# VALUE OF SHOCK INDEX IN RISK STRATIFICATION OF ILLNESS SEVERITY AMONG PATIENTS IN EMERGENCY DEPARTMENT

BY

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# LIST OF ABBREVIATIONS

| | | |
|---|---|---|
| HUSM | : | Hospital Univesiti Sains Malaysia |
| HKL | : | Hospital Kuala Lumpur |
| ED | : | Emergency Department |
| TPR | : | Total peripheral resistance |
| ADH | : | Antidiuretic hormone |
| HR | : | Heart rate |
| SBP | : | Systolic blood pressure |
| APACHE II | : | Acute Physiology and Chronic Health Evaluation |
| MPM | : | Mortality Probability Model |
| RAPS | : | Rapid Acute Physiology Score |
| GCS | : | Glasgow Coma Scale |
| REMS | : | Rapid Emergency Medicine Score |
| BP | : | Blood pressure |
| ROC | : | Receiver operating characteristic |
| AUC | : | Area under curve |

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**ABSTRAK**

**KAJIAN PENGGUNAAN NILAI SHOCK INDEX DALAM MENGKATEGORIKAN KETENATAN PENYAKIT DIKALANGAN PESAKIT-PESAKIT DI JABATAN KECEMASAN.**

Stratifikasi risiko dikalangan pesakit adalah sangat penting bermula apabila pesakit datang ke Jabatan Kecemasan. Masalah pesakit yang datang boleh di dalam pelbagai bentuk dimana ada pesakit yang kelihatan normal dengan tanda-tanda penting yang normal tetapi keadaannya boleh menjadi teruk dalam masa yang tertentu. Kita tidak boleh terlepas pandang kepada pesakit ini kerana kelewatan dalam memberikan mereka rawatan akan menyebabkan kadar mortaliti dan morbiditi mereka akan meningkat.

Kajian ini telah dilakukan di dua buah pusat iaitu Hospital Univesiti Sains Malaysia (HUSM) dan Hospital Kuala Lumpur (HKL). Pesakit yang berumur 18 tahun dan keatas yang memenuhi cirri-ciri yang telah ditetapkan dimasukkan ke dalam kajian ini. Bacaan tanda-tanda penting direkodkan oleh seorang pemerhati dan rawatan pesakit diteruskan mengikut standard yang telah ditetapkan. Pemerhati merekodkan kemasukan pesakit ke wad selepas rawatan di Jabatan Kecemasan. Nilai Shock Index diperolehi selepas pembolehubah yang penting dikenalpasti. Dengan menggunakan lengkungan ROC, nilai minima dikenalpasti. Nilai maksima ialah 0.9 dimana nilai ini telah pun disahkan. Dengan menggunakan dua nilai ini, kita boleh merangka julat nilai Shock Index.

Seramai 48 pesakit termasuk di dalam kajian ini. Pembolehubah yang penting adalah tekanan darah sistolik ($p=<0.001$), tekanan darah diastolic ($p=0.006$), kadar denyutan

jantung (p=<0.001) dan kadar pernafasan (p=0.004). Kadar denyutan jantung merupakan pembolehubah yang menunjukkan area under curve (AUC) yang paling penting dimana nilainya ialah 0.884. Oleh itu, nilai minima Shock Index ialah 0.74 (sensitivity 80%, 1-specificity 74%). Nilai maksima ialah 0.9.

Dengan itu, julat nilai Shock Index ialah <0.74 sebagai kumpulan berisiko rendah, 0.75 – 0.9 sebagai kumpulan berisiko pertengahan dan >0.9 sebagai kumpulan berisiko tinggi.

## ABSTRACT

### VALUE OF SHOCK INDEX IN RISK STRAFICATION OF ILLNESS SEVERITY AMONG PATIENTS IN EMERGENCY DEPARTMENT

Risk stratification of patient is very important, starting at the time patient arrive in Emergency Department. Wide range of patient's presentation sometimes can make them appear normal where their vital signs are normal but they actually deteriorate in time. We should not miss this group of patient as the mortality and morbidity rate will increase.

Objectives of this study is to develop range of Shock Index value to risk stratify patient into 3 different groups which are low risk group, intermediate risk group and high risk group.

This study was done in two centers which were Hospital Universiti Sains Malaysia (HUSM) and Hospital Kuala Lumpur (HKL) from June 2008 till November 2008. Patients older than 18 years old that came to Emergency Department of these two centers and meet the inclusion and exclusion criteria were enrolled. The first vital signs of patient were recorded by single reviewer and proceed with standard care of management. Then the disposition of patient was recorded by the same reviewer. Significant variables were initially identified. Then based on these values the range of Shock Index values were developed using the receiver operating characteristic (ROC) curve. A minimum value was identified, while the maximum value of 0.9 which has been extensively validated was applied.

Total of 48 patients enrolled in this study. The significant parameters were systolic blood pressure (p=<0.001), diastolic blood pressure (p=0.006), heart rate (p=<0.001) and respiratory rate (p=0.004). Heart rate was the variable with most significant area under the curve (AUR) in ROC curve which are 0.844. From there, minimum value of Shock Index obtained was 0.74 (sensitivity 80%, 1-specificity 74%).

Thus, range of Shock Index values are <0.74 as low risk group, 0.75 – 0.9 as intermediate risk group and >0.9 as high risk group.

# 1. INTRODUCTION

Emergency Department (ED) cares for all patients presenting with wide range of complaints. It is ranging from non-critical to critically ill condition that needs immediate identification in order to deliver appropriate management. They will come with variety of medical background that will influence their body response towards any illness. Because of that, some patient might present with subtle signs that can be easily missed and appropriate management will be delayed. Delay in initiating proper management will definitely affect patient outcome.

In ED, patients are triaged according to the severity of illness. Triaging is very important to sort patients requiring immediate attention or critically ill from those who can wait or not critical. There are various types and methods to triage patient (Moskop JC, 2007). Conventionally in ED, vital signs are one of the clinical tools used in triaging patient. The conventional vital sign parameters are blood pressure, heart rate, temperature, respiratory rate and oxygen saturation.

Many of time, patients present to ED during early phase of illness where the conventional vital sign parameters are normal. Despite normal vital signs, physiologically they are actually in compensatory phase. Since disease process is dynamic, these patients may eventually deteriorate and physiologically decompensate. Such groups of patient are easily missed and their clinical outcome definitely will be affected if fail to be recognized early.

For example, pregnant patients are usually young and healthy. Their physiologic reserve due to hormonal changes able to compensate any acute blood loss, thus they might present with normal conventional vital signs (Birkhahn *et al.*, 2003). They might even be evaluated as outpatient and will deteriorate later and the mortality will be increased. We might miss this group of patient just because they present with normal vital signs.

All unstable patients ideally should be recognized as early as upon arrival to ED. These patients respond very well with early emergent and appropriate treatment in the ED. Such aggressive treatment should be continued at ward with definitive care. However, occasionally disposition of patients are delayed due to access block or inappropriate because of limited bed availability especially in critical care unit or high dependency ward. In such condition of limited resources, we need a clinical tool to risk stratify patient into high, intermediate or low risk group to aid appropriate disposition.

Triage system has initiated the sorting of patient to appropriate priority of care but is it enough for us to make proper disposition. Are we missing patients that are potentially unstable initially triaged as yellow or green whom actually require high dependency monitoring during definitive treatment? Is the disposition of patient with stable vital sign always appropriate?

Answers to all of the above questions are all pertaining to risk stratification of patients at disposition phase in the ED. Appropriate risk stratification allows appropriate selection of level of monitoring to selected patients. Such stratification criteria also allow early decision-making of ward admission. Indirectly overcrowding of patients in ED from delayed disposition decision can be reduced.

There are limited risk stratification scoring systems that can be used in ED to help in patient disposition. Furthermore, in our local setting these scoring systems are not widely use. Conventional vital sign parameters are still the main clinical tool for risk stratification of patients presented to ED. Using the conventional vital sign, shock index can be calculated and used as a clinical parameter to determine the severity of illness. It has been studied in few groups of patients eg: 1st trimester pregnancy, trauma and gastrointestinal hemorrhage. Elevated value correlates with reduction in left ventricular stroke volume and associated with higher mortality (Rady *et al.*, 1992b).

In this study, the main objective is developing range of Shock Index values that can be used to risk stratify patient into low, intermediate or high risk group. It is easier to prioritize management of patients if they can be grouped into different group of illness severity from the initial point of treatment. Patients in the low risk groups may be discharged home or require only normal ward beds whereas the high risk group would require close monitoring beds such as the intensive care unit. Shock Index has such risk stratification potential. This study hopes to

expand the use of shock index not only as a sensitive indicator for left ventricular dysfunction and hypovolemia but also as a risk stratification tool to aid disposition process. In this study we will present range of Shock Index values compared to patient disposition level. Further study is required to verify and validate the range of Shock Index values found in this study.

Maybe in future, this range of Shock Index value can be use as one of parameters in scoring system that can be used specifically in ED. With the use of shock index in ED, hopefully the length of stay of patients in ED will be shortened, early decision of admission and proper level of observation can be determined in the limited of hospital facilities.

## 2. LITERATURE REVIEW

Presentation of patient to ED can be divided into 2 major groups which are trauma and non-trauma cases. Management approach for both group differ but they share the same basic crucial fundamental concept of early identification and early therapy to reduce patient mortality and morbidity.

In year 2002, health statistics report by the Ministry of Health Malaysia showed that trauma cases had been contributed up to 9.16% admission to ward and 6.76% death in government hospital (Table 1 & Table 2) (MOH, 2003).

Table 2.1: Principal causes of admission in government hospital in Malaysia 2002.

| | | |
|---|---|---|
| 1 | Normal delivery | 18.9% |
| 2 | Complication of pregnancy | 11.84% |
| 3 | Accidents | 9.16% |
| 4 | Diseases of the circulatory system | 6.94% |
| 5 | Diseases of the respiratory system | 6.61% |
| 6 | Perinatal condition | 5.62% |
| 7 | Diseases of the digestive system | 4.87% |
| 8 | Ill-defined conditions | 3.57% |
| 9 | Diseases of the urinary system | 3.49% |
| 10 | Malignant neoplasms | 2.62% |

Table 2.2: Principal causes of deaths in government hospitals in Malaysia 2002

| | | |
|---|---|---|
| 1 | Heart diseases & diseases of pulmonary circulation | 15.99% |
| 2 | Septicaemia | 14.51% |
| 3 | Malignant neoplasms | 9.16% |
| 4 | Accidents | 6.76% |
| 5 | Perinatal condition | 5.56% |
| 6 | Pneumonia | 4.98% |
| 7 | Cerebrovascular diseases | 4.48% |
| 8 | Diseases of the digestive system | 4.38% |
| 9 | Kidney diseases | 3.72% |
| 10 | Ill-defined conditions | 2.74% |

Major cause of mortality in trauma is related to inadequate intravascular volume contributed by blood loss, myocardial failure or distributive loss. It is therefore very important to identify early significant loss of volume in trauma patients and deliver the appropriate treatment as early as possible.

## 2.1 RESPONSES TO HEMORRHAGE

Loss of intravascular volume triggers a predictable systemic response mediated by both local and neuroendocrine systems (Runciman WB, 1984). Acute blood loss will lead to decrease in mean arterial pressure causing decrease in venous return and thus reduce in cardiac output. As blood loss continues, the mean

arterial pressure will eventually drop and stimulate the baroreceptor reflex, renin-angiotensin II-aldosterone system and capillaries to maintain perfusion to vital organ including brain, heart and kidney.

Baroreceptor reflex will sense the drop in mean arterial pressure and send the information to the medulla via carotid sinus nerve. Sympathetic activity to the heart and blood vessels will increase while parasympathetic outflow to heart decreases. The four consequences of these autonomic reflexes are **increase heart rate (tachycardia), increase contractility, increase total peripheral resistance (TPR)** and **constriction of the veins** (reduce unstressed volume, increase venous return and increase stressed volume). Each cardiovascular response aimed to increase the mean arterial pressure (Costanzo, 2004).

Another set of compensatory response to decrease in mean arterial pressure are renin-angiotensin II-aldosterone system. When mean arterial pressure reduce, renal perfusion pressure decreases and stimulate the secretion of renin thus increases the production of angiotensin I which converted to angiotensin II. It has 2 actions which are causes arteriolar vasoconstriction and stimulate the secretion of aldosterone which circulates to the kidneys and causes increase in reabsorption on $Na^+$. By increasing the total body $Na^+$ content, ECF will increase thus increase the blood volume (Costanzo, 2004).

The compensatory responses to hemorrhage include changes in the Starling forces across capillary walls. Increased sympathetic outflow to blood vessels and increased angiotensin II produce the arteriolar vasoconstriction. As a result of this vasoconstriction, there is a decrease in capillary hydrostatic pressure which opposes filtration out of the capillary and favors absorption. Antidiuretic hormone (ADH) is also secreted in response to decrease in blood volume. It has 2 actions which are **increases water reabsorption** by the renal collecting ducts via $V_2$ receptors and causes **arteriolar vasoconstriction** via $V_1$ receptors. Both actions will help in restore the blood volume.

Other changes are including the stimulation of the chemoreceptor due to hypoxemia following a hemorrhage. It will sense the decrease in partial pressure of the oxygen and response by increasing sympathetic outflow to the blood vessels. It will cause vasoconstriction, increase total peripheral resistance and thus increase mean arterial pressure. If cerebral ischemia occur, local $P_{CO2}$ increase and decrease in pH will activate chemoreceptors in the medullary vasomotor center to increase the sympathetic outflow to blood vessels resulting in peripheral vasoconstriction, increase TPR and increase the mean arterial pressure (Costanzo, 2004).

## 2.2 BIPHASIC HEART RATE RESPONSE TO HEMORRHAGE

A series of studies in health volunteers of the effects of simple blood loss demonstrated a slight tachycardia (seldom exceeding 100bpm) and well maintained blood pressure up to losses of approximately 1 liter (R.V *et al.*, 1941). However when losses more than 1 liter, syncope with a fall in blood pressure, bradycardia and vasodilation in skeletal muscle were frequently recorded (Barcroft H and O.G, 1945). These finding suggested a biphasic heart rate response to blood loss which is tachycardia followed by bradycardia (Secher and Bie, 1985). In explaining the biphasic response to blood loss, 3 reflexes need to be considered which are the arterial baroreceptor reflex, chemoreceptor reflex and cardiac C-fiber activation.

### 2.2.1 ARTERIAL BARORECEPTOR

This reflex is thought to be responsible for the maintenance of arterial blood pressure following the loss of 10-15% of the blood volume (Cowley A W *et al.*, 1973). The baroreceptor endings are located in the medio-adventitial border of the arterial wall mainly in the aortic arch and carotid arch. These baroreceptors are slow-adapting mechanoreceptors which response to the degree of stretch of the arterial wall produced by the intraluminal pressure rather than to the intraluminal pressure itself (J.E, 1971b).

Furthermore, the baroreceptors are rate sensitive and can response to the rate of change of arterial pressure as well as to its absolute level (J.E, 1971a). This means that the baroreceptors can respond to a change in pulse pressure as well as to changes in mean pressure. Thus, as pulse pressure reduces during hemorrhage there is a decrease in baroreceptor afferent activity even in the absence of fall in mean arterial pressure. This change in baroreceptor afferent activity will be sent to the brain via vagus nerve (from aortic arch baroreceptor) and sinus nerve, a branch of glossopharyngeal nerve (from the carotid sinus baroreceptor) (H, 1976). The efferent limb of the baroreceptor reflex is carried in the vagus nerve and the sympathetic nerve to the heart and in the sympathetic vasoconstrictor nerves to the blood vessels (H, 1976). When the baroreceptor activity is reduced due to acute hemorrhage, this will cause reflex withdrawal of vagal-cardiac activity and an enhancement of the sympatocardiac activity. This will lead to tachycardia and vasoconstriction causing increase in total peripheral resistance(H, 1976). As a consequences of these hemodynamic changes, any hemorrhage-induces falls in the arterial blood pressure are minimized or prevented up to 10 -15% of blood loss. Thus, arterial baroreceptor reflex serves to maintain blood flow to the vital tissues (e.g: brain, heart and kidney) at the expense of flow to the other nonvital organ (e.g: skeletal muscle, gut and skin) at least in the short period of time. However, as blood loss exceeds 20% of blood volume, blood pressure falls

dramatically. This is not because of a reflex possibly the activation of cardiac vagal C-fiber afferents (Little *et al.*, 1984).

### 2.2.2 CARDIAC VAGAL C-FIBER

The cardiac C-fibers form the afferent pathway from a group of receptors located mainly in the left ventricular myocardium. These receptor can be activated by mechanical or chemical (e.g: prostaglandin $E_2$) (Baker D.G *et al.*, 1979). Activation of this receptors can lead to a profound reflex bradycardia, hypotension and reduction in skeletal muscle as well as renal vascular resistance and this reflex referred as 'cardiac reflex' (Daly M.de B *et al.*, 1988). The bradycardia results from increased vagal efferent activity to the heart, while the reduction in vascular resistance is because of a withdrawal of sympathetic vasoconstrictor tone (Daly M.de B *et al.*, 1988). Thus, this 'depressor reflex' associated with severe hemorrhage can masked the conventional vital signs assessment in early phase of acute hemorrhage.

### 2.2.3 ARTERIAL CHEMORECEPTOR REFLEX

The 3rd reflex in the cardiovascular response to hemorrhage is the arterial chemoreceptor reflex. The arterial chemoreceptors are found in the carotid body and aortic body close to the carotid sinus and aortic arch. They respond to the changes in oxygen tension, a fall in oxygen tension increasing chemoreceptor afferent activity. In addition, increases in carbon dioxide tension and falls in the arterial blood pH increase the arterial chemoreceptors to hypoxia. Stimulation of the arterial chemoreceptors produces an increase in respiration while the primary cardiovascular effects are a vagally-mediated bradycardia and a vasoconstriction resulted from increased sympathetic vasoconstrictor tone (Daly M.de B, 1983). However, this response will be modified by the increased in respiratory activity, which tends to inhibit both the vagal activity to the heart and the sympathetic vasoconstrictor activity (Spyer K.M, 1984).

Following a severe hemorrhage, the arterial chemoreceptors are activated as a result of a reduction in blood flow through the carotid and aortic bodies secondary to the fall in arterial blood pressure and to the sympathetic vasoconstriction in the bodies mediated by local release of noradrenaline and its co-transmitter neuropeptide Y (Potter E.K and D.I, 1987). Therefore, during the