PREDICTION OF FUNCTIONAL OUTCOME IN PATIENTS WITH SPONTANEOUS INTRACEREBRAL HEMORRHAGE USING FUNC SCORING

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CERTIFICATE

This is to certify that the dissertation entitled **PREDICTION OF FUNCTIONAL OUTCOME IN PATIENTS WITH SPONTANEOUS INTRACEREBRAL HEMORRHAGE USING FUNC SCORING** is a bonafide work done by **Dr.T.S.KARTHIGEYAN** Post Graduate Student, Institute of Internal Medicine, Madras Medical College, Chennai-3, during March 2014 to August 2014 in partial fulfillment of the University Rules and Regulations for the award of MD Branch – I General Medicine, under our guidance and supervision, during the academic year 2012 - 2015.

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DECLARATION

I solemnly declare that the dissertation entitled **PREDICTION OF FUNCTIONAL OUTCOME IN PATIENTS WITH SPONTANEOUS INTRACEREBRAL HEMORRHAGE USING FUNC SCORING** is done by me at Madras Medical College, Chennai-3 during March 2014 to August 2014 under the guidance and supervision of **Prof. S.TITO, M.D.,** to be submitted to The Tamilnadu Dr M.G.R Medical University towards the partial fulfillment of requirements for the award of M.D DEGREE IN GENERAL MEDICINE BRANCH-I.

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ABBREVATIONS

AVM	-	Arteiovenous Malformation
CAA	-	Cerebral Amyloid angiopathy
CPP	-	Cerebral Perfusion Pressure
CSF	-	Cerebro Spinal Fluid
CT	-	Computerised Tomography
EEG	-	ElectroEncephalogram
GCS	-	Glasgow Coma Scale
ICH	-	Intracerebral Hemorrhage
ICP	-	Intracranial Pressure
IVH	-	IntraVentricular Hemorrhage
MAP	-	Mean arterial Pressure
MRI	-	Magnetic resonance Imaging
PT	-	Prothrombin Time
PTT	-	activated Partial thromboplastin Time
SAH	-	Sub arachnoid Hemorrhage
UMN	-	Upper Motor Neuron
VKA	-	Vitamin K antagonist

CONTENTS

ABSTRACT

Background and Purpose— Intracerebral hemorrhage (ICH) is the most fatal and disabling stroke subtype. Widely used tools for prediction of mortality are fundamentally limited in that they do not account for effects of withdrawal of care and are not designed to predict functional recovery. FUNC score was developed as an acute clinical score to predict likelihood of functional independence.

Methods— In our study about 50 patients with ICH are selected. On admission complete neurological examination with CT Brain plain done , FUNC score made out . Patients were followed up for 90 days , FUNC score was compared with Glasgow Outcome Score at 90 days .

Results— At 90 days, 38% patients achieved independence. Age, Glasgow Coma Scale, ICH location, volume , and pre-ICH cognitive impairment were independently associated with Glasgow Outcome Score ≥ 4. The FUNC score was developed as a sum of individual points (0 –11) based on strength of association with outcome. The proportion of patients who achieved Glasgow Outcome Score ≥ 4 increased steadily with FUNC score. No patient assigned a FUNC score ≥ 4 achieved functional independence, whereas > 80% with a score of 11 did. The predictive accuracy of the FUNC score remained unchanged when restricted to ICH survivors only, consistent with absence of confounding by early withdrawal of care.

Conclusions—FUNC score is a valid clinical assessment tool that identifies patients with ICH who will attain functional independence and thus, can provide guidance in clinical decision-making and patient selection for clinical trials.

INTRODUCTION

HISTORY OF STROKE:

400 BC -Hippocrates was probably the first to write about the medical aspects of stroke[1,2].

131 to 201 AD- Galen described the anatomy of the brain and its blood vessels in detail from dissections of animal specimen[3].

1514-1564 - Andreas Vesalius challenged the Galenic tradition by dissecting humans and relying on his own personal observations instead of Galen's writings[3].

17th century - Johann Jakob Wepfer (1620-1695) and Thomas Willis (1621-1675), made further anatomic and clinical observations.

Wepfer was the first to show clearly that bleeding into the brain was an important cause of apoplexy[4,5]. Thomas Willis a physician and neuroanatomist best known for his *Cerebri Anatome*, which contained a description of a circle of anastomotic vessels at the base of the brain.

18th century - Giovanni Battista Morgagni (1682-1771), described pathology and the cause of disease. Morgagni also described cases of

intracerebral hemorrhage and recognized that paralysis was on the side of the body opposite to the brain lesion[3].

19th century- dissemination of knowledge about the pathology of stroke came with the publication of four atlases. Hooper's atlas (1828) - illustrated pontine and putaminal hemorrhages and a subdural hematoma.

Cruveilhier[6] (1835-1842), Carswell[7] (1838), and Bright[8] (1831) also published atlases containing lithographs of systemic and neuropathologic lesions.

19th century (latter half) - Rudolf Virchow (1821 to 1902), published pathologic information about vascular disease[8]. Virchow known for his classic triad of vascular thrombosis.

Sir William osler first described cerebral Embolism in patients with rheumatic carditis [9].

During the last quarter of the 20th century, there was an explosive growth of interest in and knowledge about stroke.

By the mid-1980s, CT was readily available throughout North America and most of Europe. CT allowed clear distinction between brain ischemia and hemorrhage and allowed definition of the size and location.

The advent of magnetic resonance imaging (MRI) in the mid-1980s was a further major advance. MRI proved superior to CT in showing old hemosiderin-containing hemorrhages and vascular malformations, lesions abutting on bony surfaces, and posterior fossa structures.

MRI also made easier to visualize lesions in different planes by providing sagittal, coronal, and horizontal sections. Improved filming techniques have made it possible to image the brain vasculature through the techniques of magnetic resonance angiography, magnetic resonance venography and CT angiography.

End of 20th century – with the advent of Newer Techniques, Computerised Tomography and Magnetic Resonance modalities, it is easily possible for practicians to clearly localise the lesion, its severity, and weather any chance of reversibility of brain ischemia. Various modalities of MRI are Fluid attenuating inversion recovery, diffusion – perfusion scans, functional MRI, MR spectroscopy.

WHO – updated MAY 2014 :

Worldwide, the SECOND COMMON cause of Death is STROKE. With an estimated incidence rate of between 35 and 45 cases per 100,000 population in Europe and North America, ICH generally accounts for between 10% and 15% of acute strokes on those continents.

The incidence is higher in East Asia, with estimates that ICH accounts for as many as 30% to 40% of acute stroke cases in those countries.

Hospital-based series consistently reveal an acute mortality from ICH between 35%and 65%and substantial permanent disability in at least 50%of survivors.

ANN INDIAN ACADEMY NEUROLOGY : 2012

In INDIA, studies on Intracerebral Hemorrhage is limited. Since Indian population is Heterogenous

STROKE : INDIAN SCENARIO (2008)

The stroke subtypes and the ratio of cerebral infarct to haemorrhage range as 1.86:1 to 2.21:1[10]. ICH is more prevalent in Eastern India, Infarct to Hemorrhage is 5 :1[10]. Among ICH, Basal Ganglia is commonest, constitutes 75 % (Kolkata study) Because mortality figures are all confounded by the fact that withdrawal of aggressive care by clinicians and families is a very common precipitant of death in these patients and often occurs in patients whose ICH is survivable, it is more useful to focus on rates of disability among survivors when discussing prognosis.

Because ICH is a devastating condition that most commonly affects the elderly, physicians and families are often confronted with the question of whether their patient or loved one would choose to survive the event.

Accurate prediction of prognosis is essential to guide such decision-making. In this context, tools that predict mortality are of limited utility, as they do not give any guidance on the likelihood of functional recovery among survivors.

The FUNC SCORE [11], enables prediction of the likelihood of recovering functional independence for patients with primary ICH.

Tools such as the FUNC score calculator can be useful in guiding decisions about aggressiveness of care, but their precision remains to be proved.

FUNC score[11] has a total of 11 , higher the score more the good prognosis. Patients with FUNC score < 4 never achieve Functional independence.

The Aim of my study is to Retrospectively assess clinical profile of patients admitted with Spontaneous Intracerebral Haemorrhage in RGGGH and the utility of FUNC SCORE in predicting likelihood of functional independence.

AIMS AND OBJECTIVES

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1. To Assess the clinical profile of patients with Spontaneous Intracerebral Hemorrhage.

2. To Assess the utility of FUNC SCORE in evaluating patients with Intra cerebral Hemorrhage and prognosticating.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Intracerebral Hemorrhage

Intracranial hemorrhage is formed by bleeding directly in and around the brain[12], this produces symptoms of neurologic involvement by producing mass effect of adjoining neural structures , and also by the toxic effects of extravasated blood itself, or else by increasing intracranial pressure.

The primary cause is usually systemic hypertension which can be longstanding or recently detected, with leakage of blood by spontaneous rupture of deep penetrating artery by the sudden elevation blood pressure. ICH is otherwise most often the acute manifestation of progressive chronic blood vessel disorder of the brain. Overall Hypertension accounts 70% to 80% intracerebral hemorrhage[14]. It accounts for 10% to 15% of acute strokes in North America & Europe whereas it constitutes 30% to 40% asian population.

Intracerebral Hemorrhage is the most common type of intracranial hemorrhage. It accounts for 10% of all strokes and is associated with a 50% case fatality rate[12]. Incidence rates are particularly high in Asians and blacks. Hypertension, trauma, and cerebral amyloid angiopathy constitutes the majority of Intracerebral hemorrhages. Advancing age and alcohol

consumption increases overall risk, cocaine and methamphetamine abuse is most important causes in young individual.

Risk factors for Primary spontaneous intracerebral hemorrhage include acute physiologic derangements , chronic exposures and chronic conditions.

The common sites of a Intracerebral hemorrhage are (i) the Basal ganglia (putamen) and internal capsule (constitutes 50 percent)[12] (ii) the white matter of the Temporal, Parietal and frontal lobes (lobar hemorrhages, rarely associated with systemic hypertension) (iii) thalamus (iv) cerebellar hemisphere and (v) Brainstem pons[12].

Secondary intracerebral hemorrhage causes are head trauma, hemorrhagic infarct, metastatic brain tumor, coagulopathy, drugs, vascular malformations such as arteriovenous malformation & aneurysm, amyloid angiopathy, cavernous angioma, dural arteriovenous fistula, capillary telangiectasias. Various Hemorrhagic disorders: acute Leukemia, aplastic anemia, idiopathic thrombocytopenic purpura, liver disorders, anticoagulant or thrombolytic therapy complication, hypofibrinogenemia, clotting disorder –hemophilia.

Patients older than 55 years account for majority of cases with intracerebral hemorrhage in which chronic hypertension, cerebral amyloid

angiopathy, and chronic use of anti thrombotic medication are the leading risk factors.

Other causes for intracerebral hemorrhage include history of prior stroke, chronic alcoholic use, family history, cocaine abuse, and liver disease.

Recent data suggest aggressive lowering of serum cholesterol or chronic use of statin therapy can increase the risk for intracerebral hemorrhage especially in the elderly.

Rare Miscellaneous causes - vasopressor drugs, Moya moya disease, cocaine, herpes simplex encephalitis, meningeal melanomatosis, Tularemia, Necrotizing hemorrhagic Encephalitis (Hurst disease), Anthrax [15]...

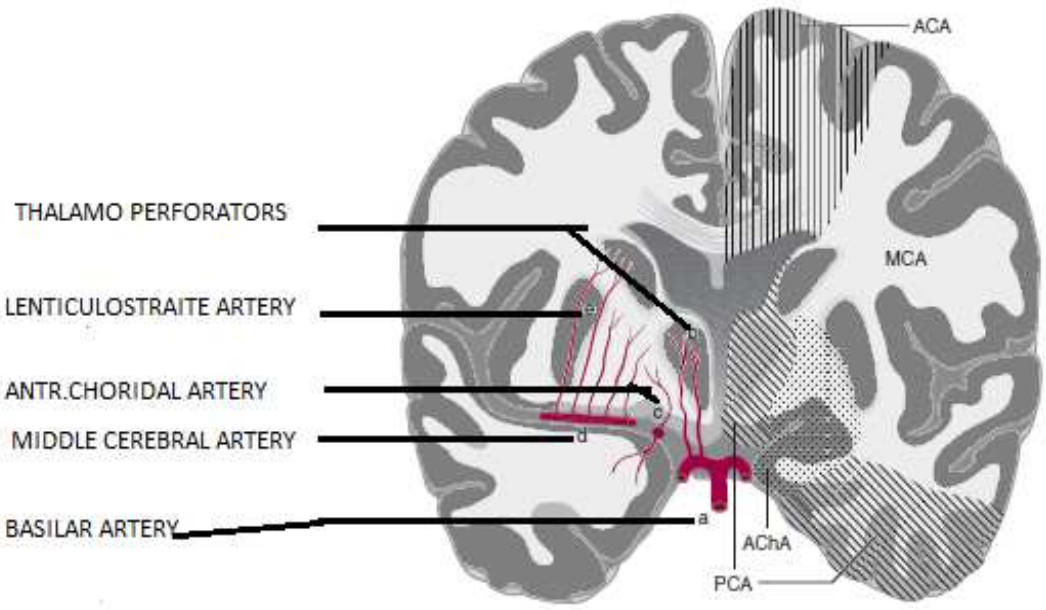
Intracerebral Hemorrhage roughly classified into

(i) Small hemorrhage[15] – with 1 to 2 cm in diameter and < 20 mL in hematoma volume.

(ii) Massive hemorrhages[15] - > 2 cm in diameter, hematoma volume greater than 50.

Coronal view of the cerebral hemispheres showing the vascular supply territories:

The picture shows territories supplied by the anterior cerebral artery (ACA), middle cerebral artery (MCA), posterior cerebral artery (PCA), and anterior choroidl artery (AChA).



PATHOPHYSIOLOGY

The factors which determine the degree of damage are location, the exact volume, the rapidity of accumulation and the pressure of bleeding. The nature of Intracerebral hemorrhages are soft at first and slowly passes in between white matter fiber tracts. When bleeding enters into the ventricular system and or the surface of the brain, the extravasated blood is introduced into the cerebro spinal fluid. The extravasated blood in the hematoma first clots and then solidifies, causes brain tissue swelling. Later, cavity or a slit is formed in the brain parenchyma due to absorption of blood and clearance of debris by macrophages.

The brain is protected from outside injury by the barrier action of bony skull and dura matter. During acute conditions such as intracerebral bleed or ischemia or cerebral edema, the extra compression leads to brain herniation into adjacent compartment and ultimate catastrophe. Hemorrhage into the brain parenchyma is often preceded by hypertensive damage to small cerebral penetrating arteries and arterioles. Small aneurysmal dilatations, first elucidated by Charcot and Bouchard in the 1870s, represent weak points that rupture under increased arterial tension.

In the majority of patients, abrupt elevation in blood pressure causes rupture of small penetrating arteries that had no prior vascular damage[16,17],

causing Leakage from these vessels producing a sudden but local pressure effect on surrounding capillaries and arterioles, causing them in turn to break[16,17].

An avalanche-type effect occurs, in which vessels at the surrounding edge break, adding extra volume to the progressively enlarging hemorrhage. The blood that is accumulating along the surrounding edge of the hematoma pictures like a snowball rolling the downhill, it gathers volume along edges as it moves downward.

The increasing blood pressure and the above avalanche type effect increases the hemorrhage, at the same time mounting local tissue pressure acting like a tamponade to the bleeding.

Serial autopsy fingings suggest large blood mass at the lesions center whereas at the periphery the fibrin globes, showing caps of fibrinous material which joins the broken and leaked small vessels. As ICH progresses, central core pressure increases and it compresses periphery small vessels of the hematoma, because of this increasing pressure the peripheral capillaries and arterioles break further increasing the hematoma size. The hematoma grows and rolls down like a smooth snowball, accumulating gradually more snow on the outer circumference as it moves. Intracranial pressure and tissue pressure increases as the enlarging lesion, until a final equilibrium is attained

between the hematoma pressure and surrounding pressure, finally stopping further bleeding.

Whenever the hematoma reaches brain surface or ventricular system, it will communicate with Cerebrospinal fluid, leading to seepage of blood. This extrablood in Cerebrospinal fluid decompress the pressure within the lesion.

If the hemorrhage becomes sizable, the increase in intracranial volume will increase intracranial pressure causing the venous pressure in the draining dural sinuses to increase. To perfuse the brain, the arterial pressure must rise to produce an effective arteriovenous difference. Thus, the patient with intracerebral hemorrhage may have a markedly elevated blood pressure merely because of the hemorrhage, not necessarily reflecting the true level of premorbid blood pressure. Although lowering this pressure does help to stop bleeding, caution must be exercised because the elevated pressure also serves to perfuse the areas of the brain not damaged by the hemorrhage.

Patients with intracerebral hemorrhage often worsen during the first 24 to 48 hours after their initial symptoms. This worsening can be explained by continued bleeding around the lesion, the edema around the lesion[18,19], the effects of the lesion on blood flow and metabolism, Some patients who had stabilized during the first 24 to 48 hours later developed progressively decreased alertness and increasing focal signs within 48 to 72 hours,

probably caused by the development of edema around the hematomas. and, in large hemorrhages, may be due to shifts in brain contents and herniations. Effects caused by masses in patients with hematomas are more common than in patients with ischemia because an extra volume of hematoma has been added in addition to the surrounding edema. Most often, pressure effects in hemispheral hematomas result in a shift of the midline without herniation of brain contents.

When mass effects are severe, brain tissue bulges out into a different compartment— called as herniation[20-22].

The various herniation of brain are herniation of the temporal lobe via the tentorial notch, leading to compression of brainstem midbrain, Herniation of cerebral hemispheres on the rostral brainstem structures causes elongation, Herniation of cingulated gyrus which forms the anterior medial frontal lobe occurs under the falx cerebri. Cerebellar herniation occurs upward through the tentorial notch, to compress the brainstem whereas downward herniation of cerebellar tonsils through the foramen magnum causes compression of lower most brainstem medulla and upper cervical spinal cord .

Herniation of brain contents may also lead to compression of arteries leading to infarction and secondary hemorrhages. These secondary vascular

changes may involve the posterior cerebral artery and or the anterior cerebral artery.

Düret hemorrhages[23] are a type of secondary hemorrhages produced by distortion of the upper brainstem midline and paramedian vessels at the tentorial opening.

The ventricular system are also liable to compression at various sites, such as putamen and cerebral lobe hematoma cause compression of foramen of Monro, leading to contralateral lateral ventricle dilatation, Thalamic hematomas usually obstruct third ventricle, causing both lateral ventricles to dilate, Cerebellar hematoma causes compression of fourth ventricle and cerebral aqueduct, causing obstructive hydrocephalus of both lateral and third ventricles. Hence the clinical worsening of signs and symptoms of intracerebral hemorrhage are herniations, and secondary Hemorrhages, and obstructive hydrocephalus.

Cerebral Autoregulation

Cerebral blood flow Autoregulation[12] is achieved by caliber changes of low resistance cerebral arteries, and it maintains a constant cerebral blood flow as and when systemic blood pressure increases and decreases.

Cerebral blood flow is constantly maintained in normal range over a great range of varying blood pressure . At a very low pressures, cerebral hypoperfusion develops, leading to syncope. Systemic Pressures that rises beyond the autoregulatory range can lead to hypertensive encephalopathy.

The shift in the autoregulatory range to higher blood pressures is by the change in Structure of cerebral arteries. Hypoperfusion and syncope may occur at normal pressures too, But pressures associated with hypertension induced encephalopathy are always increased.

In normotensive individuals, autoregulation[12] is effective is upto 60 mm Hg which corresponds to lowest mean blood pressure. On pressure Below 60 mm Hg , cerebral artery caliber changes cannot compensate for decreasing perfusion pressure leading onto decline in cerebral blood flow, thereby producing hypoxia symptoms, such as light headedness, dimming of vision and confusion, following which somnolence, stuporous and loss of consciousness occurs if the blood pressure decreases below 35–40 mm Hg. In contrast to this, at blood pressures above a upper limit of autoregulation range (ie.150–200 mm Hg), hypertensive encephalopathy occurs due to increased cerebral blood flow.

In chronically hypertensive persons, who are prone to damage of small arterial walls the lowermost limit of autoregulatory range is higher about

120 mm Hg, Hence cerebral blood flow decreases when mean arterial blood pressure decreases below this range . Hence in stroke patients the blood pressure should not be reduced, and never to a hypotensive levels.

Chronic Hypertension

Chronic hypertension found to create structural changes in the penetrating arteries walls, leading them prone for intracerebral hemorrhage. Charcot and Bouchard was the first to describe minute aneurysms in the small intraparenchymal arteries in patients with chronic hypertension and he postulated rupture of these small aneurysm causes intracerebral hemorrhage.

Subsequently, Ross Russell too showed these small resistance arterial microaneurysms in cerebral sites which are prone for hypertensive hemorrhages. Small areas of hemorrhage surround some of the Aneurysms, and they show lipohyalinosis or fibrinoid necrosis like changes. These changes lead to destruction of vessel wall with fibrinoid material deposition, focal vessel wall aneurysmal expansion, occlusion by thrombosis, and red cell extravasation .

Recent studies reveal massive intracerebral hemorrhage often due to the rupture of microaneurysm or lipohyalinotic segment of these small arteries in which the chronic hypertension found to be the underlying lesion .

Acute Hypertension

Acute elevation of blood pressure in chronic hypertension too found to play a major role in the intracerebral hemorrhage pathogenesis. In addition to chronic hypertension which causes cerebral arterial wall structural changes, Various studies suggest that a sudden rise in blood pressure may itself be enough for development of intracerebral hemorrhage even though he is not a known hypertensive, and lacks signs and symptoms of End organ damage such as Left ventricular Hypertrophy, Hypertensive Nephropathy or Hypertensive Retinopathy.

Amphetamine or cocaine abuse its may be one of the causes for sudden accelerated Hypertension.

CAUSES FOR ICH

Hypertension

It is the predominant cause for intracerebral hemorrhage, but there is know need for the blood pressure to be elevated always to malignant ranges. Many patients report to the casualty with intracerebral hemorrhage, have no past history of systemic hypertension but has peak blood pressure on admission to hospital. In this situation, its difficult to know how much the blood pressure elevation is secondary to raised intracerebral hemorrhage (Cushing response), and what is the level of blood pressure were before the intracerebral hemorrhage. When systemic hypertension first occurs, the small arteries and capillaries were exposed to a high blood pressure leading onto leak. The mitral valve stenosis hemodynamics situation is comparable to intracerebral hemorrhage. Left atrial failure in mitral valve stenosis results in increased pulmonary veins pressure to perfuse the lung parenchyma, this increased pressure causes break in pulmonary capillaries and arterioles resulting in Hemoptysis.

Later these small vessels hypertrophy, to protect the capillary bed from this high pressure. Subsequently hemoptysis settles, but the increased pressure overload to left heart leads to right heart failure.

Similarly in cerebral circulation, increased arterial pressure, in early development of hypertension, causes small vessels to rupture. Subsequently

in the chronic course of hypertension, degenerative changes occur such as lipohyalinosis and miliary Charcot Bouchard aneurysms. these degenerative changes cause small vessel rupture. Hence intracerebral hemorrhage is biphasic, occurring both at onset and later of hypertension, after developing considerable degenerative changes on penetrating small arteries. Aneurysms can have early aneurysmal dilatations and sometimes sclerosed aneurysms.

The presence of hemosiderin - laden macrophages surrounding Microaneurysms[24,25] indicates previous leakage. These lesions are mostly seen in penetrating arteries supplying Thalamus Basal Ganglia , Pons and Cerebellum , also in Areas supplying the cortical gray-white matter junctions of the cerebral hemispheres.

In hypertensive haemorrhage Putamen is the most common site and the adjacent internal capsule is usually damaged. Contralateral hemiparesis is therefore the sentinel sign. When mild, the face sags on one side over 5–30 minutes speech becomes slurred, the arm and leg gradually weaken, and the eyes deviate away from the side of the hemiparesis. The paralysis may worsen until the affected limbs become flaccid or extend rigidly.

Dysarthria and dysphagia may occur. As the hours pass, the patient often becomes stuporous and then comatose from brainstem compression or obstructive hydrocephalus; immediate surgical evacuation before brainstem

compression occurs may be lifesaving. Hydrocephalus from fourth ventricle compression can be relieved by external ventricular drainage, but definitive hematoma evacuation is essential for survival. If the deep cerebellar nuclei are spared, full recovery is common.

Various studies report that rupture of penetrating arteries can occur in the setting of acute blood pressure changes Who is not a known hypertension. Intracerebral hemorrhage can occur after severe cold weather exposure.

This occurance can be attributed to sympathetic nervous system stimulation produced artificially cold water immersion.

Various Dental procedures and surgeries directly involving the trigeminal nerve, have reported to be associated with Intracerebral hemorrhage.

Intracerebral hemorrhage also occurs in illicit drug abuse Such as cocaine and methamphetamine which has sympathomimetic action.

Older patients, often in their 70s or 80s, may present with ICH. Is this because of degenerative changes in these patients' arterial system? Does ICH occur because of coexistent amyloid angiopathy, which is recognized with increasing frequency when sought in elderly patients with lobar

hemorrhages? Older patients seem to develop ICH at relatively lower blood pressures than younger patients. Probably because of atrophy, symptoms of increased ICP, such as headache, vomiting, and reduced alertness, are less common in older patients, even with sizable lesions. This observation makes differentiation between hemorrhage and infarction more difficult in geriatric patients.

Hypertensive encephalopathy[12] is one of the malignant hypertension complication. Patient present with severe hypertension, headache, nausea, vomiting, convulsions, altered sensorium, stupor, and coma.

There are retinal hemorrhages, exudates, papilledema (hypertensive retinopathy), and evidence of renal and cardiac disease. In most cases ICP and CSF protein levels are elevated. MRI brain imaging shows a pattern of typically posterior (occipital > frontal) brain edema that is reversible and termed reversible posterior leukoencephalopathy. The hypertension may be essential or due to chronic renal disease, acute glomerulonephritis, acute toxemia of pregnancy, pheochromocytoma, or other causes. Lowering the blood pressure reverses the process, but stroke can occur, especially if blood pressure is lowered too rapidly. Neuropathologic examination reveals multifocal to diffuse cerebral edema and hemorrhages of various sizes from petechial to massive. Microscopically, there are necrosis of arterioles, minute cerebral infarcts, and hemorrhages. The term hypertensive encephalopathy

should be reserved for this syndrome and not for chronic recurrent headaches, dizziness, recurrent TIAs, or small strokes that often occur in association with high blood pressure.

Bleeding Diathesis:

A variety of coagulopathies can lead to bleeding into the brain substance, sometimes accompanied by systemic bleeding. Anticoagulation with heparin or warfarin reported most common cause for Intracerebral hemorrhage among bleeding diathesis[26,27]. Considering the large number of patients treated with anticoagulants, the number that develop ICH is small. The most consistent risk factor for intracranial or systemic bleeding was prolongation of the prothrombin time beyond the therapeutic range. As with other etiologies of ICH, hypertension aggravates the tendency to bleed intracranially. The three features that characterize anticoagulant-induced ICH as distinct from other causes are as follows:

i. Hemorrhage often develops gradually and insidiously during many hours, or even days[29-31].

ii. The cerebral lobes and cerebellum are common in hypertensive Intracerebral hemorrhage[29-31.]

iii. only patients with smaller hematomas (less than 30-cc volume) had a favorable chance for survival[29-31].

Anticoagulant-related ICH is a particularly difficult situation to treat because many patients take warfarin to prevent ischemic stroke. Patients with prosthetic heart valves, rheumatic mitral stenosis, or atrial fibrillation have a high risk for cerebral emboli without warfarin therapy. If a patient on anticoagulants develops neurologic symptoms, the cause is anticoagulant related hemorrhage until proven otherwise. If anticoagulant hemorrhage is verified, immediately give vitamin K, factor VIIa, or fresh frozen plasma. We must Pursue all measures aggressively to stop the bleeding. Although no formal prospective studies clarify the optimal time for restarting anticoagulants after ICH in patients who require long term treatment . The decision on if and when to restart anticoagulants depends on the risk of embolization from the donor source (atrial fibrillation, prosthetic heart valves, etc.) and the risk of further intracranial bleeding. When the indication for anticoagulation is relative or questionable, it is probably best to discontinue anticoagulants, perhaps using platelet anti aggregants instead[28].

Intracranial hemorrhages due to anticoagulant therapy usually lobar or subdural. Anticoagulant-related ICHs may evolve slowly, over 24–48 hours. Coagulopathy and thrombocytopenia should be reversed rapidly, as discussed below. ICH associated with hematologic disorders (leukemia, aplastic anemia, thrombocytopenic purpura) can occur at any site and may present as

multiple ICHs. Skin and mucous membrane bleeding is usually evident and offers a diagnostic clue.

Leukemia, hemophilia, thrombocytopenia, von Willebrand disease, and disseminated intravascular coagulation are other important causes of ICH, although it is unusual in these disorders for bleeding to be confined only to the brain.

ICH also occurs after recombinant tissue plasminogen activator infusion to treat occlusive cerebrovascular lesions. In this circumstance, hematomas usually begin within the region of brain infarction.

TUMORS

The first manifestation of neoplasm can be a Hemorrhage into a brain tumor[12]. Choriocarcinoma, malignant melanoma, renal cell carcinoma, and bronchogenic carcinoma are among the most common metastatic tumors associated with ICH. Glioblastoma multiforme in adults and medulloblastoma in children may also have areas of ICH.

Drugs

Perhaps best known are amphetamine[32] ("speed") hemorrhages. Hemorrhage often develops within a few minutes of drug use. The common symptoms are headache, confusion, and seizures[33] . Despite large volume

ICHs, few focal signs are present in such patients. This phenomenon is perhaps explained by the frequent coexistence of brain edema, infarcts, and a diffuse vasculopathy, in addition to the focal ICH. In some patients, acute hypertension follows amphetamine use and can potentiate ICH.

Amphetamine, methamphetamine, and dextroamphetamine were the most frequently used drugs[32,33].

Cocaine and methamphetamine are frequent causes of stroke in young (age <45 years) patients. ICH, ischemic stroke, and SAH are all associated with stimulant use. Angiographicfindings vary from completely normal arteries to large-vessel occlusion or stenosis, vasospasm, or changes consistent with vasculopathy. The mechanism of sympathomimetic-related stroke is not known, but cocaine enhances sympathetic activity causing acute, sometimes severe, hypertension, and this may lead to hemorrhage. Slightly more than one-half of stimulant-related intracranial hemorrhages are intracerebral, and the rest are subarachnoid. In cases of SAH, a saccular aneurysm is usually identified. Presumably, acute hypertension causes aneurysmal rupture.

A potent solid form of d-methamphetamine base that can be smoked is now peddled in the streets (known as ice). This form is more potent and rapid acting.The ice-amphetamine relation has the potential to prove similar to the

crack-cocaine-hydrochloride relationship in terms of complications and potency.

Angiography has often shown striking abnormalities in chronic amphetamine users and other patients with amphetamine-related ICH. The focal vascular abnormalities usually emphasize superficial cortical arterial branches and are often referred to as beading. Unfortunately, these arteriographic changes have often been falsely attributed to arteritis.

Amphetamines are known to be potent vasoconstrictors. Vasoconstriction can become chronic and produce chronic morphologic changes in the media of involved arteries. Segmental changes and beading in some amphetamine users is probably caused by pharmacologic effects of the drugs used, and does not represent a true arteritis. Since the early 1980s, cocaine has become a very important cause of stroke and drug related

Cocaine hydrochloride is usually snorted nasally. Some addicts use crack cocaine, a substance made by mixing aqueous cocaine hydrochloride with ammonia and sometimes baking soda. Crack cocaine is smoked or inhaled after the cocaine is mixed in the alkaline solution and is precipitated as alkaloidal cocaine. Cocaine hydrochloride can be taken in a variety of ways - orally, vaginally, rectally, sublingually, nasally, and by subcutaneous, intramuscular, or intravenous injection. Cardiovascular effects begin

immediately after use and consist of an increase in pulse, blood pressure, temperature, and metabolism.

Headache, focal neurologic signs, and sudden loss of consciousness may be a presenting symptom .Concurrent use of alcohol was common. ICH followed use of cocaine by any route.

The most common location of cocaine related ICH was lobar, other sites are caudate, thalamic, and putaminal hematomas. Of great interest and importance was the frequent presence of an underlying vascular lesion.

Cocaine-related ICH has a high mortality and high frequency of underlying aneurysms and AVMs. Clearly, cocaine-related intracranial bleeding is an indication for angiography, especially when the bleeding is subarachnoid or lobar. Underlying vascular lesions are less common when the ICH is deep. Most authors have attributed cocaine-related hemorrhage to the sympatheticomimetic effects of the drug Some patients have had a hypertensive encephalopathy with multiple ICHs and brain edema.

Another drug known to have sympatheticomimetic capabilities is phencyclidine[34], known as PCP or angel dust also a cause for ICH . Two other hallucinogens, lysergic acid diethylamide (known as LSD) and mescaline, are also known to raise blood pressure and cause vasoconstriction. More controversial is the issue of ICH after use of amphetamine-like drugs

that are mostly used as anorexic agents to lose weight, and were an ingredient in some cold remedies.

These drugs are usually sold over the counter as diet suppressants or stimulants. The most commonly cited agent is phenylpropanolamine[35] (PPA), which is often combined with an antihistamine and caffeine. PPA was used by individuals who developed ICH, but the numbers are relatively small, considering the frequency of use. PPA-related hemorrhages have been most often described after use for weight control and the dosage was often higher than suggested. PPA ingestion in the usual amounts elevates blood pressure by only a few millimeters.

In some individuals, there may be an idiosyncratic reaction with more severe blood pressure rises. There are very few cases of hemorrhage after use of cold remedies, but PPA has been removed from cold remedies in many countries.

A risk of ICH clearly exists for those who use PPA in a higher-than suggested dose. Prior hypertension; additional use of alcohol, coffee, or caffeine; concomitant use of monoamine oxidase inhibitors; and use during the postpartum period increase the risk of hemorrhage after PPA ingestion.

Talc, methylcellulose crystals, and cornstarch obliterate the lung arterioles, allowing the injected particles to reach the systemic circulation after intravenous use.

Occasional causes are after the use of phosphodiesterase inhibitors (sildenafil)[36a,c] [Viagra], vardenafil[36b] [Levitra], and tadalafil[36d] [Cialis] prescribed to enhance erectile function in men. It is not clear if the intracerebral hemorrhages in these patients was explained by increased blood pressure during sexual intercourse, or by the phosphodiesterase inhibitors alone, or a combination of the agents and circulatory changes accompanying sexual activity.

Cerebral Amyloid Angiopathy[37-49]

Awareness of CAA has led to wider use of special stains and recognition that an ever increasing percentage of ICH, especially in the elderly, is related to CAA. It affects small arteries and arterioles in the leptomeninges and cerebral cortex , stains positively with periodic acid-Schiff stains, has apple-green birefringence with polarized Congo red stain. Sometimes, the vessel wall seems to be reduplicated or split. CAA predominantly affects aged persons > 65 years. At necropsy, most patients have senile plaques, and many patients have been diagnosed clinically with Alzheimer's disease. Amyloid-laden arteries are most often found in the

occipital and parietal regions, less often in the other cerebral lobes; rarely, if ever, are these arteries found in the deep basal gray matter or brainstem. They are occasionally found in the cerebellum. Some patients have recurrent ICH or SAH in different lobar sites, a finding in an elderly person that is virtually diagnostic of CAA. At necropsy, small scattered cerebral infarcts and Alzheimer's related changes are found, along with evidence of old slit-like lobar hemorrhages. Echo planar MRI scans shows microbleeds[48].

It accounts for some intracranial hemorrhages associated with IV thrombolysis given for MI. This is the elderly disease , arteriolar degeneration with amyloid deposits in cerebral arterial walls occurs .This disorder can be suspected in patients who present with multiple hemorrhages (and infarcts) over several months or years, or in patients with "micro-bleeds" seen on brain MRI sequences sensitive for hemosiderin, but it is definitively diagnosed by pathologic demonstration of Congo red staining of amyloid in cerebral vessels.

The ε2 and ε4 allelic variations of the apolipoprotein E gene are associated with increased risk of recurrent lobar hemorrhage and may therefore be markers of amyloid angiopathy. Currently, there is no specific therapy, although antiplatelet and anticoagulating agents are typically avoided.

Some patients have a Binswanger-like picture, with chronic white matter gliosis and atrophy. Like anticoagulant related hemorrhages, CAA-related hemorrhages may develop insidiously. Perhaps because of coexisting atrophy, pressure symptoms, such as headache and vomiting, are less frequent than in younger patients with hypertensive or AVM related hemorrhages.

Trauma

Trauma is an important cause of intracerebral bleeding[50,51]. In some patients, a traumatic etiology is not clear from the history. Patients develop retrograde amnesia after the head injury, and patients have no recollection of a fall or other injury. A search for superficial head bruises or lacerations is worthwhile when the etiology of ICH is not obvious.

Head injury is the major cause intracranial bleeding. The common sites are intracerebral (mainly Temporal and inferior frontal lobes) and into the subarachnoid, subdural, and epidural spaces. Trauma must be considered in any patient with an unexplained acute neurologic deficit (hemiparesis, stupor, or confusion), particularly if the deficit occurred in the context of a fall.

Other Causes:

Rare patients with a mutation in a gene that encodes type IV collagen alpha 1 chain (COL4A1) have genetically influenced intracerebral and subdural hemorrhages that develop especially after trauma, dilated perivascular spaces and a diffuse leukoencephalopathy.

Primary intraventricular hemorrhage is rare. It usually begins within the substance of the brain and dissects into the ventricular system without leaving signs of intraparenchymal hemorrhage.

Alternatively, bleeding can arise from periependymal veins. Vasculitis, usually polyarteritis nodosa or lupus erythematosus, can produce hemorrhage in any region of the central nervous system; most hemorrhages are associated with hypertension, butthe arteritis itself may cause bleeding by disrupting the vessel wall.

Nearly one-half of patients with primary intraventricular hemorrhage have identifiable bleeding sources seen using conventional angiography. Sepsis can cause small petechial hemorrhages throughout the cerebral white matter. Moyamoya disease , mainly an occlusive arterial disease that causes ischemic symptoms, may on occasion produce intraparenchymal hemorrhage, particularly in the young. Hemorrhages into the spinal cord are usually the result of an AVM, cavernous malformation, or metastatic tumor. Epidural

spinal hemorrhage produces a rapidly evolving syndrome of spinal cord or nerve root compression. Spinal hemorrhages usually present with sudden back pain and some manifestation of myelopathy.

CLINICAL COURSE

In Intracerebral hemorrhage, the bleeding that occurs is because of arteriolar or capillary pressure from deep penetrating arteries hence it occurs gradually over minutes or sometimes hours.

Whereas in subarachnoid Hemorrhage the bleeding is due to systemic arterial pressure, hence Symptoms begin suddenly causing Severe headache (thunderclap), vomiting and altered conciousness. These symptoms are due to sudden hike in intracranial tension (ICP) , because of rapid flow of blood through the CSF surrounding the brain substance.

Hypertensive hemorrhage usually occurs without prior warning, mostly in awake patients and during work.

Headache may be present in Half of overall patients & it can be severe, nausea and vomiting are common.

Blood pressure is mostly elevated after the hemorrhage event, hence a normal blood pressure rules out intracerebral bleed.

Following hemorrhage, there is gradual increase in edema surrounding hemorrhage area. This produces worsening of clinical symptoms over a period from minutes to days. However the active bleeding duration is brief in most of intracerebral hemorrhage .

After stabilisation of deficit, improvement starts to occurs slowly. Since the deficit is principally caused by edema and hemorrhage, these compress the brain tissue rather than destructing, hence appreciable neurologic function returns. Visualizing the actual pathology of the disease and its gradual development helps to predict the tempo of the symptoms.

When Bleeding is directly over brain parenchyma, instead of CSF, patient does not have headache. Its because the brain parenchyma doesn't have pain fibers, so this initial release of blood wont cause symptoms whereas it contrast subarachnoid hemorrhage .

Rather, blood disturbs the function of nearby brain region in particular. For example If the hematoma starts in left putamen, the patient may have symptoms of right-limb paralysis . the paralysis becomes more severe during the next few minutes to hours as the hematoma enlarges . Sensory symptoms in form of numbness, aphasia (motor or sensory) , and conjugate deviation of eye ipsilateral side of the hemorrhage can occur. The patient may develop headache, vomiting, and altered sensorium when the hematoma expands thereby leading to increased Intracranial pressure and thereby distorting adjacent structures of meninges.

CLINICAL SYMPTOM

Headache

Headache is a rare symptom, was much more common in larger lesions in contrast with small lesions where headache is absent or very minimal. Headache Found to be common in lobar or parenchymal and cerebellar hematomas, locations near to meningeal surface and in patients with meningeal involvement. Headache is associated with vomiting and altered conciousness when hematoma enlarges .

Altered Level of Consciousness

Loss of consciousness produced only by large hematomas and brainstem hemorrhage. In intracerebral hemorrhage Decreased alertness is usually caused by midline shift , mass effect and Raised Intracranial pressure or direct involvement of the reticular activating system of brainstem . decreased level of consciousness indicates poor prognostic sign .

Vomiting

Vomiting is also one of the important symptom in intracerebral hemorrhage. Vomiting is due to Raised Intracranial pressure or fourth ventricle distortion. Vomiting is more in patients with intracerebral hemorrhage when compared to ischemic infarcts. In posterior circulation

lesion, dysfunction of vestibular nuclei, or vomiting center near the floor of the fourth ventricle are the major causes for vomiting .It occurs in one third of posterior circulation infarcts compared to posterior circulation hemorrhages where vomiting is present in one half of patients. In cerebellar hemorrhages vomiting in their early clinical course is the rule.

Seizures

Seizures are more common in ICH .In comparison to various stroke types, one exceptional is embolism. Lobar hemorrhages, small slit like hemorrhages near the cortical gray-white junction and hemorrhage in basal ganglia adjacent to cortex are the conditions epileptogenic. continuous EEG monitoring helps in detecting causes for seizures .

Other Symptoms and Signs

Neck stiffness is commonly associated with cerebellar hemorrhages and occasionally with caudate and Thalamic hemorrhage. Fever is common, may be due to infections such as pulmonary and catheter related urinary tract infections. Subhyaloid retinal hemorrhages visualised through fundus examination rare in intracerebral hemorrhage compared to subarachnoid hemorrhage . Cardiac arrhythmias and non cardiogenic pulmonary edema may occur in intracerebral hemorrhage its because of changes in intracranial pressure and sudden catecholamine release.

PUTAMEN HEMORRHAGE:

Just as there are physicians who believe that the introduction of chest x-rays made the stethoscope obsolete, some doctors believe that detailed knowledge of the findings on neurologic examination of patients with central nervous system lesions is no longer necessary since the advent of CT and MRI scans. Because hemorrhages are so well imaged by CT, why bother to learn the physical findings? In the future, physicians will probably not have an inexpensive pocket or portable CT or MRI to replace the examination of patients. Prognosis and treatment of ICH often depend on the location of the hematoma . The locations such as cerebral lobes, right putamen, and cerebellum are accessible to surgical intervention, whereas thalami and brainstem are not surgically approachable ones.

Clinical distinction between ICH and superficial cerebral infarction caused by large vessel occlusive disease or cerebral embolism depends on localization of the lesion to deep (ICH) or superficial (infarct) location. Knowledge of findings in patients with hemorrhages at various locations teaches clinicians to search for tumors, abscesses, demyelinating lesions, and other disorders in the same locations in other patients. Historically, hemorrhages in the cerebellum, thalami, and caudate nucleus were recognized before cerebellar, thalamic, or caudate infarction. Awareness of

the clinical syndromes associated with ICH in these locations made it possible to later recognize infarcts in these regions.

Keys to localization of ICH follow:

i. Motor signs-quadriparesis, hemiparesis, or no paresis

ii. Pupillary function-asymmetry, size, and light reaction

iii. Extraocular movements-supranuclear, nuclear, internuclear gaze palsies

iv. Gait abnormalities, especially ataxia

The putaminal hemorrhage[52] often begin in the putamen. The usual findings include contralateral hemiparesis, contralateral hemisensory loss, and conjugate deviation of the eyes toward the side of the hematoma. The pupils are generally normal and gait is hemiparetic. Patients with a left putaminal hemorrhage[55] usually have a nonfluent aphasia with relative preservation of ability to repeat spoken language. Right-sided lesions are associated with left visual neglect, motor impersistence, and constructional dyspraxia. These abnormalities of higher cortical function are probably caused by disconnection and undercutting of cortical zones, and are usually more transient than in patients with cortical infarcts of equal size. Some patients develop ipsilateral adventitious movements that the family or

observers call “tremor”; these movements are probably caused by involvement of ipsilateral descending projections of the extrapyramidal system.

The Loci of putaminal hemorrhages[55]:

(1) Anterior type involving anterior putamen and anterior limb of the internal capsule,

(2) Middle type involving the capsular genu and the globus pallidus and middle portion of the putamen,

(3) Posterior type involving the far posterior limb of the internal capsule and often affecting the optic radiations and spreading into the temporal lobe isthmus.

In patients with large putaminal hemorrhages, stupor increases as the lesion enlarges; the ipsilateral pupil at first becomes smaller, and later, larger than the opposite pupil; the ipsilateral plantar response becomes extensor; and a bilateral horizontal gaze palsy develops. The presence of sign ipsilateral Babinski's sign, abnormal pupil, or gaze paresis indicates poor prognosis. These additional findings are caused by midline shift or compression of the rostral brainstem by the expanding hematoma.

The location medial and most anterior portions of the posterior putamen, and the anterior two thirds of the posterior limb of the internal capsule is the most common site for putaminal hemorrhage[55] because it is supplied by the largest of the lateral lenticulostriate arteries.

When hematomas are in the posterior third of the internal capsule and far posterior extreme of the putamen, sensory abnormalities predominate, with little or no hemiparesis. An inferior quadrantanopia or hemianopia may be present. Patients with lesions in the far posterior left putamen may have fluent Wernicke-like aphasia because of undercutting of the temporal lobe or extension of the lesion into the temporal isthmus, giving the hematoma a hockey stick-like configuration Lateral ganglionic hemorrhages occur in the distribution of the various medial and lateral lenticulostriate arteries .

When the hematoma occupies CT sections containing the bodies of the lateral ventricles, then the middle type of hematoma is usually present and

hemiplegia is likely to persist. The multiple planes shown in MRI imaging make it easier to visualize the location, spread, and size of hematomas. T2*-weighted (susceptibility) images[58] show hemorrhages best.

Cerebral angiography may be helpful in studying patients with putaminal hemorrhage. Anterior lesions affect same side frontal lobe function and posterior lesions affect the temporal and parietal lobes.

Caudate Hemorrhage

Hematomas at this site can lead to pass blood quickly into the adjacent lateral ventricle, or the internal capsule or nearby hypothalamus[59]. The larger hematomas causes opposite side hemiparesis, same side conjugate eye deviation, contralateral conjugate gaze palsy, and an ipsilateral horners syndrome. Sensory findings are usually absent or minimal.

The usual cause of caudate hemorrhage[59,60] is hypertension, but AVMs are also common, especially in the young. Caudate hematomas have a better prognosis than comparable-sized putaminal hemorrhages.

Symptoms and signs of caudate hemorrhage closely mimic SAH, but the CT appearance of blood in the caudate and lateral ventricles is distinctive.

Thalamic Hemorrhage

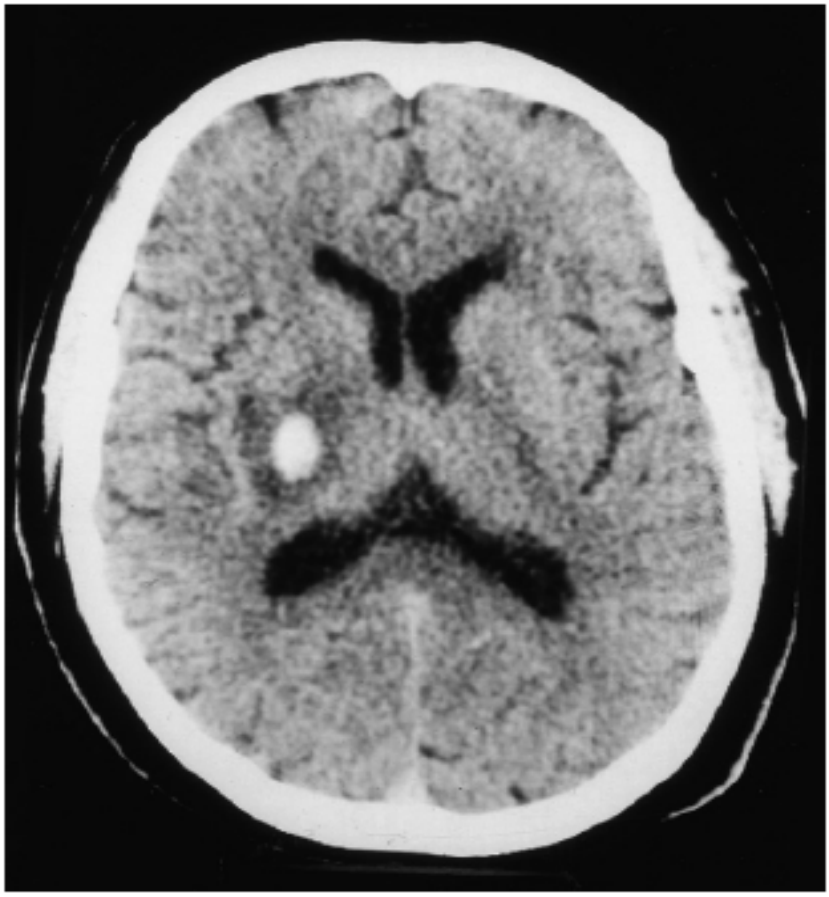
The hemorrhages occur in ventrolateral & posteromedial portions of thalamus corresponding to thalamogeniculate and thalamic-subthalamic

arteries. Other located anteriorly the tuberothalamic (polar) artery territory and lateral posterior choroidal artery territory .

Most thalamic hematomas are posterior to the pyramidal-tract fibers in the internal capsule, so that contralateral sensory abnormalities are usually more prominent than contralateral hemiparesis[62-68].

Some large thalamic hematomas dissect laterally and rostrally and involve the anterior portion of the posterior limb of the internal capsule, causing a hemiplegia. Sometimes, limbs contralateral to hematomas are slightly ataxic or have choreic movements. The contralateral hand may rest in a fisted or dystonic posture. The key neurologic findings that separate thalamic from caudate or putaminal hemorrhages are the eye signs. Patients with caudate or putaminal hemorrhages have conjugate deviation of the eyes toward the side of the lesion and paresis of conjugate gaze to the opposite side.

CT BRAIN showing Thalamic Hemorrhage



The characteristic oculomotor abnormalities in patients with thalamic hematomas are as follows:

i. Paralysis of upward gaze

ii. Hyperconvergence of eyes, with a combination of these findings giving patients the appearance of peering downward and inward at the tops of their noses.

iii. Ocular skewing, in which one eye rests below the other, with this divergence in vertical eye position remaining constant in gaze in all directions.

iv. Eyes gazing

v. Disconjugate gaze, pseudo sixth nerve paresis these oculomotor abnormalities are caused by direct extension of thalamic hematoma to mesencephalic - diencephalic junction and quadrigeminal plate region compression by the thalamic hematoma. In thalamic hemorrhage, the pupils are usually small and react poorly to light because of interruption of the afferent limb of the pupillary reflex arc.

After beginning a conversation almost normally, patients may lapse into a remarkable fluent aphasia, with many jargon or nonexistent words and poor communication of ideas. In contrast to patients with Wernicke's aphasia, comprehension of spoken language is good. Patients with thalamic ICH may repeat and duplicate words or syllables at the ends of words in spoken and written language. Paraphasic errors and poor naming are also common. Decreased levels of alertness and hypersomnolence are common at the onset of thalamic hemorrhage because of involvement of the rostral reticular activating system. The prognosis for recovery from thalamic hemorrhages is not as good as caudate or putaminal hemorrhages of comparable size, but coma is not as dire a prognostic sign in thalamic lesions as it is in other supratentorial sites. Also, unlike putaminal hemorrhage, the severity of the deficit and mortality do not correlate with ventricular

extension in patients with thalamic haemorrhage. Thalamic hemorrhages are not accessible surgically unless they extend far laterally.

Posterolateral thalamic hematomas in the territory of the thalamogeniculate arteries are the most common and largest type of thalamic hematomas. These lesions often spill out of the thalamus laterally and may cause motor paralysis by involving the internal capsule Anterior or anterolateral thalamic hematomas are in the distribution of the tuberothalamic (polar) artery, cause apathy and abulia. Posteromedial hematomas are in the distribution of the thalamic-subthalamic thalamo perforating arteries; abnormalities of consciousness, pupillary function, and vertical gaze predominate. These hematoma gradually seep into third ventricle, thereby obstructing the third ventricle leading to obstructive hydrocephalus. Oculomotor abnormalities improve after intraventricular shunt procedure. The posterior and dorsal lesions in pulvinar region of posterior choroidal arteries, transient sensorimotor signs aphasia and behavioral abnormalities results.

Lobar Hemorrhages

ICH may develop beneath the region of the graywhite junction of the cerebral cortex. These subcortical hemorrhages usually spread in a linear direction along white matter pathways. When the hematomas absorb, linear cavities remain, giving the lesions the name “slit hemorrhage”[74-76]

Subcortical hemorrhages are important to diagnose because the symptoms and signs are often erroneously attributed to cerebral infarction. Inappropriate therapy might be prescribed unless brain imaging shows the hematomas. Also, if subcortical hemorrhages are large, they are relatively superficial and are more accessible to surgical drainage than deeper hematomas. In the past, subcortical hemorrhages were rarely diagnosed antemortem, but CT and MRI greatly enhance recognition of these lesions. Many lobar hemorrhages are caused by AVMs, cavernous angiomas, and amyloid angiopathy, each of which has a predilection for cortical and subcortical regions.

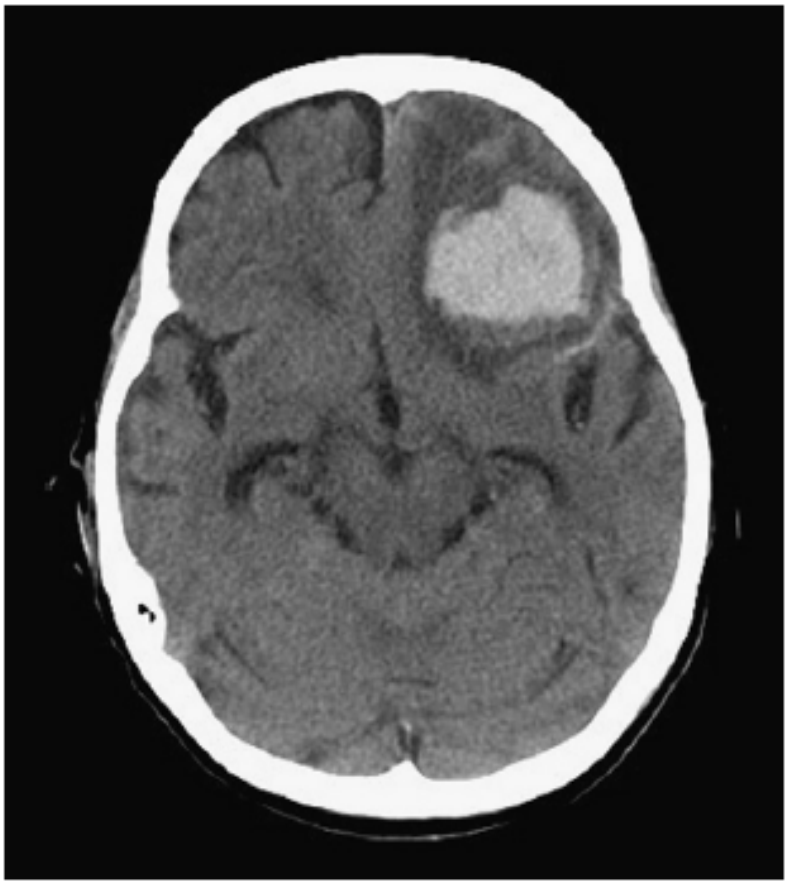
Symptoms and signs appear over several minutes. Most lobar hemorrhages are small and cause a restricted clinical syndrome that simulates an embolus to an artery supplying one lobe. For example, the major neurologic deficit with an occipital hemorrhage is hemianopia; with a left temporal hemorrhage, aphasia and delirium; with a parietal hemorrhage, hemisensory loss.

Most patients with lobar hemorrhages have focal headaches, and more than one-half vomit or are drowsy. Stiff neck and seizures are uncommon.

Hypertension is also an important cause of lobar hematomas. The parietal and occipital lobes are affected more often than the frontal and parietal regions.

i. Frontal hematomas: Far anterior lesions usually cause abulia. Patients appear apathetic and have reduced spontaneity, prolonged latency in responding, and short, terse replies. If the lesions extend deeply or toward the precentral gyrus, conjugate eye deviation toward the side of the hematoma and contralateral hemiparesis are found.

CT BRAIN showing Left Frontal Hemorrhage



ii. Paracentral hematomas: Lesions near the central sulcus produce contralateral motor and sensory signs, sometimes with aphasia if the lesion is in the left hemisphere.

iii. Parietal hemorrhages: Parietal hemorrhages are usually accompanied by contralateral hemisensory loss, with neglect of the contralateral visual field. The limbs contralateral to the hemorrhage are often uncoordinated.

Aphasia and disorders of reading, writing, and arithmetic functions are present when the lesions involve the left inferior parietal lobule. Patients with right inferior parietal hematomas have defective drawing and copying and may have difficulty with visualspatial functions.

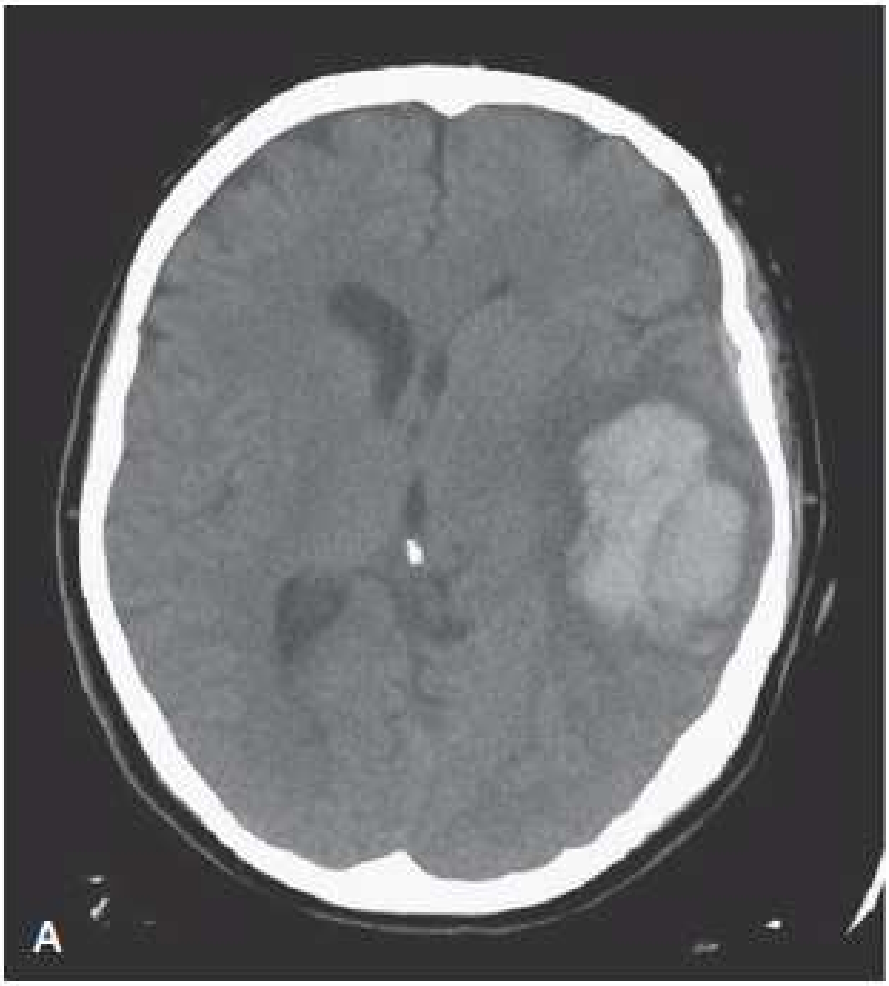
iv. Occipital hematomas: Occipital hemorrhages cause a severe contralateral hemianopia, often with slight contralateral hemisensory or motor signs and visual neglect.

v. Temporal-lobe lesions: Temporal-lobe lesions often cause agitation and delirium. Wernicke type aphasia accompanies left temporal lesions. Temporal-lobe hematomas are particularly likely to swell and may cause herniation without preceding hemiparesis. Brainstem compression may develop insidiously, with deepening stupor. An ipsilaterally dilated pupil follows.

The functional outcome in patients with lobar ICH is also generally better than other forms of ICH. The exception to good outcome is the occurrence of lobar hemorrhage in patients taking anticoagulants. Anticoagulant hemorrhages have a predilection for the cerebral lobes and the

cerebellum, and often gradually increase in size. The diagnosis of lobar hemorrhage is often quite difficult without CT or MRI.

CT BRAIN showing Left Temporal Lobar haemorrhage



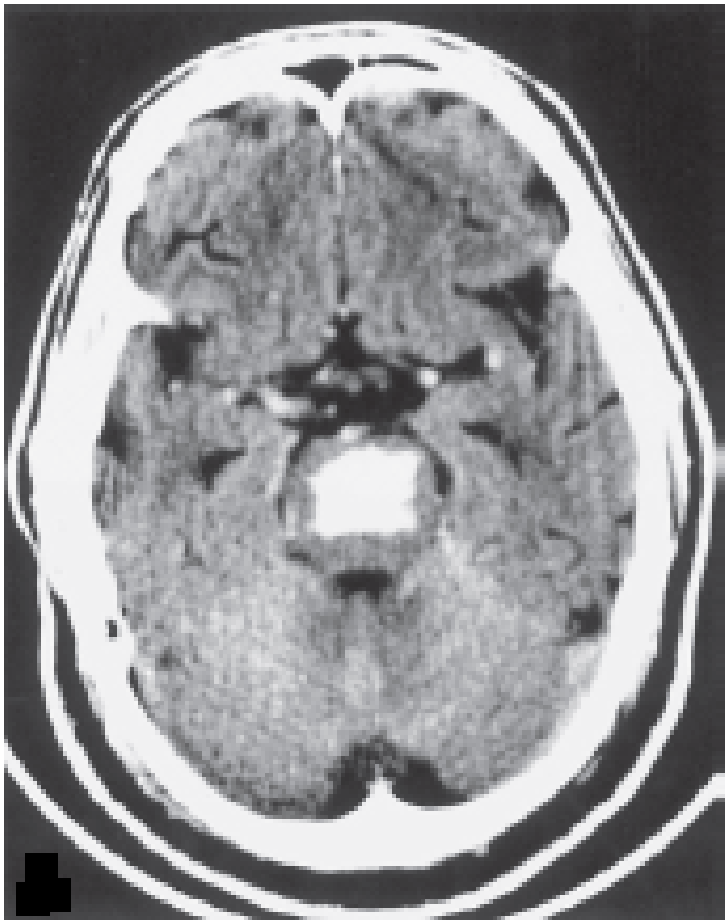
Primary Intraventricular Hemorrhages - Occurs usually in parenchymal Hemorrhages that develop close to ventricular system. Caudate and thalamic hemorrhages are the most common sites for direct ventricular

Drainage, Ventricular bleeding usually arises from small subependymal AVMs or cavernous angiomas or from hemorrhage into the caudate nucleus or thalamus just adjacent to the ventricles. Primary intraventricular

hemorrhage is especially common in premature newborns in whom bleeding arises from the germinal matrix adjacent to the cerebral ventricles. The clinical syndrome closely mimics SAH, with sudden headache, stiff neck, vomiting, and lethargy. At times, bilateral, usually symmetric hyper-reflexia and extensor plantar responses occur. Also at times, the bleeding is primarily into one lateral ventricle, and asymmetric focal signs may predominate. Decreased consciousness is almost an invariable sign. CT shows blood distending the lateral ventricles and third ventricle and some blood density within the subarachnoid space. In childhood, the most common cause is an AVM, which can destroy itself as it ruptures. In adults Hypertension is the common cause for Intraventricular hemorrhage[77-81] .

Pontine Hemorrhage

CT BRAIN showing Pontine Hemorrhage



Primary brainstem hemorrhages most common in pons. The Midbrain and medullary hemorrhages are extremely rare and if at all occurs is due to bleeding diathesis. Primary pontine hemorrhages[85-100] usually begin in the center of the pons at the tegmental-basal junction. Blood may dissect rostrally into the midbrain, but rarely extends caudally into the medulla. Hematomas frequently dissect into the fourth ventricle.

Signs (i) UMN quadriparesis (ii) coma; (iii) absent Dolls eye movements; (iv) pinpoint pupils; and (v) rapid or irregular varying respirations.

Headache and vomiting occasionally occur.

A hemiparesis is common early in the course. Deafness, dysarthria, facial numbness, asymmetric facial or limb weakness, and dizziness occasionally precede the development of coma. Some patients have twitching, shivering, or spasmodic movements of the limbs, usually culminating in decerebrate rigidity. These adventitious movements are often misinterpreted as convulsive seizures. Vertical refl ex eye movements are preserved unless the lesion extends rostrally into the midbrain. Very large pontine hemorrhages are invariably fatal, but not usually instantaneously. Death usually occurs 24 to 48 hours after onset. Survival for 7 to 10 days, however, is not rare. Some patients with large medial pontine hematomas survive with quadriplegia. Hyperthermia is sometimes noted.

Lateral basal lesions can spread into the adjacent lateral tegmentum, causing unilateral cranial nerve signs and contralateral hemiparesis.

Lateral tegmental hematomas arise from penetrating vessels that course from lateral to medial after branching from the lateral circumferential pontine arteries.

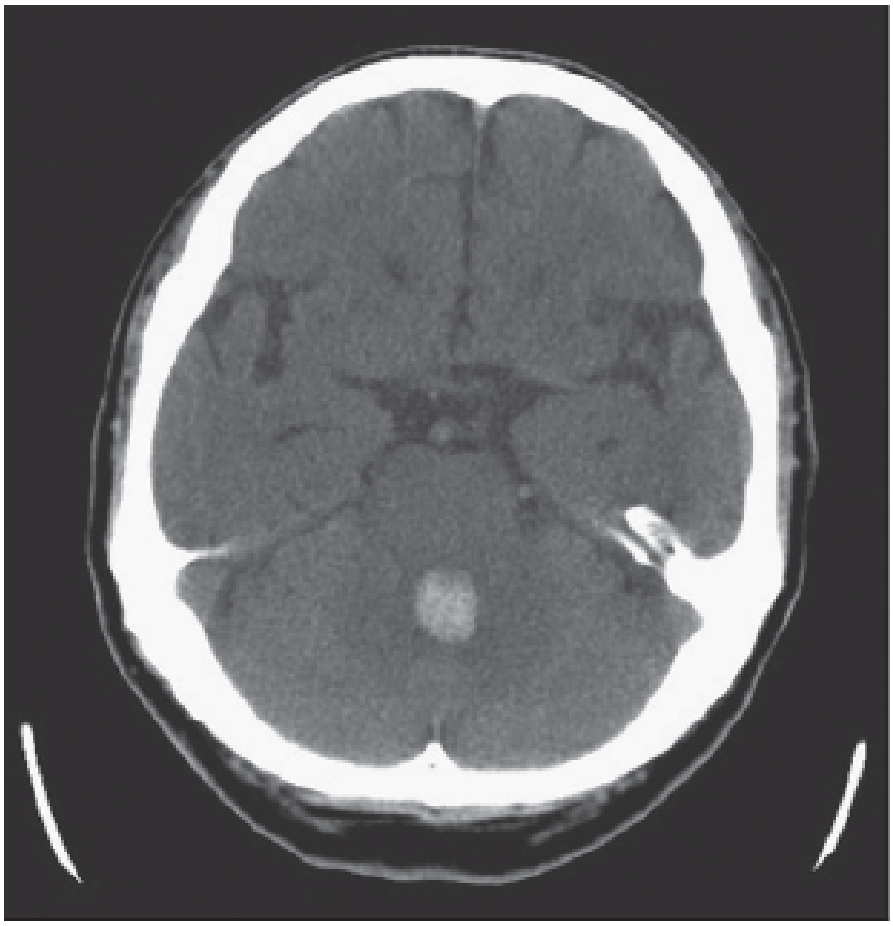
These lesions involve the rostral pons. Findings on neurologic examination are those of a predominantly unilateral tegmental lesion. Most distinctive and diagnostic of lateral tegmental pontine hematomas are the oculomotor abnormalities, which include ipsilateral conjugate gaze paresis, ipsilateral internuclear ophthalmoplegia or a combination of ipsilateral internuclear ophthalmoplegia and gaze palsy (a “one and one-half syndrome”), in which the only preserved eye motion is abduction of the contralateral eye. Because the sensory lemniscus (joining of the medial lemniscus and spinothalamic tracts) is lateral tegmental, accompanying loss of pinprick, temperature, and position sense on the opposite side of the body is common. Limb and truncal ataxia are usually present and may be bilateral or predominantly ipsilateral. Unilateral facial numbness or weakness, ipsilateral miosis, and transient deafness may also be present. When contralateral hemiparesis occurs, it is usually slight and transient. Patients with small pontine hematomas generally survive with slight to moderate clinical neurologic deficits.

Cerebellar Hemorrhages

Hemorrhage into the cerebellum probably accounts for approximately 10% of ICH, approximating the relative percentage of weight of the cerebellum in reference to the entire brain. Anticoagulant usage and bleeding diatheses account for a disproportionate percentage of cases of cerebellar

hemorrhage. Although the frequency of cerebellar hemorrhage is low, establishing the diagnosis is important as some hematomas can be surgically treatable and hence the prognosis is improved . Cerebellar hemorrhage usually originates in dentate nucleus, arising from the posterior inferior cerebellar, artery and superior cerebellar artery. [102-109]

CT BRAIN showing Vermal Cerebellar Hemorrhage



Hematomas collect around the dentate and spread into the cerebellar hemispheral white matter, frequently extending into the fourth ventricle. The adjacent brainstem is seldom directly involved, but is compressed from above by the lesion. Occasional cerebellar hemorrhages arise in the vermis in

medial branches of the posterior inferior cerebellar artery or the superior cerebellar artery.

Some patients even have difficulty remaining in a sitting or standing position, often leaning or tilting toward the side of the hematoma.

Patients have been known to crawl, slide, or bump on their bottom to get to the bathroom or telephone. Vomiting, Headache is also common, usually affecting the occiput, neck, or frontal region. Dysarthria, hiccups, and tinnitus occur, but are less frequent patient can anytime have his consiousness lost.

Neurologic signs are (i) ipsilateral abducen nerve palsy or gaze palsy (ii) small pinpoint pupils (iii) gait ataxia. Hemiparesis rarely occurs in patients with cerebellar hemorrhage, but cerebellar lesions do produce an apparent asthenia or slowness of the affected limbs. Inferior extremity reflexes are usually symmetrically exaggerated, but plantar responses are flexor. Knee jerks are typically pendular with an increased span of leg movement.

Classic cerebellar-type incoordination of the arm on finger-to-nose or toe-to-object testing and frank intention tremor are uncommon. Patients with cerebellar infarction and hemorrhage, the single most useful cerebellar sign is elicited when the patient is asked to raise both arms together rapidly, then to

brake the ascent quickly. Next, the patient is directed to drop the arms quickly, again braking the descent before the hands hit the bed or table. The arm on the side of the cerebellar lesion lags behind the other arm and overshoots the endpoint. Patients with large cerebellar hematomas often have brainstem compression. They develop increasing stupor, lateral gaze palsy toward the side of the hematoma, and bilateral extensor plantar responses. Untreated patients with cerebellar hemorrhage who become comatose invariably die of brainstem compression. CT and MRI not only document the size, locale, and position of the hematoma, but also give considerable information about posterior-fossa pressure. An expanding lesion obliterates the cerebellopontine angle and ambient cisterns, and displaces the fourth ventricle toward the opposite side. Usually, the fourth ventricle compression leads to hydrocephalus, with early dilatation of the temporal horns of the lateral ventricles.Occasionally, patients with cerebellar hemorrhage have a more indolent course, presenting with symptoms and signs of hydrocephalus. Abulia, dementia, slow-stepped shuffling gait, and incontinence are the characteristic signs of hydrocephalus.

The patient and family may fail to emphasize the preceding symptoms of dizziness, headache, and vomiting that had been interpreted as influenza or other viral illness. Other patients have laterally placed cerebellar hematomas that compress the cerebellopontine angle structures.

These patients develop dysfunction of the fifth, sixth, seventh, and eighth cranial nerves, in addition to ataxia. These large medially placed vermal hemorrhages quickly compress the fourth ventricle and create pressure on the bilateral pontine tegmentum. At times, vermal hemorrhages are smaller and present with dizziness and gait ataxia.

The disease course in unpredictable, so its better to for surgical procedure in hematoma of size > 3 cm and Prognosis mostly depends hematoma size and the Glasgow coma scale score before treatment . The outlook for patients with small cerebellar hematomas is excellent.

Patients should have routine blood chemistries and hematologic studies. Specific attention to the platelet count and PT/PTT are important to identify coagulopathy. CT imaging reliably detects acute focal hemorrhages in the supratentorial space. Small pontine hemorrhages may not be identified because of motion and boneinduced artifact that obscure structures in the posterior fossa.

After the first 2 weeks, x-ray attenuation values of clotted blood diminish until they become isodense with surrounding brain. Mass effect and edema may remain. In some cases, a surrounding rim of contrast enhancement appears after 2–4 weeks and may persist for months. MRI, although more sensitive for delineating posterior fossa lesions, is generally

not necessary in most instances. Images of flowing blood on MRI scan may identify AVMs as the cause of the hemorrhage. MRI, CT angiography, and conventional x-ray angiography are used when the cause of intracranial hemorrhage is uncertain, particularly if the patient is young or not hypertensive and the hematoma is not in one of the four usual sites for hypertensive hemorrhage. Postcontrast CT imaging may reveal acute hematoma enhancement signifying bleeding at the time of imaging; this “dot-sign” portends increased mortality. Some centers routinely perform CT and CT angiography with postcontrast Hypertensive hemorrhage. Transaxial noncontrast CT scan through the region of the basal ganglia reveals a hematoma involving the left putamen in a patient with rapidly progressive onset of right hemiparesis. CT imaging in one sitting to rapidly identify any macrovascular etiology of the hemorrhage and provide prognostic information at the same time. Since patients typically have focal neurologic signs and obtundation, and often show signs of increased ICP, a lumbar puncture should be avoided as it may induce cerebral herniation.

DIAGNOSIS – PROGNOSIS - AND – TREATMENT

Diagnosis

Accurate bedside diagnosis of ICH rests on the presence of an appropriate ecologic background, such as hypertension or bleeding diathesis; the nonfluctuating, usually gradually progressive course over minutes or hours; accompanying symptoms, such as headache and vomiting; and neurologic signs compatible with a deep lesion.

With Both CT and MRI scans Hematoma age can be found. Hematomas are at initially regular and smooth. During initial 48 hours, large hematomas are like fluid blood levels, suggesting they are liquid not solidified . During the next 72 hours and above, surrounding edema with mass effect can be seen.

From 3 to 20 days small dense area with peripheral border contrast enhancement is visualised. Decrease in mass effect and the surroundind edema. The absorption coefficient of the hematoma decreases gradually, and the lesion develops a lucent appearance, with absorption characteristics resembling edema fluid or CSF. By 9 weeks, slight hypodensity remains, the mass effect and enhancement disaapears.

On MRI scans, the zone of altered attenuation or abnormal metabolism is usually much larger than the hypodensity seen on CT. Acute hematomas are isointense T1-weighted image and bright & hyperintense on T2-weighted images. Chronic hematomas are bright on T2-weighted images.

Angiography is generally unnecessary unless the lesion is in an unusual locus or the patient has no risk factors for hemorrhage, such as hypertension or bleeding diathesis. Catheter subtraction angiography is used to show AVMs or aneurysms that might have caused ICH. Angiography can also suggest the likelihood of hematoma enlargement.

During angiography Extravasation of contrast Occurs in enlarging hematoma and Indicates poor outcome. MR and CT angiography can be used in a similar fashion. Extravasation of contrast predicts subsequent enlargement of a hematoma.

Prognosis

The three most important predictors of outcome after ICH are haemorrhage size, bleeding location, and conscious level of the patient at admission. Hemorrhage expansion also indicates a worse prognosis when the hematoma attains a large size. Size and locale of the lesion on brain imaging scans renders very useful prognostic information. High systolic, mean blood

pressure, and pulse pressure correlate with poor outcome. Patients with hydrocephalus in supratentorial hemorrhages is a poor prognostic sign.

During the acute phase of ICH, the mass effect of the developing hematoma presents a much greater risk of death than does a comparable-sized brain infarct. In the case of ICH, something extra (blood) has been added to the intracranial contents. In brain infarction, the already existing contents (brain tissue) are ischemic, but an acute mass has not been added. Later, infarcts and hematomas become edematous, increasing ICP. In the chronic phase, if the patient with ICH has survived, the prognosis for recovery is actually much better than brain infarcts of similar size and location. Hematomas have dissected and separated the cerebral cortex and other brain parts, but usually the surrounding cortex is preserved. In contrast, infarcts leave dead, nonfunctioning cortex when they heal. Unlike SAH, recurrence of ICH during the acute illness is rare. These simple facts dictate the approach to ICH treatment-that is, aggressively try to limit the expanding hematoma to prevent death and late morbidity. In patients with ICH, the concern is control of acute mass effect, whereas in SAH, the goal is to prevent rebleeding and arterial vasoconstriction.

Treatment Careful medical management of patients with ICH may be lifesaving and is important, even in those patients who later have surgical drainage of their hematomas. Control of systemic blood pressure helps stop

intracranial bleeding, but must be done cautiously. In some patients with ICH, systemic blood pressure is further increased to ensure adequate perfusion of the brain. Increased ICP causes increased venous pressure, so elevated arterial pressure is needed to overcome the increased venous pressure to perfuse the tissues. Overzealous lowering of blood pressure can lead to underperfusion and clinical deterioration. Blood pressure should be lowered quickly, but not to hypotensive levels. Patients must be watched carefully during the treatment. American Heart Association recommends maintaining the mean arterial pressure below 130 mm Hg in all intracerebral hemorrhage with corticosteroids, mannitol, hypertonic saline, or glycerol called as medical decompression. Because edema develops around a hematoma and adds to mass effect, reduction of the surrounding edema is an important therapeutic goal. The perihematomal edema volume peaks during the third or fourth day after bleeding.

Other treatment to control intracranial pressure are Elevation of head end, hyperventilation, temperature control, and ventricular drainage.

Concern exists that hypertonic agents could diffuse into the ICH and cause a secondary increase in volume of the hematoma because of ingress of fluid.

When considering surgery and other therapies, hematomas, in practice, can be divided into the following three main groups:

i. Massive, rapidly developing lesions that effectively kill or devastate patients before they reach the hospital. For these lesions, little can or should be done.

ii. Small hematomas, from which the patient will make an excellent spontaneous recovery.

Treatment consists of controlling the etiologic factors, such as hypertension, to prevent recurrences.

iii. Medium-sized hemorrhages (hematoma volumes between the two extremes) with developing mass effect after the patient reaches the hospital. Within this third group, medical measures and surgery are most helpful.

Timing

Unfortunately the present CT and MRI technologies do not reliably show the liquidity of hematomas unless there is a fluid level within the lesion. Clinicians posited that very early surgery, within 4 hours after symptom onset, might allow drainage of liquid blood and lead to better outcomes than surgery after 12 hours.

Clearly, too early surgery can promote rebleeding, which adversely affected outcome. The ideal time to operate is unknown

The factors to be considered are whether patient is improving, stable, or worsening. In patients with putaminal hemorrhage, poor prognostic signs include presence of ipsilateral pupillary dilation, ipsilateral babinski response, ipsilateral conjugate gaze paresis. These signs are indicative of midline shift or early brainstem compression. Bilateral extensor plantar responses In patients with cerebellar hemorrhage, is a adverse prognostic sign. In deteriorating patients with accessible lesions, surgery intervension to be done as early as possible . Decreased consciousness (Glasgow Coma Scale score of − 14) was the most important single predictor of deterioration. Large hematoma volume (60mL), midline shift, effacement of the contralateral perimesencephalic and ambient cisterns, and dilatation of the contralateral temporal horn of the lateral ventricle were the other predictive features. Patients who deteriorate during the first 12 hours usually have enlargement of hematoma on follow-up CT scans. Those that deteriorate after the first day usually do so because of brain edema around the hematoma.

TREATMENT

ACUTE MANAGEMENT

Nearly 50% of patients with a hypertensive ICH die, but others have a good to complete recovery if they survive the initial hemorrhage. The ICH scoring system is a validated metric that is useful for prediction of mortality and clinical outcomes. Any identified coagulopathy should be reversed as soon as possible. For patients taking VKAs, rapid reversal of coagulopathy can be achieved by infusing prothrombin complex concentrates which can be administered quickly, followed by fresh-frozen plasma and vitamin K[12]. When ICH is associated with thrombocytopenia (platelet count <50,000/μL), transfusion of fresh platelets is indicated. The role of urgent platelet inhibition assays in the decision to transfuse platelets remains unclear. A phase 3 trial of treatment with recombinant factor VIIa reduced hematoma expansion[12]; however, clinical outcomes were not improved, so use of this drug cannot be advocated at present.

Evacuation of supratentorial hematomas does not appear to improve outcome. Overall, these data do not support routine surgical evacuation of supratentorial hemorrhages; however, many centers operate on patients with progressive neurologic deterioration. Surgical techniques continue to evolve,

and minimally invasive endoscopic hematoma evacuation may prove beneficial in future trials.

For cerebellar hemorrhages, a neurosurgeon should be consulted immediately to assist with the evaluation; most cerebellar hematomas >3 cm in diameter will require surgical evacuation. If the patient is alert without focal brainstem signs and if the hematoma is <1 cm in diameter, surgical removal is usually unnecessary. Patients with hematomas between 1 and 3 cm require careful observation for signs of impaired consciousness and precipitous respiratory failure.

Tissue surrounding hematomas is displaced and compressed but not necessarily infarcted. Hence, in survivors, major improvement commonly occurs as the hematoma is reabsorbed and the adjacent tissue regains its function. Careful management of the patient during the acute phase of the hemorrhage can lead to considerable recovery.

ICP can even be normal in large intraparenchymal hemorrhages. When hematoma does marked midline shift or hydrocephalus, osmotic agents with induced hyperventilation can be the ideal treatment to lower ICP until a ventriculostomy is done or ICP monitor is placed . Once ICP is recorded, further hyperventilation and osmotic therapy can be tailored to the individual patient to keep cerebral perfusion pressure (MAP-ICP) above 60 mm Hg. For

example, if ICP is found to be high, CSF can be drained from the ventricular space and osmotic therapy continued; persistent or progressive elevation in ICP may prompt surgical evacuation of the clot or withdrawal of support. Alternately, if ICP is normal or only mildly elevated, induced hyperventilation can be reversed and osmotic therapy tapered. Since hyperventilation may actually produce ischemia by cerebral vasoconstriction, induced hyperventilation should be limited to acute resuscitation of the patient with presumptive high ICP and eliminated once other treatments (osmotic therapy or surgical treatments) have been instituted. Glucocorticoids are not helpful for the edema from intracerebral hematoma.

Stereotactic techniques have been used more often in Asian countries than in the west. Drainage is performed through a small burr hole and no cortisectomy is involved. Stereotactic surgery has been performed with and without a stereotactic frame and with and without administration of a thrombolytic agent directly into the intracerebral clot. The results show promise and are likely to prove superior in the hands of experienced surgeons to direct surgical drainage. Drainage of subacute hemorrhages, by reducing intracranial pressure can improve.

I believe that in the foreseeable future, more aggressive drainage of intracerebral and intraventricular hematomas using advanced stereotactic

and endoscopic techniques and thrombolytic agents to liquefy clots will result in improved outcomes for patients with intracerebral hematomas.

Although there still may be a group of patients with intracerebral hemorrhages that can benefit from rFVIIa [111-115], the conclusion of these trials is that reduction in volume of hematoma, in general, does not exert a major effect on outcome.

Much must be learned regarding therapy for patients with ICH. The technological revolution has made diagnosis easy. More well-designed studies of different modes of treatment in patients with lesions of various etiologies, sizes, locations, and varying levels of consciousness are needed.

PREVENTION

Being, Hypertension a leading cause of primary spontaneous ICH. Prevention is by reducing hypertension, stopping alcohol use, and discontinuing illicit drug (cocaine and amphetamines) abuse . Patients with amyloid angiopathy should avoid antithrombotic agents.

MATERIALS AND METHODS

MATERIALS AND METHODS

SETING	:	In- patients, Government General Hospital, Madras Medical College, Chennai.
Ethics Committee Approval	:	obtained
Design of study	:	Single center, observational study. (Prospective and Retrospective)
Period of study	:	6 mths
Sample size	:	50 patients

SELECTION OF STUDY SUBJECTS

Inclusion Criteria :

Patients with Spontaneous Intracerebral hemorrhage .

Exclusion Criteria :

Traumatic ICH

Hemorrhagic infarct

AVM and Aneurysmal ICH

Primary IVH

METHODOLOGY

The study was carried out in the wards of Government General Hospital, MMC, Chennai. Patients with spontaneous Intracerebral Hemorrhage as confirmed by computed tomography of the brain were selected.

A total of 50 patients were included as per the selection criteria. They were enrolled into our study after informed consent was signed from the patient and patients closest relative if patient in altered sensorium.

On admission, the detailed history, physical examination and routine laboratory investigations as per profoma are done.

Initial examination include thorough Physical Examination, Neurological examination as per a performa, vital signs, Assessment of Glasgow coma scale score, Assessment of National institute of Health stroke scale (NIHSS) score. Non contrast head CT scan is done in all patients. CT scan were studied to document the anatomical location, Volume of hematoma and the presence of Intraventricular extension, associated Subarachnoid

Hemorrhage, obstructive hydrocephalus. Approximate hematoma volume is calculated by the formula – ABC / 2.

In the bedside ABC/2 method,

CHOOSE CT SLICE - with Largest size of Hematoma

A - Largest Diameter of Hematoma

B - Largest Diameter 90 degrees to A

C - Approximate number of 10mm slices

Haemorrhage area - > 75% area the slice is considered 1

Haemorrhage area - 25% to 75% area, the slice is considered half a haemorrhage slice

Haemorrhage area - less than 25% area , the slice is not considered a haemorrhage slice .

C is measured by adding all the above.

A and B measured - with the centimeter scale on CT scan to the nearest 0.5 cm.

Patients were followed up for a period of three months to assess short term clinical outcome by FUNC Score at 3 months.

NIHSS SCALE

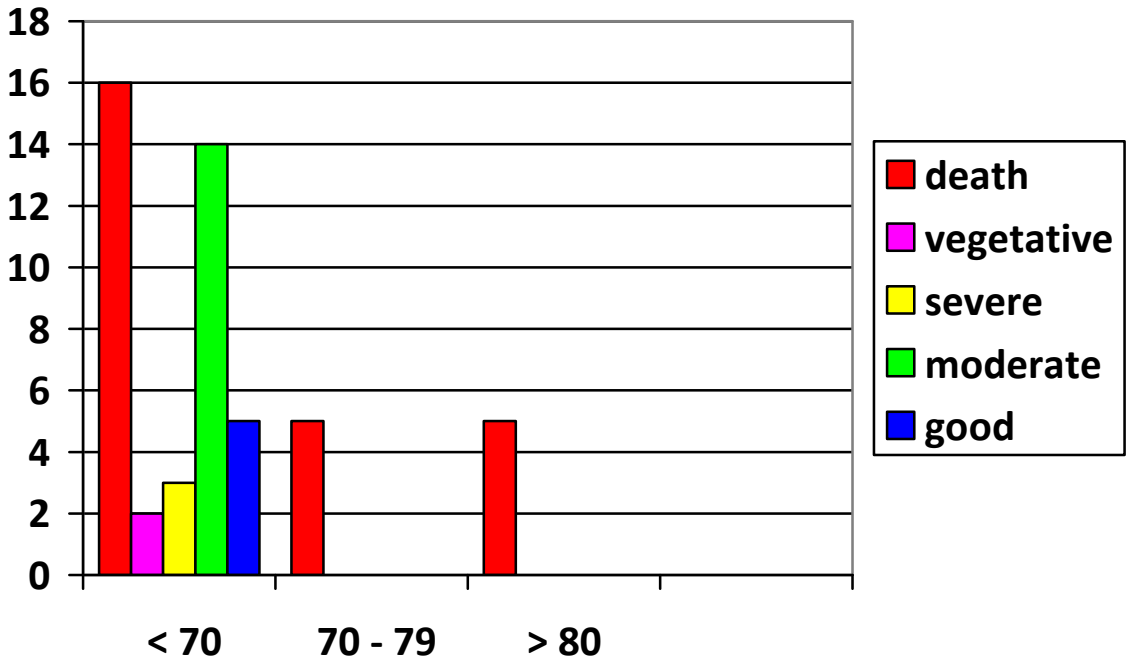
Tested Item	Title	Responses and Scores
1A	Level of consciousness	0—alert
		1—drowsy
		2—obtunded
		3—coma/unresponsive
1B	Orientation questions (two)	0—answers both correctly
		1—answers one correctly
		2—answers neither correctly
1C	Response to commands (two)	0—performs both tasks correctly
		1—performs one task correctly
		2—performs neither
2	Gaze	0—normal horizontal movements
		1—partial gaze palsy
		2—complete gaze palsy
3	Visual fields	0—no visual field defect
		1—partial hemianopia
		2—complete hemianopia
		3—bilateral hemianopia
4	Facial movement	0—normal
		1—minor facial weakness
		2—partial facial weakness
		3—complete unilateral palsy
5	Motor function (arm)	0—no drift
	a. left	1—drift before 5 seconds
	b. right	2—falls before 10 seconds
		3—no effort against gravity
		4—no movement
6	Motor function (leg)	0—no drift
	a. left	1—drift before 5 seconds

	b. right	2—falls before 5 seconds
		3—no effort against gravity
		4—no movement
7	Limb ataxia	0—no ataxia
		1—ataxia in one limb
		2—ataxia in two limbs
8	Sensory	0—no sensory loss
		1—mild sensory loss
		2—severe sensory loss
9	Language	0—normal
		1—mild aphasia
		2—severe aphasia
		3—mute or global aphasia
10	Articulation	0—normal
		1—mild dysarthria
		2—severe dysarthria
11	Extinction or inattention	0—absent
		1—mild (loss 1 sensory modality)
		2—severe (loss 2 modalities)

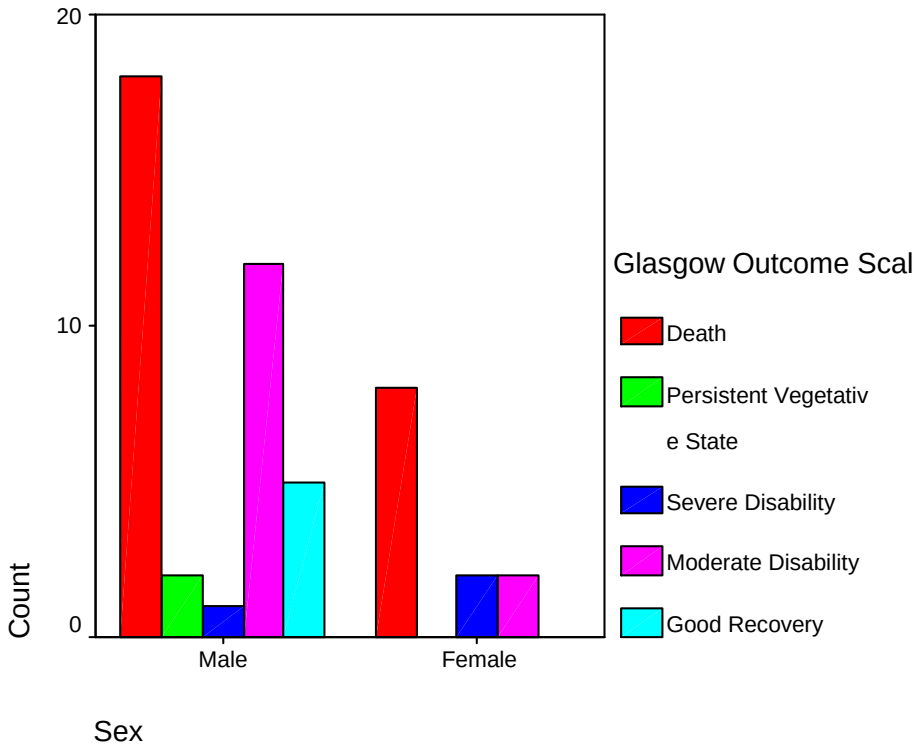
OBSERVATION

OBSERVATIONS

BAR CHART SHOWING AGE DISTRIBUTION

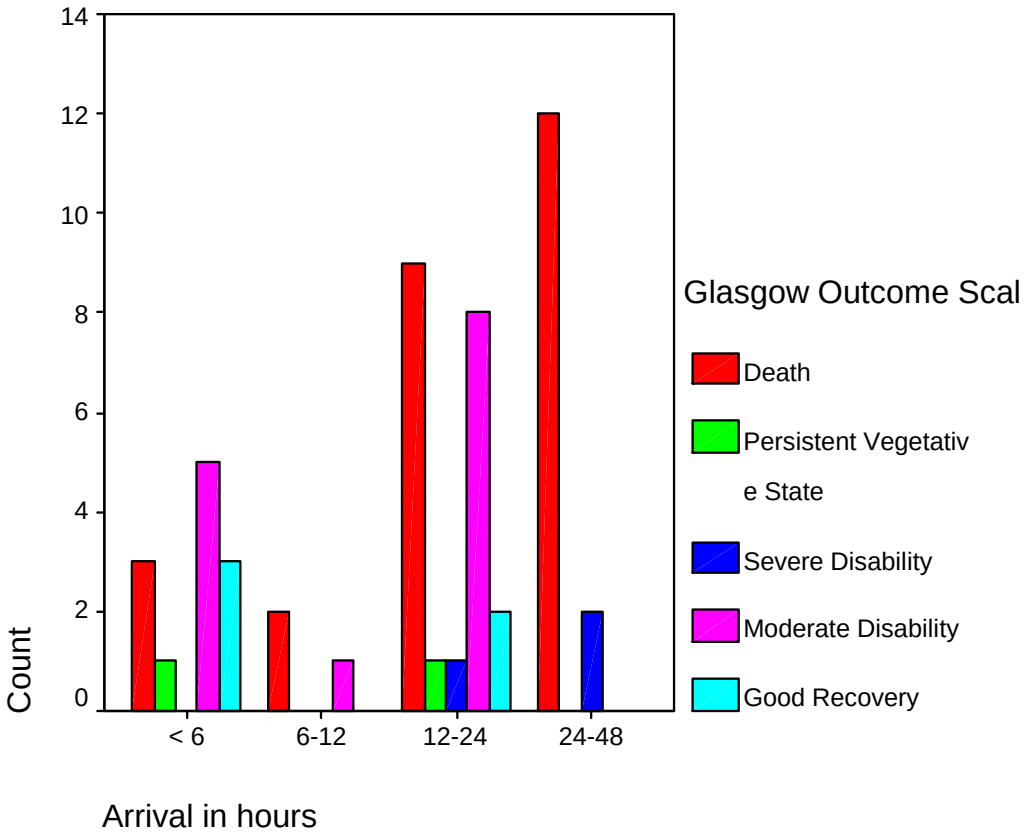


BAR CHART SHOWING SEX DISTRIBUTION



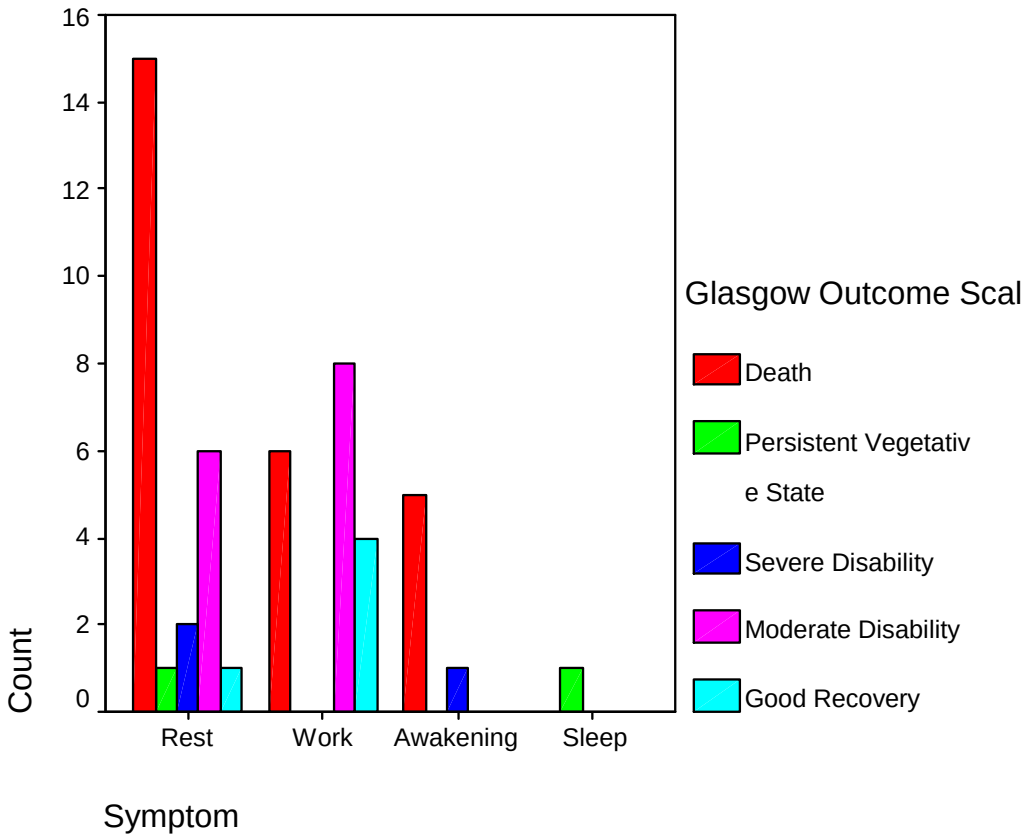
BAR CHART SHOWING ARRIVAL TO HOSPITAL

FROM SYMPTOM ONSET



BAR CHART SHOWING CONDITION OF PATIENT

AT SYMPTOM ONSET



BAR CHART SHOWING GLASGOW COMA SCALE

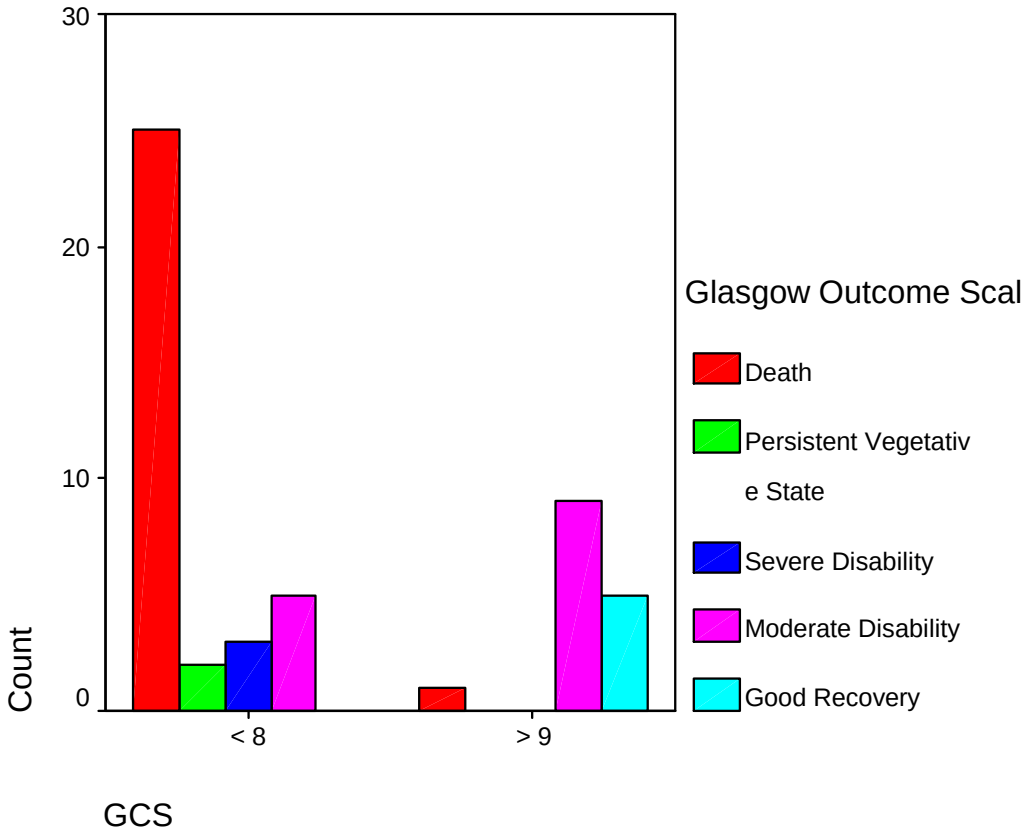


TABLE SHOWING COMPARISON BETWEEN GLASGOW COMA SCALE AND GLASGOW OUTCOME SCALE

			Glasgow Outcome Scale					
			Death	Persistent Vegetative State	Severe Disability	Moderate Disability	Good Recovery	Total
GCS	< 8	Count	25	2	3	5	0	35
		% within GCS	71.4%	5.7%	8.6%	14.3%	.0%	100.0%
		% within Glasgow Outcome Scale	96.2%	100.0%	100.0%	35.7%	.0%	70.0%
	> 9	Count	1	0	0	9	5	15
		% within GCS	6.7%	.0%	.0%	60.0%	33.3%	100.0%
		% within Glasgow Outcome Scale	3.8%	.0%	.0%	64.3%	100.0%	30.0%
Total		Count	26	2	3	14	5	50
		% within GCS	52.0%	4.0%	6.0%	28.0%	10.0%	100.0%
		% within Glasgow Outcome Scale	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	30.115(a)	4	.000
Likelihood Ratio	34.360	4	.000
Linear-by-Linear Association	25.858	1	.000
N of Valid Cases	50		

BAR CHART SHOWING

NATIONAL INSTITUTE OF HEALTH STROKE SCALE

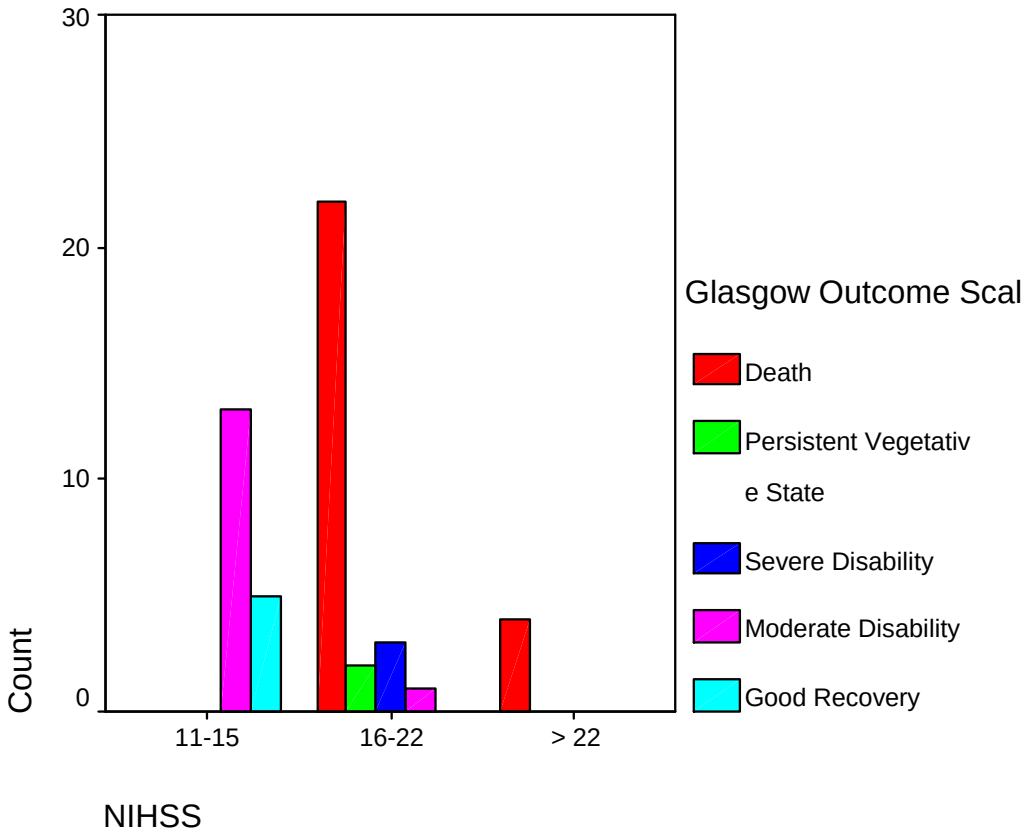


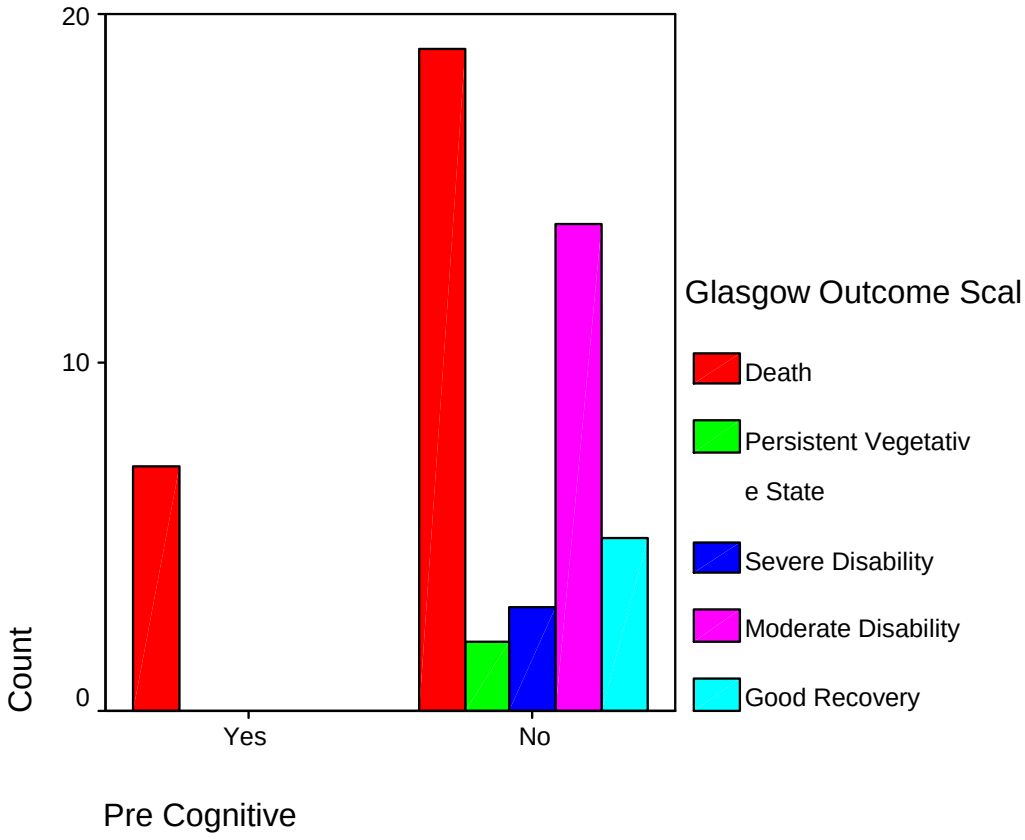
TABLE SHOWING COMPARISON BETWEEN NIHSS AND GLASGOW OUTCOME SCALE

			Glasgow Outcome Scale					
			Death	Persistent Vegetative State	Severe Disability	Moderate Disability	Good Recovery	Total
NIHSS	11-15	Count	0	0	0	13	5	18
		% within NIHSS	.0%	.0%	.0%	72.2%	27.8%	100.0%
		% within Glasgow Outcome Scale	.0%	.0%	.0%	92.9%	100.0%	36.0%
	16-22	Count	22	2	3	1	0	28
		% within NIHSS	78.6%	7.1%	10.7%	3.6%	.0%	100.0%
		% within Glasgow Outcome Scale	84.6%	100.0%	100.0%	7.1%	.0%	56.0%
	> 22	Count	4	0	0	0	0	4
		% within NIHSS	100.0%	.0%	.0%	.0%	.0%	100.0%
		% within Glasgow Outcome Scale	15.4%	.0%	.0%	.0%	.0%	8.0%
Total		Count	26	2	3	14	5	50
		% within NIHSS	52.0%	4.0%	6.0%	28.0%	10.0%	100.0%
		% within Glasgow Outcome Scale	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	47.411(a)	8	.000
Likelihood Ratio	59.925	8	.000
Linear-by-Linear Association	34.485	1	.000
N of Valid Cases	50		

a. 11 cells (73.3%) have expected count less than 5. The minimum expected count is .16.

BAR CHART SHOWING PRE COGNITIVE IMPAIRMENT



BAR CHART SHOWING COMPLICATIONS OF ICH

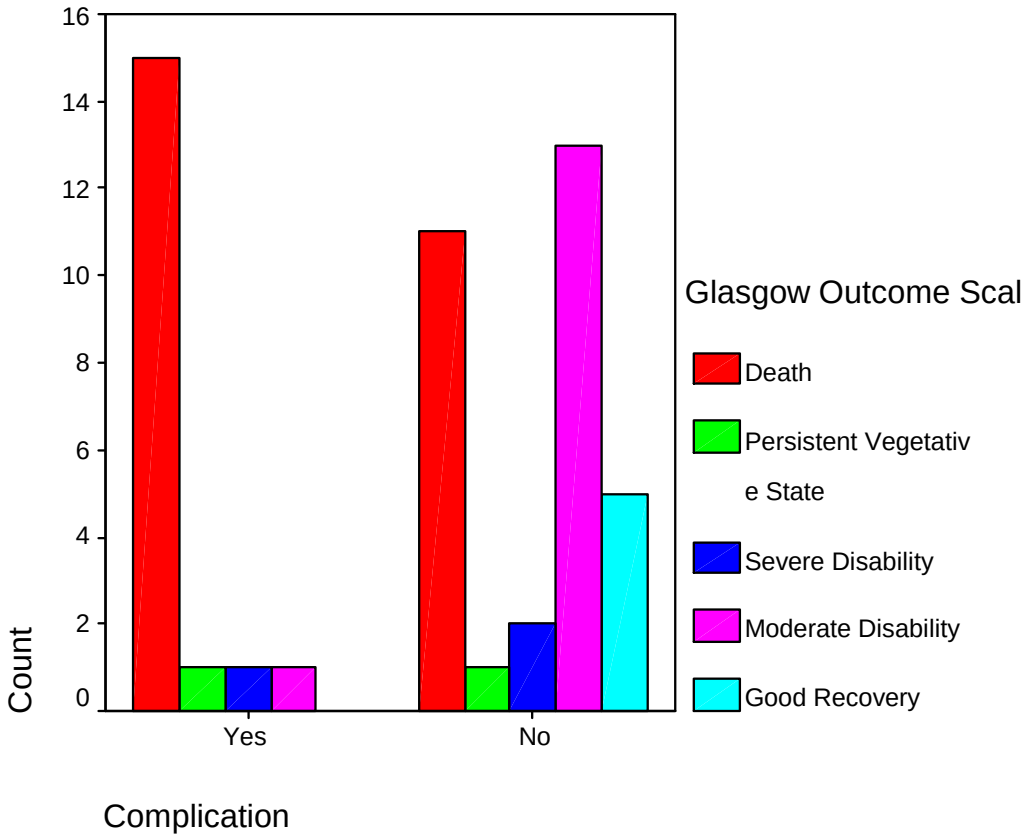


TABLE SHOWING COMPARISON BETWEEN COMPLICATION OF ICH AND GLASGOW OUTCOME SCALE

			Glasgow Outcome Scale					
			Death	Persistent Vegetative State	Severe Disability	Moderate Disability	Good Recovery	Total
Complication	Yes	Count	15	1	1	1	0	18
		% within Complication	83.3%	5.6%	5.6%	5.6%	.0%	100.0%
		% within Glasgow Outcome Scale	57.7%	50.0%	33.3%	7.1%	.0%	36.0%
	No	Count	11	1	2	13	5	32
		% within Complication	34.4%	3.1%	6.3%	40.6%	15.6%	100.0%
		% within Glasgow Outcome Scale	42.3%	50.0%	66.7%	92.9%	100.0%	64.0%
Total		Count	26	2	3	14	5	50
		% within Complication	52.0%	4.0%	6.0%	28.0%	10.0%	100.0%
		% within Glasgow Outcome Scale	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	13.362(a)	4	.010
Likelihood Ratio	16.119	4	.003
Linear-by-Linear Association	12.852	1	.000
N of Valid Cases	50		

a. 6 cells (60.0%) have expected count less than 5. The minimum expected count is .72.

BAR CHART SHOWING VOLUME OF ICH

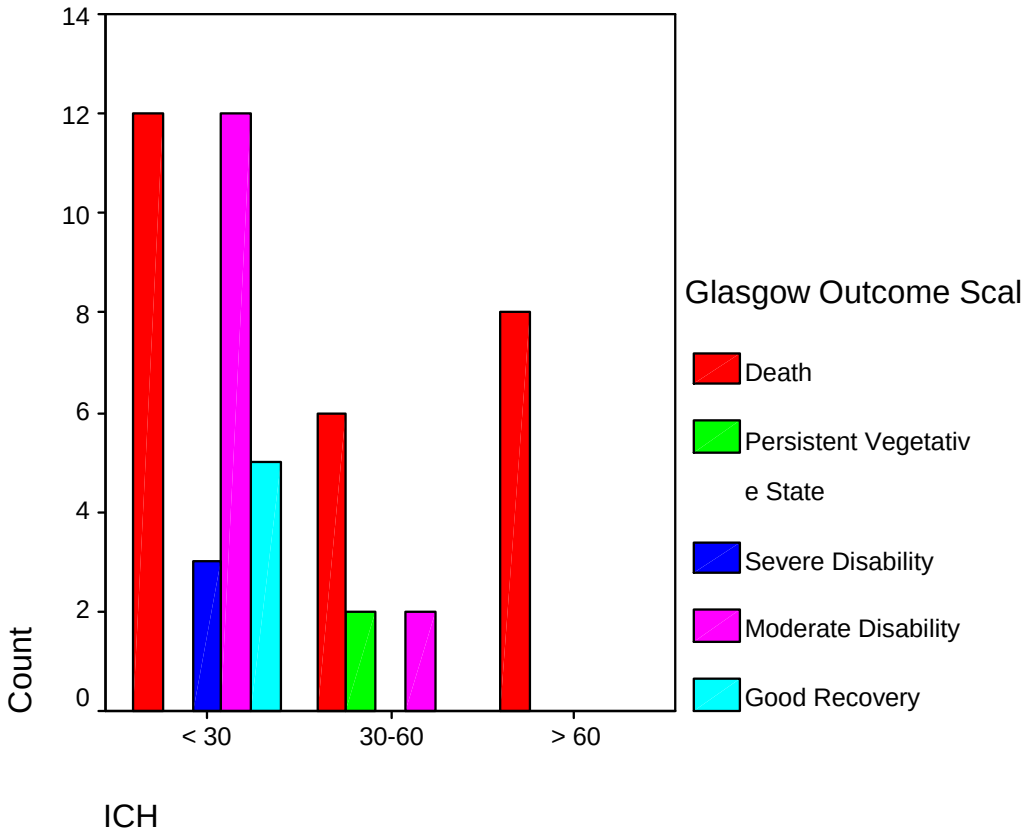


TABLE SHOWING COMPARISON BETWEEN ICH VOLUME AND GLASGOW OUTCOME SCALE

			Glasgow Outcome Scale					
			Death	Persistent Vegetative State	Severe Disability	Moderate Disability	Good Recovery	Total
ICH	< 30	Count	12	0	3	12	5	32
		% within ICH	37.5%	.0%	9.4%	37.5%	15.6%	100.0%
		% within Glasgow Outcome Scale	46.2%	.0%	100.0%	85.7%	100.0%	64.0%
	30-60	Count	6	2	0	2	0	10
		% within ICH	60.0%	20.0%	.0%	20.0%	.0%	100.0%
		% within Glasgow Outcome Scale	23.1%	100.0%	.0%	14.3%	.0%	20.0%
	> 60	Count	8	0	0	0	0	8
		% within ICH	100.0%	.0%	.0%	.0%	.0%	100.0%
		% within Glasgow Outcome Scale	30.8%	.0%	.0%	.0%	.0%	16.0%
Total		Count	26	2	3	14	5	50
		% within ICH	52.0%	4.0%	6.0%	28.0%	10.0%	100.0%
		% within Glasgow Outcome Scale	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	20.962(a)	8	.007
Likelihood Ratio	23.578	8	.003
Linear-by-Linear Association	11.375	1	.001
N of Valid Cases	50		

a. 12 cells (80.0%) have expected count less than 5. The minimum expected count is .32.

BAR CHART SHOWING LOCATION OF ICH

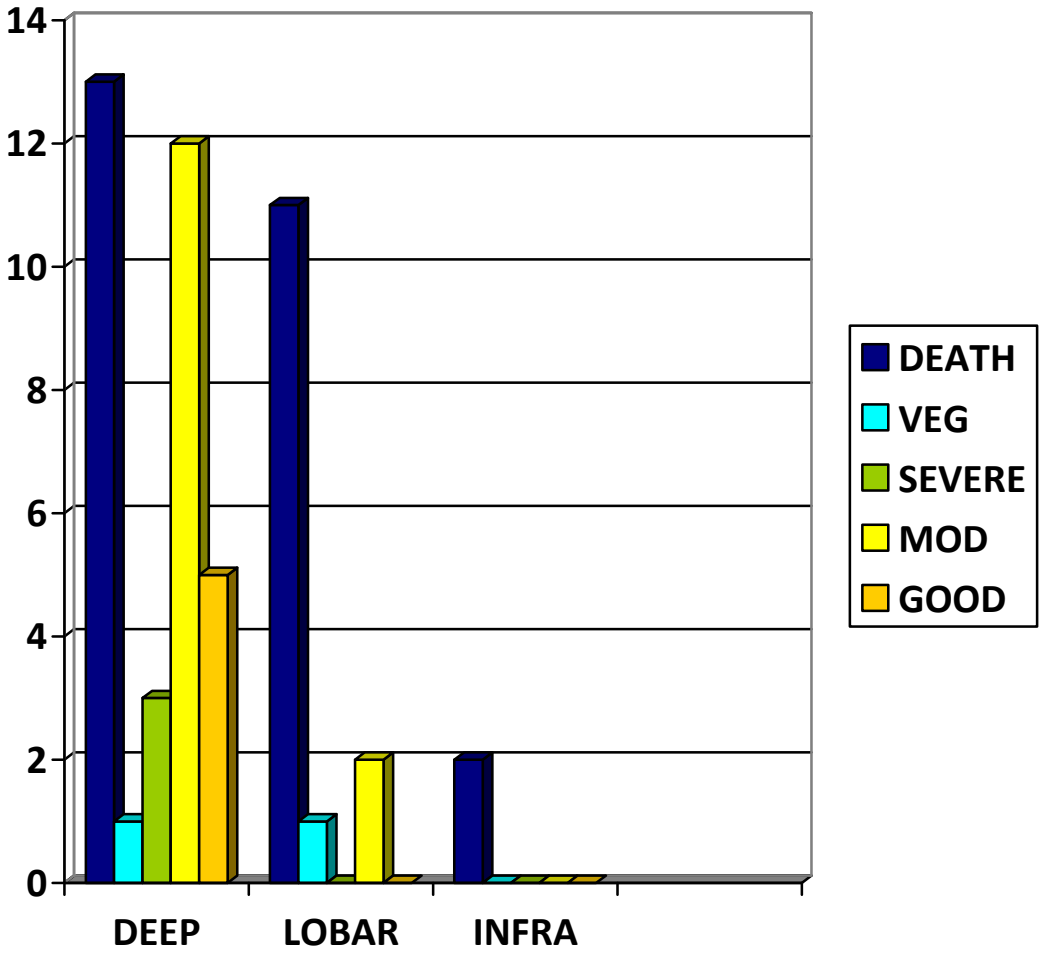


TABLE SHOWING COMPARISON BETWEEN ICH LOCATION AND GLASGOW OUTCOME SCALE

			Glasgow Outcome Scale					
			Death	Persistent Vegetative State	Severe Disability	Moderate Disability	Good Recovery	Total
ICH	Deep	Count	13	1	3	12	5	34
		% within ICH	38.2%	2.9%	8.8%	35.3%	14.7%	100.0%
		% within Glasgow Outcome Scale	50.0%	50.0%	100.0%	85.7%	100.0%	68.0%
	Lobar	Count	11	1	0	2	0	14
		% within ICH	78.6%	7.1%	.0%	14.3%	.0%	100.0%
		% within Glasgow Outcome Scale	42.3%	50.0%	.0%	14.3%	.0%	28.0%
	Intratentorial	Count	2	0	0	0	0	2
		% within ICH	100.0%	.0%	.0%	.0%	.0%	100.0%
		% within Glasgow Outcome Scale	7.7%	.0%	.0%	.0%	.0%	4.0%
Total		Count	26	2	3	14	5	50
		% within ICH	52.0%	4.0%	6.0%	28.0%	10.0%	100.0%
		% within Glasgow Outcome Scale	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	10.458(a)	8	.234
Likelihood Ratio	13.282	8	.103

TABLE SHOWING COMPARISON BETWEEN FUNC SCORE AND GLASGOW OUTCOME SCALE

Descriptives

Func Score

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Death	26	6.23	1.142	.224	5.77	6.69	4	8
Persistent Vegetative State	2	6.50	.707	.500	.15	12.85	6	7
Severe Disability	3	8.00	.000	.000	8.00	8.00	8	8
Moderate Disability	14	9.29	1.069	.286	8.67	9.90	7	10
Good Recovery	5	10.40	.548	.245	9.72	11.08	10	11
Total	50	7.62	1.915	.271	7.08	8.16	4	11

ANOVA

Func Score

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	130.607	4	32.652	29.881	.000
Within Groups	49.173	45	1.093		
Total	179.780	49			

DISCUSSION

DISCUSSION

AGE DISTRIBUTION:

In our Study about 20% were in the Age group of <40years, 50% were in the age group of 40 – 60 years and 30 % were in the age group of > 60years. Mean age is 55.44 yrs

ICH is rare before the age of 45years and its increasing with advancing age. In our study only 20% below 45years, suggesting that advancing age increases the risk of ICH[116,117].

SEX DISTRIBUTION:

Among the 50 patients enrolled, Male patients constituted 38 (76%) and Female patients 12 (24%) making up a male : female ratio of 3:1 .

From previous studies (Sethi et al 2002) it is obvious in India the prevalence of ICH is 7 :1 (male : female)[118] . ICH occurs more commonly in Males than Females [119].

RISK FACTORS :

Almost all patients in our study were known Hypertensives , but most of them on irregular treatment. The prevalence of hypertension has been found to rise in the recent years globally. Kearney et al., pooled data from all over 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.

In our study, 44% 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.

According to a study done by Thakkappan the overall hypertension prevalence is 36.7% in southern part of India[120]. Hypertension is directly responsible for 57% of all stroke deaths in India.

On analysing the arrival of patients to Hospital after the symptom onset, it showed most of the patients who arrive after 48hours had a Glasgow Outcome scale of 1 .

The analysis of Educational Status of the patient population showed that only 20% are illiterate, this probably reflects that educated people in higher socio economic status are more prone for Hypertension and its complication.

On analysing the Blood pressure , average Systolic Blood Pressure is 181.80mmHg and average Diastolic Blood Pressure is 107.80mmHg. Glasgow Outcome scale of 1 correlates with Higher Blood Pressure.

On analysing the symptoms at Onset, Limb weekness was Predominant one and is similar to loss of consciousness with limb weekness.

In our study, we found that there was a correlation between Glasgow coma scale and Glasgow outcome scale. Patients who presented with low GCS had poorer outcomes.

We assessed NIHSS for all patients on presentation in our study , when we followed these patients , we found those with lower NIHSS had better outcomes.

In our study we found that deep intracerebral bleeds were the most common type among intracranial hemorrhages. Although deep cerebral bleeds were common, mortality rates were higher among patients with Infratentorial and lobar hemorrhages. Both the patients with infratentorial

haemorrhage died. Among the fourteen patients with lobar haemorrhage, eleven patients died (78.6%).

In our study, all the eight patients with Hematoma volume > 60 ml died. Among the ten patients with hematoma volume between 30 and 60 ml, six patients died, two patients entered persistant vegetative state and the remaining two patients were left with moderate disability. There were 32 patients with hematoma volume < 30 ml in our study. Within this group, five patients experienced good recovery. Among the remaining 27 patients in this group, twelve had moderate disability, three had severe disability and another twelve died. While comparing the FUNC score with Glasgow outcome scale, we found a strong correlation between the two as demonstrated in earlier studies. As the FUNC score increased there was a strong tendency towards better outcomes.

CONCLUSION

CONCLUSION

1. Male predominate among patients of ICH with a ratio of 3:1.

2. Advancing Age and Hypertension is the most important causative factor for ICH.

3. Among patients of ICH, Limb weekness with loss of consciousness is reported by 44% of patients.

4. The frequency of ICH location is as follows: Deep (68%) ; lobar (14%) and Infratentorial (2%) .

5. The FUNC score strongly correlates with Glasgow outcome scale . As the FUNC score increases, there is a strong tendency towards better outcome.

“FUNC score is a valid clinical assessment tool that identifies patients with ICH who will attain functional independence and thus, can provide guidance in clinical decision-making and patient selection for clinical trials ”.

LIMITATIONS

The Sample Size is small in our study, further studies involving larger patients required to improve functional outcome.

Chronic Alcohol intake also influences the incidence of Intracerebral Hemorrhage.

In future further research is needed to improve survival among those patients who survive from ictus and it is more usefull to focus on disability rates among survivors.

SUMMARY

Because mortality figures are all confounded by the fact that withdrawal of aggressive care by clinicians and families is a very common precipitant of death in these patients and often occurs inpatients Whose ICH is survivable, it is more useful to focus on rates of disability among survivors when discussing prognosis.

Because ICH is a devastating condition physicians and families are often confronted with the question of whether their patient would choose to survive the event. Accurate prediction of prognosis is essential to guide such decision-making.

The FUNC score enables prediction of the likelihood of recovering functional independence for patients with primary ICH. Tools such as the FUNC score calculator can be useful in guiding decisions about aggressiveness of care, but their precision remains to be proved

“ The FUNC score is a valid clinical assessment tool used to identify those patients with ICH who will attain long-term functional independence.. This prediction scale provides essential guidance for physicians and families who are confronted with decision making about direction of care for their patients and selection strategy for clinical trials”

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BIBLIOGRAPHY

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ANNEXURES

PROFOMA

Name: Age/Sex:

Address: Occupation:

Symptoms:

Past history:

Diabetes mellitus, Hypertension, Coronary artery disease, Heart failure Kidney disease, Tuberculosis , Other co morbid illnesses

Personal history:

- Smoking / Alcoholism

Vitals:

PULSE:

BLOOD PRESSURE:

RESPIRATORY RATE

General examination:

Systemic examination:

CVS:

RS:

ABDOMEN:

CNS:

Investigations :

CBC			**LIPID PROFILE**		
RBC			TOTAL CHL		mg/dl
TC	DC N	L	TGL		mg/dl
	E	B	HDL		
HB			LDL		
ESR					
PLATELET					
RFT			**LFT**		
Glucose		mg/dl	Total bilirubin		mg/dl
Urea		mg/dl	Direct bilirubin		mg/dl
Creatinine		mg/dl	SGOT		U/l
Na+		mEq/l	SGPT		U/l
K+		mEq/l	ALP		U/l
			Total protein		g/dl
			Albumin		g/dl

Chest X-ray:

ECG;

ECHO :

FUNC SCORE CALCULATION

NON CONTRAST CT BRAIN :

AGE :

GCS :

NIHSS :

ABC / 2 VOLUME :

Pre – COGNITIVE IMPAIRMENT :

FUNC SCORE :

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.T.S.Karthigeyan,
Post Graduate, MD (General Medicine),
Institute of Internal Medicine,
Madras Medical College,
Chennai – 600 003.

Dr.T.S.Karthigeyan,

The Institutional Ethics Committee has considered your request and approved your study titled **"Prediction of Functional outcome in primary spontaneous Intracerebral hemorrhage using FUNC scoring"** No.48072014.

The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr.C.Rajendran, M.D.,	: Chairperson
2.	Dr.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3.	Prof.B.Kalaiselvi, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4.	Prof.R.Nandhini, M.D., Inst.of Pharmacology, MMC	: Member
5.	Dr.G.Muralidharan, Director Incharge, Inst.of Surgery	: Member
6.	Prof.Md.Ali, M.D., D.M., Prof & HOD of MGE, MMC	: Member
7.	Prof.K.Ramadevi, Director i/c, Inst.of Biochemistry, MMC	: Member
8.	Prof.Saraswathy, M.D., Director, Pathology, MMC, Ch-3	: Member
9.	Prof.Tito, M.D., Director i/c, Inst.of Internal Medicine, MMC	: Member
10.	Thiru S.Rameshkumar, Administrative Officer	: Lay Person
11.	Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
12.	Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

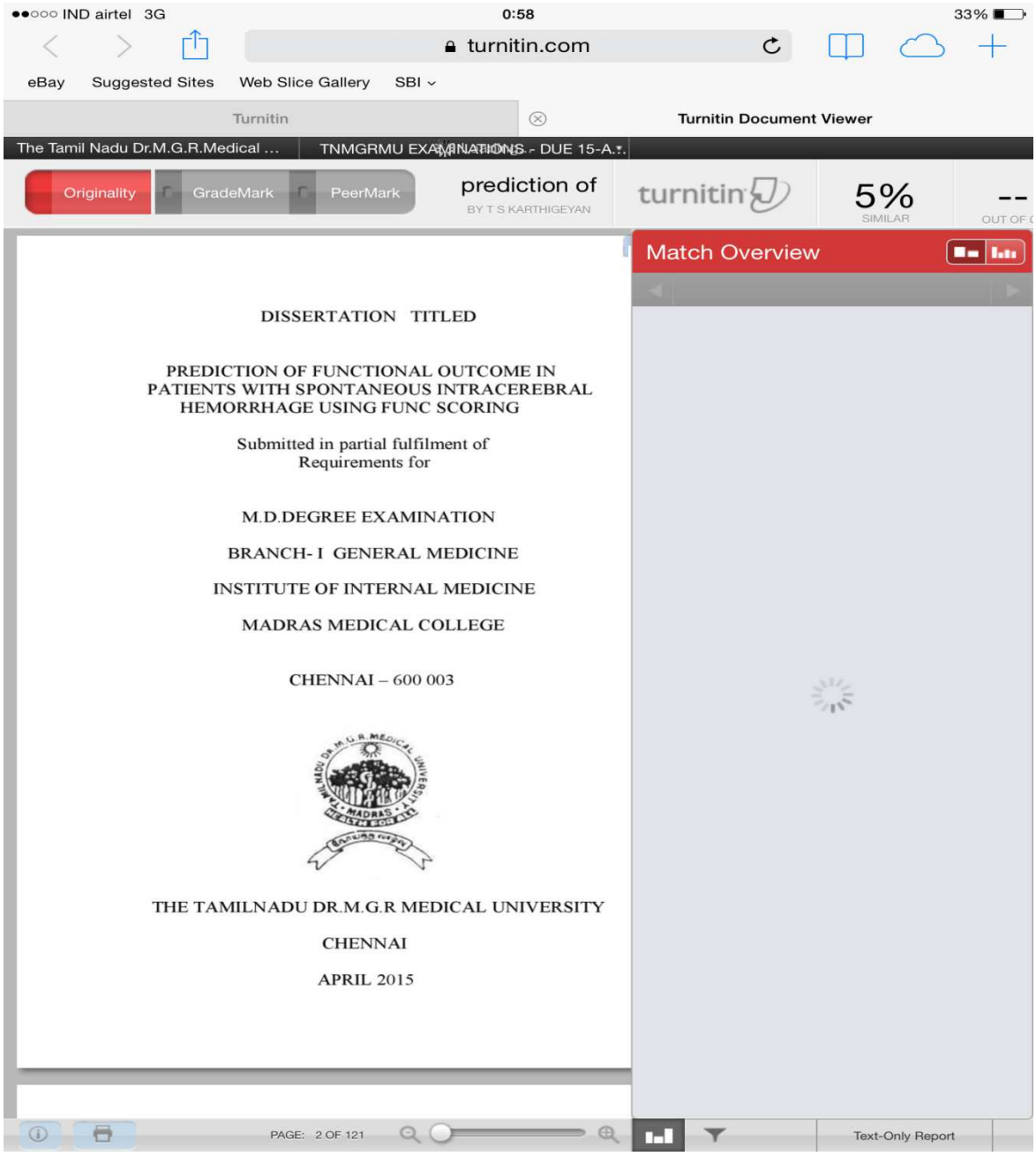
Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COL.
CHENNAI-600 003

TURNITIN PLAGIARISM SCREEN SHOT



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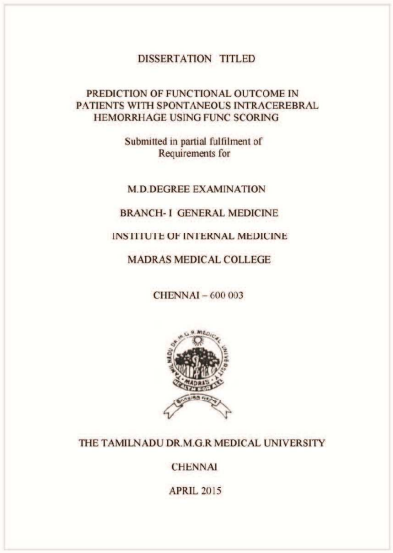

DISSERTATION TITLED

PREDICTION OF FUNCTIONAL OUTCOME IN PATIENTS WITH SPONTANEOUS INTRACEREBRAL HEMORRHAGE USING FUNC SCORING

Submitted in partial fulfilment of Requirements for

M.D.DEGREE EXAMINATION

BRANCH- I GENERAL MEDICINE

INSTITUTE OF INTERNAL MEDICINE

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

APRIL 2015

PATIENT CONSENT FORM

Study Detail	:	**PREDICTION OF FUNCTIONAL OUTCOME IN PATIENTS WITH SPONTANEOUS INTRACEREBRAL HEMORRHAGEUSING FUNC SCORING**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (☑) these boxes

The details of the study have been provide to me in writing and explained to me in my own language	
I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.	
I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.	
I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.	
I hereby consent to participate in this study.	
I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests.	

Signature/thumb impression
Patient's Name and Address:
Signature of Investigator
Study Investigator's Name: **Dr. T.S.KARTHIGEYAN**

INFORMATION SHEET

We are conducting a study on "**PREDICTION OF UNCTIONAL OUTCOME IN PATIENTS WITH SPONTANEOUS INTRACEREBRAL HEMORRHAGE USING FUNC SCORING**" among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to assess clinical profile of patients admitted with Spontaneous Intracerebral Haemorrhage in RGGGH and the utility of FUNC SCORE in predicting likelihood of functional independence.

We are selecting certain cases and if you are found eligible, after filling up the questionnaire, CT Brain plain will be taken during admission . These tests and special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator Signature of Participant

Date :

Place :

MASTER CHART

SERIAL NO	AGE yrs	SEX	ARRIVAL	EDUCATION	SYMPTOMS	SYSTOLIC BP	DIASTOLIC BP	SYMPTOM	GCS	NIHSS	RISK	ICH	PRE COGNITIVE	COMPLICATION	ICH	FUNC SCORE	Glasgow Outcome Scale
								NOTICED FIRST	SCORE		FACTORS	LOCATION	IMPAIRMENT	IVH - SAH	VOLUME		At 90 Days
1	57	male	12-24hrs	primary	loc ,limb weekness	210	130	work	≤ 8	16 - 22	HTN , alcoholic	Deep	No	Yes	30 -60ml	7	1
2	45	male	12-24hrs	primary	loc ,limb weekness	200	110	rest	≤ 8	16 - 22	HTN , alcoholic	Deep	No	Yes	30 -60ml	6	2
3	46	male	12-24hrs	secondary	limb weekness	180	100	rest	≥ 9	11-15	HTN	Deep	No	No	< 30ml	10	4
4	63	female	6 - 12hrs	uneducated	limb weekness	140	100	rest	≥ 9	11-15	HTN	Deep	No	No	<30ml	10	4
5	40	male	12-24hrs	secondary	limb weekness	150	100	work	≥ 9	11-15	HTN , alcoholic	Deep	No	No	<30ml	10	5
6	35	female	24-48hrs	uneducated	loc ,limb weekness	180	100	awakening	≤ 8	16-22	HTN	Deep	No	No	<30ml	8	3
7	38	male	12-24hrs	secondary	limb weekness	150	100	work	≥ 9	11-15	HTN , alcoholic	Deep	No	No	<30ml	10	4
8	64	female	>2days	uneducated	loc ,limb weekness	200	110	rest	≤ 8	16 -22	HTN	Deep	No	No	30 -60ml	6	1
9	65	male	0 - 6hrs	degree	limb weekness	180	100	work	≥ 9	11-15	HTN , old CVA	Deep	No	No	<30ml	10	5
10	32	male	0 - 6hrs	degree	limb weekness	150	100	work	≥ 9	11-15	HTN ,Alcoholic	Deep	No	No	<30ml	10	5
11	49	male	12-24hrs	degree	limb weekness	180	100	rest	≤ 8	11-15	HTN, alcoholic	Deep	No	No	<30ml	8	4
12	45	male	0 - 6hrs	degree	limb weekness	150	110	work	≥ 9	11-15	HTN, alcoholic	Deep	No	No	<30ml	10	4
13	60	male	12- 24hrs	primary	limb weekness	180	100	rest	≤ 8	16 -22	HTN, alcoholic	Deep	No	Yes	<30ml	8	1
14	36	male	24 -48hrs	degree	loc ,limb weekness	220	130	rest	≤ 8	16 -22	HTN, alcoholic	Deep	No	No	<30ml	8	3
15	45	male	0 - 6hrs	degree	limb weekness	160	100	work	≤ 8	11-15	HTN, alcoholic	Deep	No	No	<30ml	10	4
16	80	female	6 -12hrs	uneducated	loc ,limb weekness	150	100	awakening	≤ 8	16 -22	HTN	Deep	Yes	No	<30ml	5	1
17	37	male	0 - 6hrs	degree	limb weekness	180	100	work	≥ 9	11-15	HTN, alcoholic	Deep	No	No	<30ml	10	4
18	80	female	12 -24hrs	uneducated	loc ,limb weekness	210	120	awakening	≤ 8	16 -22	HTN	Deep	No	No	<30ml	6	1
19	55	male	12 -24hrs	degree	limb weekness	180	110	work	≥ 9	11-15	HTN, alcoholic	Deep	No	No	<30ml	10	4
20	52	male	24 -48hrs	secondary	loc ,limb weekness	210	130	work	≤ 8	16 -22	HTN, alcoholic, DM	Lobar	No	No	> 60ml	7	1
21	65	female	24 -48hrs	uneducated	loc ,limb weekness	210	120	rest	≤ 8	16 -22	HTN	Deep	No	Yes	<30ml	8	1
22	70	male	24 -48hrs	secondary	loc ,limb weekness	200	100	awakening	≤ 8	16 - 22	HTN,alcoholic	Deep	Yes	Yes	<30ml	6	1

23	37	male	0 -6hrs	degree	limb weekness	150	100	work	≥ 9	11-15	HTN	Deep	No	No	<30ml	11	5
24	50	male	24 -48hrs	degree	loc ,limb weekness	210	110	rest	≤ 8	16 -22	HTN,alcoholic	Lobar	No	No	>60ml	7	1
25	74	male	6 -12hrs	secondary	limb weekness	160	100	rest	≤ 8	16 -22	HTN	Lobar	Yes	Yes	30 -60ml	6	1
26	55	male	24 -48hrs	secondary	loc ,limb weekness	210	120	rest	≤ 8	16 -22	HTN	Lobar	No	Yes	>60ml	5	1
27	27	male	24 -48hrs	degree	loc ,limb weekness	220	140	work	≤ 8	>22	HTN,alcoholic	Lobar	No	Yes	>60ml	5	1
28	95	male	24 -48hrs	secondary	loc	160	100	rest	≤ 8	16-22	HTN	Lobar	Yes	No	<30ml	6	1
29	70	male	24 -48hrs	uneducated	loc ,limb weekness	220	140	rest	≤ 8	16-22	HTN	Lobar	Yes	Yes	>60ml	4	1
30	46	male	24 -48hrs	degree	limb weekness	210	100	work	≤ 8	16 -22	HTN,alcoholic	Deep	No	Yes	30-60ml	7	1
31	60	female	24 -48hrs	uneducated	loc ,limb weekness	200	110	awakening	≤ 8	16 -22	HTN	Deep	No	No	30-60ml	6	1
32	72	male	12 -24hrs	secondary	limb weekness	180	100	rest	≥ 9	16 -22	HTN,alcoholic	Deep	Yes	No	< 30ml	8	1
33	40	male	0 -6hrs	degree	seizure , limb weekness	140	100	sleep	≤ 8	16 -22	HTN,alcoholic	Lobar	No	No	30-60ml	7	2
34	60	female	12 -24hrs	uneducated	loc ,limb weekness	210	120	rest	≤ 8	16-22	HTN	Lobar	No	Yes	30-60ml	7	1
35	55	female	12 -24hrs	uneducated	limb weekness	130	70	rest	≥ 9	11-15	HTN	Lobar	No	No	30-60ml	9	4
36	55	male	0-6 hrs	degree	limb weekness	150	100	work	≤ 8	16 -22	HTN,alcoholic	Lobar	No	Yes	30 -60ml	7	4
37	40	female	12 -24hrs	primary	limb weekness	220	140	rest	≤ 8	16 -22	HTN	Lobar	No	No	>60ml	5	1
38	55	male	0 -6hrs	degree	loc ,limb weekness	160	100	work	≤ 8	16-22	HTN,alcoholic	Lobar	No	Yes	>60ml	5	1
39	62	male	0-6hrs	degree	loc ,limb weekness	220	140	rest	≤ 8	>22	HTN,alcoholic	Lobar	no	Yes	>60ml	7	1
40	75	male	12 -24hrs	secondary	loc ,limb weekness	180	100	rest	≤ 8	16-22	HTN	Deep	No	No	<30ml	7	1
41	56	male	12 -24hrs	degree	loc ,limb weekness	210	110	rest	≤ 8	16 -22	HTN,alcoholic	Deep	No	Yes	<3oml	6	1
42	50	male	0 -6hrs	degree	limb weekness	170	100	rest	≥ 9	11-15	HTN,alcoholic	Deep	No	No	<30ml	10	4
43	60	male	12 -24hrs	degree	limb weekness	140	100	rest	≤ 8	11-15	HTN	Deep	No	No	<30ml	10	4
44	45	male	12 -24hrs	degree	limb weekness	150	100	work	≥ 9	11-15	HTN,alcoholic	Deep	No	No	<30ml	8	4
45	47	male	12 -24hrs	degree	loc	210	110	work	≤ 8	>22	HTN	infratentorial	No	Yes	<30ml	7	1
46	85	female	24 -48hrs	uneducated	loc ,limb weekness	150	100	awakening	≤ 8	16 -22	HTN	Deep	No	Yes	< 30ml	7	1
47	46	male	12 -24hrs	degree	limb weekness	200	100	work	≤ 8	11-15	HTN,alcoholic	Deep	No	No	<30ml	8	4
48	63	male	12 -24hrs	degree	limb weekness	180	100	rest	≥ 9	11-15	HTN	Deep	No	No	<30ml	11	5
49	48	female	12 -24hrs	degree	limb weekness	200	110	rest	≤ 8	16-22	HTN	Deep	No	Yes	<30ml	8	3
50	85	male	0 -6hrs	secondary	loc	180	100	rest	≤ 8	>22	HTN	infratentorial	Yes	No	<30ml	4	1