Questions :_____ (Month/Age)
0 Both correct 1 one correct 2 Incorrect

1c LOC commands :_____(open,closeeyes,make fist,let go)
0 obeys both correctly 1 obeys one correctly 2 Incorrect

2 Best gaze :_____
(eyes open patient follows examiner's finger or face)
0 Normal 1 partial gaze palsy 2 Forced deviation

3 visual:_____
(introduce visual stimulus/threat to patient's visual quadrants)
0 No loss 1 partial hemianopia 2 complete hemianopia
3 Bilateral hemianopia

4 Facial palsy : _____(show teeth,raise eyebrows and squeeze eyes shut) 0 Normal
1 minor asymmetry 2 partial(lower face paralysis) 3 complete

5a Motor Arm-Left :_____(Elevate extremity 90deg and score

drift/movement)
0 drift 1 drift 2 some effort against gravity 3 No effort against gravity 4 no movement 9 amputation,joint fusion

5b Motor Arm-right :_____
(Elevate extremity 90deg and score drift/movement)
0 No drift 1 drift 2 some effort against gravity
3 No effort against gravity 4 No movement 9 Amputation,joint fusion

6a Motor leg-left :_____
(Elevate extremity 90deg and score drift/movement)
0 No drift 1 drift 2 some effort against gravity
3 No effort against gravity 4 No movement 9Amputation,joint fusion

6b motor leg-right :_____
(Elevate extremity 90deg and score drift /movement)
0 No drift 1 drift 2 some effort against gravity
3 No effort against gravity 4 No movement 9 Amputation,joint fusion

7 Limb ataxia :_____
0 Absent 1 present in upper or lower 2 present in both

8 sensory :_____
(pin prick to face,arm trunk and leg compare side to side)
0 Normal 1 partial loss 2 Dense loss
9 Best language :_____
(Name items,describe a picture and read sentences)
0 No aphasia 1 Mild-moderate aphasia 2 severe aphasia 3 Mute

10 Dysarthria:_____
(Evaluate speech clarity by patient repeating listed words)
0 Normal articulation 1 Mild-moderate slurring
2 severe,nearly intelligible 9 Not scored

11 Extinction and Inattention :_____(use information from prior to testing identify
neglect or double stimuli testing)

0 No neglect 1 partial neglect 2 profound neglect

MODIFIED RANKIN SCALE :_____

0 = No symptoms at all
1 = No significant disability despite symptoms; able to carry out all usual duties and activities
2 = slight disability; unable to carry out previous activities, but able to look after own affairs without assistance
3 = moderate disability; requiring some help, but able to walk without assistance
4 = moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5 = severe disability; bedridden, incontinent and requiring constant nursing care and attention
6 = dead

ICHSS:_____

GCS score : _____

0 = GCS 13 – 15 1 = GCS 5 – 12 2 = GCS 3-4

ICH volume cm^3:_____

0 less than 30 1 greater than 30

IVH:_____

0 No 1 Yes

Age / year : _____

0 less than 80 1 greater than 80

Time interval between onset of ictus and first CT scan:

Less than ½ hr ( ); 1 – 2 hrs ( ); 2-3 hrs( ); 3-6 hrs( ); 6 -12 hrs( ); 12 – 24 hrs( ); 24 – 48 hrs( ); greater than 48 hrs( )

Time interval between the first and second CT scan:
Less than ½ hr ( ); 1-2 hrs( ); 2- 3 hrs( ); 3 – 6 hrs( ); 6 – 12 hrs( )
12 – 24 hrs( ); 24 – 48 hrs( ); greater than 48 hrs( )

CT Findings :
ICH (location, volume, PH,oedema) / IVH (Volume)

rFVII Treatment/ surgery/ conservative treatment/Inhospital complications/Investigations :

EGOSS :_____

1 Dead; 2 veg state;3 lower sev dis; 4 upper sev dis; 5 lower mod dis;
6 upper mod dis; 7 lower good rec; 8 upper good rec
(sev-severe;mod-moderate;dis-disability;rec-recovery)

GUY'S HOSPITAL, SCORING SYSTEM (ALLEN):_____

VARIABLE	**CLINICAL FEATURE**	**SCORE**
1. APOPLECTIC ONSET		
a.LOC		
b.Headache	one or none	0
c.vomiting	Two or more	+21.9
d.Neck stiffness		
2. LEVEL OF CONCIOUSNESS		
	Alert	0
	Drowsy	+7.3
	Unconscious	+14.6
3.PLANTER RESPONSE		

	Both flexor/single extensor	0
	Both extensors	+7.1
4.DIASTOLIC B.P. (24hrs after admission)	B.P In mmof Hg	+(B.P.x 0.17)
5.ATHEROMA MARKERS (claudication, Angina, diabetes history)	None	0
	one or more	-3.7
6.HISTORY OF HYPERTENSION	Not present	0
	present	-4.1
7.PREVIOUS EVENT (TIA,Stroke)	None	0
	any number	-6.7
8.HEART DISEASE	None	0
	Each one	-4.3
	Constant	-12.6

(Heart diseases-a valvular murmur, cardiac failure, cardiomyopathy, atrial fibrillation, cardiomegally and a recent myocardial infarction)