INTRACRANIAL PRESSURE, CEREBRAL PERFUSION PRESSURE AND CEREBRAL COMPLIANCE MONITORING AND THEIR RELATIONSHIP AMONGST ADULT SEVERE HEAD INJURY

BY

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ABSTRAK

Pengenalan

Kajian ini menjurus kepada pemerhatian hubungan antara parameter fisiologi intrakranial iaitu tekanan intrakranial (ICP), tekanan perfusi serebral (CPP), dan kekenyalan otak (CI). Selain itu pemerhatian berkenaan hubungan antara parameter-parameter di atas dengan imbasan tomografi otak berkomputer, faktor-faktor klinikal dan bukan klinikal serta kesan terhadap pesakit juga dilakukan.

Prosedur kajian

Ini adalah kajian yang dilakukan secara prospektif di mana pesakit-pesakit yang mengalami kecederaan kepala serius dengan nilai GCS terkumpul kurang daripada 8 dimasukkan di dalam kajian. Pesakit dimasukkan ke dalam Unit Rawatan Rapi Neurosains selepas menjalani pembedahan mengeluarkan darah beku dan parameter-parameter intrakranial dan ekstrakranial telah direkodkan. Pesakit-pesakit yang mempunyai saiz anak mata yang besar dan tidak reaktif, mengalami kecederaan parah yang dijangkakan boleh menyebabkan kematian dalam tempoh 24 jam tidak dipilih ke dalam kajian. Selain itu pesakit yang mempunyai masalah pembekuan darah, kecederaan otak pangkal dan tidak boleh disusuli selepas pulang ke rumah juga tidak dimasukkan ke dalan kajian ini. Semua pesakit telah dirawat mengikut protokol rawatan kecederaan kepala serius yang telah ditetapkan. Pesakit-pesakit menerima rawatan rapi sehingga semua parameter kembali normal atau pesakit meninggal dunia. Imbasan otak tomografi berkomputer telah dibuat dalam masa 48 jam selepas pembedahan mengeluarkan darah

beku atau lebih awal jika pemerhatian menunjukkan tanda-tanda tekanan otak yang tinggi. Pesakit diperiksa kembali sebulan dan enam bulan selepas kecederaan dan pemerhatian menggunakan GOS telah direkodkan.

Keputusan

Seramai 30 orang pesakit dengan 24 orang lelaki dan 6 orang perempuan berumur antara 13 hingga 65 tahun yang mengalami kecederaan kepala yang teruk telah dimasukkan ke dalam kajian ini. Pemerhatian kami telah menunjukkan nilai CPP dan Cl yang lebih tinggi direkodkan sekiranya ICP dikekalkan pada atau di bawah 20 mmHg. Sementara nilai CPP dan CI yang lebih rendah dicatatkan sekiranya ICP meningkat lebih daripada 20 mmHg selepas pembedahan mengeluarkan darah beku, masing-masing dengan nilai p = 0.001 dan 0.030. Pemerhatian serupa telah dicatatkan jika kekenyalan otak diukur dengan PVI, nilai $p < 0.001$. Hubungan statistik yang jelas telah diperolehi antara CPP dan Cl juga antara CPP dan PVI jika ICP meningkat lebih daripada 20 mmHg dengan nilai $p < 0.001$. Pemerhatian kami juga menunjukkan ICP yang lebih tinggi dan Cl serta PVI yang lebih rendah dicatatkan apabila CPP berada di bawah paras 60 mmHg. ICP telah berkurangan (nilai p <0.001) dan kedua-dua Cl dan PVI masing-masing dengan nilai $p = 0.002$ dan $p < 0.001$ telah bertambah apabila CPP dikekalkan pada paras lebih daripada 60 mmHg. Selepas pembedahan, kajian kami telah menunjukkan hubungan yang jelas antara ICP dan CPP dengan keadaan basal cistem (nilai $p < 0.001$ dan p = 0.022). Hubungan yang serupa diperhatikan antara ICP dan Klasifikasi Marshall, nilai $p <$ 0.001. Walaubagaimanpun pemerhatian kami gagal untuk melihat hubungan antara kekenyalan otak dengan imbasan otak berkomputer selepas pembedahan biarpun keadaan

kekenyalan otak yang lebih baik direkodkan jika imbasan otak menunjukkan basal cistern yang terbuka, tiada atau sedikit pergerakan struktur otak tengah dan Marshall I atau II. Kajian juga menunjukkan perhubungan yang jelas antara GCS sebelum pembedahan dengan ICP dan cara pembedahan dengan kekenyalan otak, masing-masing dengan nilai $p = 0.006$ dan $p = 0.033$. Faktor umur, GCS sebelum pembedahan, anak mata, basal cistern, imbasan otak berkomputer selepas pembedahan dan ICP mempunyai hubungan dengan proses pemulihan pesakit seperti yang direkodkan dengan skala GOS, nilai $p < 0.050$, walaupun kajian ini hanya menunjukkan saiz dan kereaktifan anak mata (nilai $p = 0.025$) dan mungkin ICP (nilai $p = 0.057$) yang boleh menjadi faktor pengukur bebas terhadap kesan kecederaan otak yang serius. Saiz sampel yang lebih besar mungkin akan membuktikan faktor-faktor lain juga bebas menjadi penentu kepada kesan kecederaan otak. Akhir sekali prosedur yang invasif seperti memasukkan alat tekanan pengukur dalam kepada juga boleh menyebabkan komplikasi yang serius.

Kesimpulan

Kajin menunjukkan hubungan yang jelas antara ICP, CPP serta kekenyalan otak (Cl dan PVI) sekiranya autoregulasi otak berada dalam keadaan normal. Namun begitu kajian kami menunjukkan tiada hubungan antara parameter-parameter tersebut dengan kesan kecederaan kepala ke atas pesakit selepas sebulan dan enam bulan. Selain daripada itu, imbasan otak tomografi berkomputer selepas pembedahan mengeluarkan darah beku boleh meramalkan tekanan intrakranial.

ABSTRACT

Introduction

The study is to observe the correlation between intracranial physiological parameters namely intracranial pressure (ICP), cerebral perfusion pressure (CPP) and cerebral compliance in adult severe head injured patients. In addition the relationship of the above parameters with post operative computed tomography scan and other potential clinical, non clinical factors and outcome were also analyzed.

Research methodology

This is a prospective cohort study on severe head injured patients with Glasgow Coma Score of eight and below. The patients were admitted into Neuroscience Intensive Care Unit for monitoring of intracranial and extracranial physiological parameters after evacuation of mass lesion. We excluded patients with bilateral fixed and dilated pupil, those who suffered from severe injury not expected to live longer than 24 hours, those with bleeding diathesis, those whose follow up was not possible or those with significant brain stem involvement. All patients were treated with standard protocols and guidelines in the management of severe head injury. These patients were monitored continuously until the parameters were normal and stabilized or death. Post operative computed tomography was obtained within 48 hours after surgical evacuation of mass lesion or earlier if intracranial hypertension was refractory. Outcomes of the patients at first and sixth month after the injury were assessed using Glasgow Outcome Scale.

Results

The study included 30 patients with 24 males and 6 females, between 13 and 65 years of age who were admitted with severe head injury. We demonstrated higher CPP and compliance values were obtained whenever the ICP was maintained at 20 mmHg or lower, while reduced CPP and compliance value once the ICP was elevated above 20 mmHg following surgery to remove the mass lesion in severe head injured patients, p value = 0.001 and 0.030 respectively. The similar findings were demonstrated when cerebral compliance was measured using PVI, p value < 0.001. There was a statistically significant correlation between CPP and compliance as well as CPP and PVI at ICP above 20 mmHg, p value < 0.001. Our study also revealed higher ICP and lower compliance and PVI when the CPP was below 60 mmHg. The ICP reduced (p value < 0.001) and both compliance (p value = 0.002) as well as PVI (p value < 0.001) improved when the CPP was preserved above 60 mmHg.

Post operatively, our study revealed significant relationship between ICP and CPP with states of basal cistern (p value = 0.001 and 0.022) as well as ICP with Marshall Classification (p value < 0.001). However we failed to demonstrate significant relation between compliance with post operative CT scan findings even though higher compliance value was observed when the basal cistern was opened, less midline shift and Marshall I or II. We also found an association between preoperative GCS with ICP and type of operation with compliance value which may help in the management plan of severe head injured patients, p value = 0.006 and 0.033 respectively.

Age, preoperative GCS, pupil equality and reactivity, basal cistern, post operative CT scan edema and ICP were associated with the outcome of the patients at one month after

severe head injury, p value < 0.050, although we only found pupil equality and reactivity (p value = 0.025) and barely ICP (p value = 0.057) as independent outcome predictors. Larger sample size is required to demonstrate the validity of other parameters as an independent predictor. Lastly the invasive procedure such as insertion of ICP monitoring is not without catastrophic complication.

Conclusion

There were correlation between ICP, CPP and cerebral compliance as long as the cerebral autoregulation remained intact. However no significant relation was observed between these intracranial parameters with the patient's outcome. Other than that, the findings of post operative CT scan obtained after evacuation of mass lesion may help in predicting the intracranial pressure.

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1. INTRODUCTION

Road traffic accident is the leading cause of disability and death in Malaysia with an ever rising trend (Kareem, 2003). Majority of the deaths in road traffic accident are attributed to severe head injury. A patient is said to suffer from severe head injury when he is admitted in a comatose state with Glasgow Coma Score of eight or below after adequate resuscitation or his score deteriorates to eight or below during observation. Majority of the patients who sustain severe head injury have poor outcome. The high morbidity and mortality following severe head injury result in social burden and financial constraint not only to the patient and family members but also to the society and country. Many researches have been conducted in this group of patients during the acute phase of head injury to reduce the mortality and prevent subsequent morbidity (Panerai *et al.*, 2004, Narayan *et al.*, 1981, Zhang *et al.*, 2001, Bullock *et al.*, 2002). Early recognition and aggressive treatment during this acute phase has been shown to improve the outcome. In addition further studies during the rehabilitation phase have also been conducted aiming at improving the outcome to allow victim to return to almost normal life (Schatz *et al.*, 2002, Poon *et al.*, 2005, Levin *et al.*, 1991, Formisano *et al.*, 2004).

Continuous monitoring of the intracranial parameters during intensive care helps in the understanding of the complex pathophysiology of severely injured brain. Correlations between cerebral compliance, ICP and CPP have been reported by Gray and Rosner in animal models (Gray and Rosner, 1987). Inversed correlation between compliance and ICP was observed in pathological brain including in severe head injured patients. Few authors had found direct correlation between compliance and CPP within certain range of

ICP (Portella *et al.*, 2005, Pillai *et al.*, 2004, Yau *et al.*, 2002b). There are many factors that affect the pathophysiology of the brain after injury which include the severity of brain damage at the time of impact, cerebral autoregulation and effectiveness of the treatment. In addition, factors which include age, Glasgow Coma Scale, pupillary response, eye movements, surgical mass, ICP and hypotensive episodes have been reported as the predictors of GOS in several studies (Narayan *et al.*, 1981, Cremer *et al.*, 2006). Furthermore the relation between the intracranial parameters with neuroradiological imaging especially computerized tomography scanning as well as non clinical and other clinical parameters also affects patient's management. Therefore a better understanding of the injured brain pathophysiology, the affecting factors and the association with neuroradiological findings may help us improve treatment strategies during intensive management and eventual patient's outcome.

We were able to carry out the study due to adequate neurointensive care resources and the availability of trained personnel in our department. Three Spiegelberg craniospinal compliance monitoring device were readily available for us to continuously monitor the intracranial pressure, mean arterial pressure, cerebral perfusion pressure, cerebral compliance and pressure volume index after evacuation of surgical mass lesion.

2. LITERATURE REVIEW

2.1 EPIDEMIOLOGY

Head injury is a major cause of morbidity and mortality worldwide with ever rising trend. Severe head injury remains one of the leading causes of death and permanent disability among productive age group in Malaysia. Head injury directly causes a great burden and economic loss to the family and country. It is estimated that 23 to 24 million people are injured worldwide in road crashes and about 1.17 million deaths occur per year worldwide due to road traffic accident. The total number of road traffic accident in Malaysia exceeded 223 000 in 1999 with an average of 16 deaths every single day. The cost involved is calculated at RM 4.6 million a day and a total of RM1.67 billion, making it a major public health problem in Malaysia. Hospital Universiti Sains Malaysia with a tertiary neuroscience centre registered a total of 522 cases in 1997, which increased to 570 in 1999 for the management of head injury following road traffic accident (Kareem, 2003).

2.2 PATHOPHYSIOLOGY

Primary brain injury is due to acceleration and deceleration of the head or the mechanical impact to scalp, skull, brain matter and blood vessels at the moment of injury in a focal, multi focal or diffuse pattern. The physical force at the time of injury commonly causes contusion and laceration of the brain which in contact with the inner skull surface mainly at the temporal pole and orbital surface of frontal lobe. In addition the diffuse axonal injury that occurs at the subcortical surface and deeper structure is commonly seen in

severe head injured patients. Extradural or epidural hematoma following head injury commonly occurs in the convexity of brain and is mostly due to torn middle meningeal artery or bleeding from the skull fracture. Subdural hematoma is usually due to tearing of bridging vein or laceration of the cortical brain and it is not uncommon that subdural hematoma may associate with underlying contusion. Intra parenchymal hematoma which manifests as a sizeable clot in the brain is commonly resulted from brain laceration. Intracranial hematomas may be multiple dependants on the site and velocity of impact. All this hematoma presented as mass lesion that contribute to the raised intracranial pressure and the sizeable clot need surgical evacuation.

The fundamental pathophysiological process following primary impact that cause secondary brain damage is the development and propagation of vicious cycle of brain edema with subsequent increase in intracranial pressure (ICP), reduction in blood supply and oxygen delivery causing cerebral ischemia, energy failure and further edema enhancing the primary brain damage and poor outcome. Other factors that enhance the brain damage following primary impact are pyrexia, anemia, vasospasm, seizure, infection and hyponatremia. Autopsy study has demonstrated ischemic brain damage in 80-92% of patients who died following head injury (Pillai *et al.*, 2004, Graham and Adams, 1971).

The process that occurs at cellular level in the brain following severe head injury includes the release of amino acids, glutamate and aspartate, free radicals, lactate and hydrogen ion above toxic level. These processes eventually result in influx of calcium ions into cells and subsequently lead to cell swelling. In addition, the damage of blood brain barrier leads to increase transcapillary exudation. Therefore the presence of intracranial

hematoma with combination of cytotoxic and vasogenic cerebral edema distort the anatomy of brain and blood vessels which worsen the cerebral hypoxia, responsible for inflammation and micro vascular dysfunction. The extra vascular blood also releases free radicals, predisposing to large vessel spasm hence further reduction in cerebral blood flow, hypoperfusion and ischemia (Ikeda and Long, 1990, Unterberg *et al.*, 1988).

2.3 COMPUTED TOMOGRAPHY (CT) SCAN IN SEVERE HEAD INJURY.

The standard non contrast enhanced computerized tomography (NCECT) scanning of the brain is an invaluable diagnostic tool and help in the management of severe head injury. A focal mass lesion such as epidural, subdural, intra parenchymal hematoma, brain contusion and associated lesions can be clearly demonstrated on CT brain and emergency evacuation of hematoma can be planned accordingly. The progressive enlargement of hematoma and swelling of the brain tissue results in shift of the brain and displaced the midline structure. The mass lesion in the supratentorial compartment may cause brain herniation through the tentorial herniatus. The herniation can be demonstrated on the CT scan as the effaced basal cistern.

A classification of brain injury according to the structural changes demonstrated on CT scan has been published based on the status of basal cistern, midline shift and the hematoma of less than 30mls (Marshall *et al.*, 1992). The system defines brain injury into 4 categories from DI I, II, III and IV. A modified Marshall system has included DI V and VI which includes the non evacuated mass of more than 30mls and evacuated mass lesion respectively. Midline shift and effacement of basal cistern as demonstrated on computed tomography correlate with the degree of cerebral edema and are predictors of lowering

the cerebral compliance and perfusion pressure along with developing intracranial hypertension and the eventual outcome of severely head injured patients (Toutant *et al.*, 1984, Miller *et al.*, 2004).

2.4 EVACUATION OF MASS LESION.

Craniotomy or craniectomy and evacuation of blood clot remove significant amount of mass lesion, such as epidural, subdural, intra parenchymal hematoma and contusion, hence restoring the brain anatomy, reducing intracranial volume and ICP with eventual improvement of the outcome (Zhang *et al.*, 2001, Schneider *et al.*, 2002). The removal of this mass lesion is crucial to prevent secondary brain damage. In addition, surgical evacuation of hematoma may result in reduced cerebral edema and minimize microvascular dysfunction and large vessel spasm. In the extreme cases of refractory intracranial hypertension, decompressive craniectomy has been shown to normalize cerebral compliance and pressure volume index in patients who survive severe head injury (Abdullah *et al.*, 2005).

2.5 INTRACRANIAL PRESSURE

Monitoring of ICP in severe head injury is recommended and associated with decreased death rate (Tjuvajev *et al.*, 1990). ICP monitoring in patients with GCS score of 8 and below forms the guideline in the management of severe head injury and is widely practiced throughout the world. One can monitor the ICP continuously and management of severe head injury is done accordingly. In addition to the intracranial hematoma that formed following head injuries, the other factors that contribute to the raised ICP are

cerebrovascular congestion, brain edema and development of acute hydrocephalus. ICP was found to be an independent factor that significantly related to the outcome. Intracranial pressure varies following head injury. Normal ICP in adult during resting at horizontal position ranges between 7 and 15 mmHg. A sustained elevation of ICP of more than 20 mmHg in severe head injured patient is considered abnormal and treatment should be initiated before the ICP reaches 25 mmHg (Ratanalert *et al.*, 2004). Treatment protocol in severe head injury should aim to maintain ICP below 20 mmHg (Juul *et al.*, 2000). ICP between 20 and 40 mmHg is considered moderate and may result in poor outcome and elevation of ICP more than 40 mmHg is severe and usually fatal (Lundberg, 1960). The increased ICP in cases of diffuse injury without associated mass lesion is not as common as other severe head injured patients which harbor mass lesion (Lee *et al.*, 1998). Evacuation of mass lesion thus may result in normal ICP provided the secondary insults and hydrocephalus are well controlled.

2.6 CEREBRAL PERFUSION PRESSURE

CPP measurement can be obtained manually by subtracting the ICP from the MAP and in usual circumstances is calculated by the patient's monitoring device. Therefore CPP is directly influenced by blood pressure and ICP. Arterial hypotension due to hypovolemia, cardiodepressant drug and septicemia and those that increase ICP as mentioned above will compromise CPP. Cerebral perfusion pressure targeted therapy has been shown to produce better survival outcome in severe head injury (Huang *et al.*, 2007, Juul *et al.*, 2000). Maintenance of base line CPP between 60 to 70 mmHg reduces the possibility of cerebral ischemia. This CPP oriented protocol generally requires vasopressors. High

CPP reduces the ICP as long as the cerebral autoregulation remains intact. Autoregulation of cerebral blood flow is the mechanism by which a constant and steady blood flow is delivered to the brain over a certain range of MAP or CPP. In a normal condition, cerebral pressure autoregulation remain intact as long as the CPP is kept between 50 mmHg and 140 mmHg. The study of relationship between MAP, CPP, ICP, transcranial Doppler peak flow velocity (TAP) and brain tissue oxygenation reactivity (Ptio2) during gradual increasing of CPP using noradrenaline infusions in head injured patients demonstrated a plateau TAP and Ptio2 at CPP between 70-90 mmHg. Therefore if the cerebral vessels become non reactive and fail to autoregulate the blood flow following head injury, maintenance of high CPP elevates the cerebrovascular hydrostatic pressure. The condition may result in hyperemia, significant vasogenic edema and addition of intracranial volume, thus reducing cerebral compliance and further increase in ICP. Hence it was suggested by a study that patients with head injury may benefit from CPP of slightly less than 60 mmHg (Elf *et al.*, 2005).

2.7 CEREBRAL COMPLIANCE

Following head injury, the ability of the intracranial compartment to counteract to the additional volume is an important factor in the development of increased pressure (March, 2000, Salci *et al.*, 2006). This is known as cerebral compliance (ΔV=ml/ΔP=mmHg) or its inverse, elastance or brain stiffness. Cerebral compliance is defined as the capability of the intracranial compartment and craniospinal axis to buffer the added volume before the definite increase of intracranial pressure. Cerebral compliance is calculated as the ratio of added volume to the ICP changes resulting from

volume addition: compliance = $\Delta V/\Delta P = \Delta V$ / (Pmax - Pbase) in which V = volume, P = pressure, Δ = change, Pmax = highest ICP and Pbase = baseline ICP (Yau *et al.*, 2000) (Figure 2.7a).During the compensated phase of increasing volume the increased intracranial pressure is small but once it reaches the decompensated phase the ICP rises steeply and produce exponential curve. Normal physiological values of cerebral compliance using calculation method by Marmarou is reported between 0.25 to 1.5 ml/mmHg (Portella *et al.*, 2005). The cerebral compliance less than 0.5 ml/mmHg is measured low whereas more than 1 ml/mmHg are considered high. Compliance reduction below 0.5 ml/mmHg is the verge for ICP instability indicating that the patient is at risk of increased ICP (Piper *et al.*, 2000). The exact vascular and non vascular mechanisms that influence the cerebral compliance following traumatic brain injury are poorly understood. Several factors have been considered which include the cerebral blood volume (CBV), systemic blood pressure (MAP) and cerebral perfusion pressure. Intact cerebral autoregulation is important to control the variations of CBV, MAP and CPP and indirectly intracranial compliance.

Compliance = $\Delta V/\Delta P = \Delta V/$ (Pmax - Pbase)

V = volume, P = pressure, Δ = change, Pmax = highest ICP and Pbase = baseline ICP

Figure 2.7a: The method for calculation of cerebral compliance.

2.8 MEASUREMENT OF ICP AND COMPLIANCE.

ICP can be measured with an intraventricular catheter connected to a pressure transducer, microsensor transducer or fibreoptic transducer. The first method is the gold standard to measure ICP but the last two options have the advantages of ease of insertion into the brain parenchyma or subdural space in patient with compressed ventricle.

Pressure Volume Index (PVI), introduced by Marmarou and Volume Pressure Response (VPR) of Miller are the methods developed to measure intracranial compliance clinically (Marmarou *et al.*, 1975, Marmarou *et al.*, 1978, Miller and Leech, 1974). The methods use a known volume of fluids injected into or withdrawn from the CSF space manually and the ICP is measured before and after the changes in CSF volume. The method consist of injecting or withdrawing 1-2ml of CSF (V) and ICP prior to procedure (Po) and following the procedure (Pm) are recorded and the PVI is calculated as V/log (Po/Pm) (Maset *et al.*, 1987) (Figure 2.8a).The calculated PVI using Marmarou's technique is dependant upon the opening pressure which is affected by the hydrostatic pressure (Raabe *et al.*, 1999). PVI or VPR assumes a linear mono exponential relationship between volume and pressure and is considered a single index that characterizes the intracranial volume-pressure relationship. During continuous measurement, PVI of 0.5 ml was taken as normal value (Abdullah *et al.*, 2005). A higher the PVI value suggests better brain compliance. Other method to measure intracranial compliance is the analysis of ICP waveform but this has yet to be validated clinically (Avezaat *et al.*, 1979, Avezaat and van Eijndhoven, 1986, Robertson *et al.*, 1989).

Continuous and automated measurement of ICP and compliance can be made using Spiegelberg Brain Pressure monitor and compliance monitor; GmbH & Co. The

monitoring device comprises of a double lumen intra ventricular catheter. One lumen has an air pouch balloon mounted near the tip of the catheter and the other lumen is filled with fluid and ends directly into the CSF compartment. The lumen with balloon mounted to the end is able to measure ICP as well as the added volume during measurement of brain compliance. The lumen that connects directly to the CSF space can be used to measure the ICP simultaneously during inflation or withdrawal of specific volume of air in the balloon. In addition, the latter can therapeutically drain the CSF during the period of elevated ICP. The Spiegelberg compliance monitor calculates intracranial compliance ($C=\Delta V/\Delta P$) from a moving average of small ICP perturbations (ΔP) resulting from a sequence of up to 200 cycles of added volume ($\Delta V = 0.1$, total $V = 0.2$ ml) made into a double lumen intra ventricular balloon catheter. Once a stable average has developed, the monitor will show minute by minute measurement of intracranial compliance (Piper *et al.*, 2000). This method has the advantages of less inoculation of infective organisms into the brain and CSF spaces and is more reliable due to fixed volume of inflated air. Clinical studies had a satisfactory agreement on the use of Spiegelberg monitoring device as a gold standard method to measure compliance, PVI and ICP (Piper *et al.*, 2000, Yau *et al.*, 2000, Yau *et al.*, 2002a).

$$PVI = V/\log (Po/Pm)$$

V = CSF volume, Po = ICP prior to procedure,

Pm = ICP following the procedure

Figure 2.8a: The method for calculation of PVI

2.9 SURVIVAL OUTCOME

Glasgow Outcome Scale (GOS) is used to assess the survival of patients after severe head injury (Jennett and Bond, 1975, Miller *et al.*, 2005). Favorable outcome includes those patients who made good recovery or survived with moderate disability. This group of patients is independent and able to resume their normal daily life. On the other hand unfavorable outcome are patients who did not survive or remain in persistent vegetative state or severely disabled. Generally, persistently high pathological ICP with low cerebral perfusion pressure are associated with poor survival outcome. A trend of low post traumatic cerebral compliance is associated with higher risk of increased ICP and unfavorable survival outcome (Maset *et al.*, 1987, Tans and Poortvliet, 1983, Stocchetti *et al.*, 1993).

3. OBJECTIVES

3.1 GENERAL OBJECTIVES

The general objective of this study was to observe the associations between brain compliance (Cl) with intracranial pressure (ICP) and cerebral perfusion pressure (CPP) during therapeutically induced or natural fluctuations following surgery to evacuate intracranial mass lesion in severely head injured patients. Early intervention can be initiated once abnormal value or trend is detected with the standard protocol of management of severe head injury, hence preventing further reduction of cerebral compliance, CPP and elevation of ICP which result in poor outcome of severe head injury.

3.2 SPECIFIC OBJECTIVES

1) To determine whether cerebral compliance, ICP and CPP correlate with post operative computed tomography (CT) indicating status of cerebral physiology after surgery.
2) To identify factors that influences the ICP, CPP and cerebral compliance after surgical evacuation of mass lesion.
3) To determine association between cerebral compliance, ICP and CPP values and survival outcome.

4. RESEARCH METHODOLOGY

This was a prospective cohort study, carried out in the Neuro Intensive Care Unit, Department of Neurosciences, Hospital Universiti Sains Malaysia, Kubang Kerian Kelantan. The study was approved by local ethics committee with reference letter: USM/PPSP®/Ethics Com./2005 (146.4[1]). The study was conducted between November 2005 and September 2007 with a total of 30 patients included in the study.

Severe head injured patient was defined as a patient who was admitted or deteriorated preoperatively with Glasgow Coma Score (GCS) of ≤ 8 cumulative points after adequate resuscitation. The inclusion and exclusion criteria were as follows:

Inclusion and exclusion criteria:

a) Inclusion criteria

1) Consented by next of kin or relatives.
2) Patient age between 10 to 65 years old
3) Acquired traumatic head injury.
4) An abnormal CT scan brain that requires surgery to evacuate a mass lesion.
5) Post operative ICP and craniospinal compliance monitoring.

b) Exclusion criteria

1) Bilateral fixed and dilated pupil.
2) Coagulopathy and bleeding diathesis.