# Fitness Between The Value of Etco<sub>2</sub> Rate with The Valance of Paco<sub>2</sub> in Hemodynamic Repair on Severe Brain Injury Patients

\*Muhammad Isya Firmansyah<sup>1</sup>, Nanik Setijowati<sup>2</sup>,Dwiwardoyo Triyuliarto<sup>3</sup>, A.A.Asmoro<sup>4</sup>,Muhammad Istiajid Edy Santoso<sup>5</sup>

<sup>1</sup>Resident of Emergency Medicine, Faculty of Medicine, Universitas Brawijaya, Indonesia
<sup>2</sup>Staff of Public Health Science, Faculty of Medicine Universitas Brawijaya, Indonesia
<sup>3</sup>Staff of Emergency Medicine, Faculty of Medicine, Universitas Brawijaya, Indonesia
<sup>4</sup>Staff of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya, Indonesia
<sup>5</sup>Professor of Neurosurgery, Faculty of Medicine, Universitas Brawijaya, Indonesia
\*Corresponding Author: Muhammad Isya Firmansyah

**Abstract:** This research is to determine the correlation and suitability between clinical diagnosis and the value of  $EtCO_2$  with the  $PaCO_2$  value at 15 minutes and 120 minutes later and to know the hemodynamic improvement and outcome in severe brain trauma patients after the target of  $PaCO_2$  was reached 120 minutes later and at day 6. This study was an observational analytic study to assess the correlation and suitability between the values of  $EtCO_2$  and  $PaCO_2$  of severe brain trauma patients in ER RSSA. We collected patient data for 5 months (October 2016 until February 2017) at ER RSUD Saiful Anwar Malang, Indonesia. The results indicated that there was no correlation between clinical diagnosis and the value of  $EtCO_2$  with the  $PaCO_2$  value at 15 and 120 minutes, there was a correspondence between the  $PaCO_2$  value at the 15th and 120th minutes, there was haemodynamic improvement at 15-120 minutes, although there was a significant improvement in RR At 60-120 minutes, the target value of  $PaCO_2$  can improve the patient's haemodynamics at 120 minutes in marginal margin and there was no significant difference in outcome on day 6 based on the value of  $EtCO_2$  and  $PaCO_2$  and  $PaCO_2$  can improve the patient's haemodynamics at 120 minutes in  $PaCO_2$  at 120 minutes.

Keywords: Severe brain injury, EtCO<sub>2</sub>, PaCO<sub>2</sub>, hemodynamics, outcome.

Date of Submission: 02-08-2017

Date of acceptance: 08-08-2017

## I. Introduction

Handling of the airway is the basis of the determinants of successful resuscitation. Each emergency physician has a primary obligation to maintain airway patency and must master all airway handling techniques. Intubation is one of the procedures performed to secure the airway in patients with severe brain injury. The indications of intubation in the ER are hypoxia or hypercarbia repair, threatening hypoventilation, maintaining airway patency, where emergency medications are included(Vissers & Danzl, 2011).

EtCO<sub>2</sub> is the partial pressure or maximum concentration of carbon dioxide (CO<sub>2</sub>) at the expiratory patient, where the unit is mmHg. Normal values are 5% to 6% CO<sub>2</sub> where equal to 35-45 mmHg. CO<sub>2</sub> represents cardiac output (CO) and pulmonary blood flow where the flow is transported by the right venous system of the heart and pumped into the lungs by the right ventricle of the EtCO<sub>2</sub> detection device can also be used to estimate the levels of PaCO<sub>2</sub> in the blood without having to do blood gas analysis so it is a monitor tool that is more non-invasive, cost-effective and results that can quickly be known every time. But the accuracy of the EtCO<sub>2</sub> detection tool in assessing PaCO<sub>2</sub> levels is sometimes questionable especially in critical patients (Grmec & Klemen, 2001). PaCO<sub>2</sub> is the pressure caused by CO<sub>2</sub> dissolved in the blood. Normal value of PaCO<sub>2</sub> referred to as hypokapnia, means hyperventilation due to respiratory stimulation, if high PaCO<sub>2</sub> (hypercapnia) means alveolar ventilation failure (hypoventilation). At the beginning of the increase in PaCO<sub>2</sub> the respiratory system will be aroused to lower the PaCO<sub>2</sub>. Conversely, if PaCO<sub>2</sub> is very high it will suppress the respiratory system (Visser & Danzel, 2011).

The characteristic of brain circulation is that blood flow of the brain dynamically adjusts to protect the blood from the brain from changes in perfusion pressure, the blood circulation of the brain remains constant at the systemic blood pressure (cerebral autoregulation). The increase and decrease in  $PaCO_2$  values will increase and decrease cerebral blood flow by vasodilation and brain vasoconstriction, independent cerebral autoregulation. In most patients with brain injury there is damage to autoregulation in the bloodstream of the brain, whereas in severe brain injury  $CO_2$  damage occurs during the initial phase, in patients with severe head injury can occur acidosis due to lactate production due to glycolysis process so high mortality / outcome and

also duration. The first hour maintains the value of  $PaCO_2$  and the value of  $EtCO_2$  is very crucial (Ni Made Ayu Apsari Dewi, 2013).

In resuscitation situations, pulmonary blood flow determines oxygenation and CO excretion. Examination of blood gas analysis 10 to 15 minutes after administration to avoid inadequate perfusion resulting in lactic acidosis. Examination of blood gases was re-examined several hours after resuscitation and BGA was usually taken 2 to 4 hours post resuscitation (Menzel et al., 2001). At ER RS Saiful Anwar in airway management, intubation often becomes one of the selected measures in the management of the airway, especially in severe brain injury patients. Selection of severe brain injury patients in this study is due to eliminate the bias such as lung abnormalities, heart abnormalities.

In determining the level of  $PaCO_2$  in which the mortality rate of severe brain injury patients worldwide is 76% in 6 days (Rondinna & Videtta, 2005), blood gas analysis is the best test but the use of blood gas analysis has drawbacks such as arterial blood sampling Is invasive, but it takes a long time about 3-4 hours in getting the results and the cost is quite expensive.

#### II. Research Method

The study design was an observational analytic study to examine the correlation and suitability between the value of  $EtCO_2$  and  $PaCO_2$ . In determining the level of  $PaCO_2$  in which the mortality rate of severe brain injury patients worldwide is 76% in 6 days (Rondinna & Videtta, 2005), blood gas analysis is the best test but the use of blood gas analysis has drawbacks such as arterial blood sampling Is invasive, but it takes a long time about 3-4 hours in getting the results and the cost is quite expensive. The samples of this study were all patients with severe brain injury who came to RSSA of 50 samples with the largest number of patients were men (72%) with inclusion kriteia: all severe brain injury patients with GCS 3-6 who came to Doctor Saiful Anwar Hospital, Malang, Indonesia, aged> 14 years. Severe brain injury patient data retrieval was conducted for 5 months in the period of October 2016 until February 2017. This study used data from each severe brain injury patient who came to RSSA after intubation and ventilator installed and then analyzed blood gas 15 minutes post intubation and assessed the value of  $EtCO_2$ ,  $PaCO_2$  value and clinical patient after standard therapy then at 120 minutes post patient intubation Was examined again the value of  $EtCO_2$ ,  $PaCO_2$  value, clinical patient after that seen outcome patient for 6 days treatment and analyzed patient sample data.

#### III. Results

The following is the result of analysis of research data on severe brain injury patients in accordance with established inclusion and exclusion criteria and aims to determine the correlation between clinical diagnosis and the value of  $EtCO_2$  with the value of  $PaCO_2$  at minute 15 and 120 minutes later, knowing the suitability between clinical diagnosis and value  $EtCO_2$  with a  $PaCO_2$  value at 15 minutes and 120 minutes later, confirmed haemodynamic improvement in severe brain injury patients after the target of  $PaCO_2$  on day 6. Data collection of patients with severe brain injury conducted for 5 months in the period of October 2016 - February 2017 conducted at Emergency InstallationDoctor Saiful Anwar Malang Hospital (RSSA).

|--|

Variable	Mean ±SD	Median (min-max)
EtCO <sub>2</sub> 15 min post intubation	$32,4 \pm 3$	32,8 (25,8-39,3)
EtCO <sub>2</sub> 120 min post intubation	$33,3 \pm 3,9$	33,7 (19-38,9)
PaCO <sub>2</sub> 15 min post intubation	$33,5 \pm 4,3$	33,2 (22,7-44,2)
PaCO <sub>2</sub> 120 min post intubation	$34,8 \pm 2,9$	34,6 (28,4-42,4)
Clinical Diagnose 15 min post intubation	$11,5 \pm 2,3$	12 (5-15)
Clinical Diagnose 120 min post intubation	9,1 ± 3	9 (4-14)
Systolic 15 min post intubation	$144,7 \pm 30,3$	140 (54-215)
Systolic 30 min post intubation	$146,1 \pm 30,2$	140 (65-229)
Systolic 60 min post intubation	$140,5 \pm 27,8$	140 (59-235)
Systolic 120 min post intubation	$131,4 \pm 27,6$	122,5 (61-218)
Diastolic 15 min post intubation	$81,7 \pm 15,8$	80,5 (21-116)
Diastolic 30 min post intubation	$82,8 \pm 13,9$	85 (29-120)
Diastolic 60 menit post intubasi	$79,02 \pm 14,3$	80 (26-100)
Diastolic 120 min post intubation	$77,64 \pm 15,2$	80 (33-132)
Heart rate 15 min post intubation	$103,8 \pm 24,7$	106,5 (55-168)
Heart rate 30 min post intubation	$102,2 \pm 24,7$	100 (49-168)
Heart rate 60 min post intubation	$97,6 \pm 23,9$	91 (51-155)
Heart rate 120 min post intubation	$89,6 \pm 21,7$	88 (52-139)
RR 15 min post intubation	$22,02 \pm 7,6$	22 (1-50)
RR 30 min post intubation	$21,92 \pm 7,07$	22 (1-48)
RR 60 min post intubation	$21,86 \pm 5,5$	22 (10-44)
RR 120 min post intubation	$18,78 \pm 6,2$	18 (5-41)

Table 1 shows that the normal distributed variables (p > 0.05) were EtCO<sub>2</sub> minute 15, systolic minute 15, heart rate at minute 15, heart rate 30 minute, systolic minute to 60, PaCO<sub>2</sub> minute to 120 post intubation.

15th min Median (min	-max)	р	1 Median (n	20th Min nin-max)	р	
EtCO <sub>2</sub>	PaCO <sub>2</sub>		EtCO <sub>2</sub>	PaCO <sub>2</sub>		
32,8 (25,8-39,3)	33,2 (22,7-44	0,156 ,2)	33,8 34,7 (19-38,5)	0,06 (28,4-42,4)		

Table 2.Conformity	EtCO <sub>2</sub>	with	PaCO <sub>2</sub>
--------------------	-------------------	------	-------------------

Table 2 shows that there was a match between  $EtCO_2$  value and  $PaCO_2$  value at minute 15 and minute 120 with median value respectively is 32.8 and 33.2 at minute 15, while at minute 120 each median is 33, 8 and 34.7.

Table 3. Haemodynamic Changes (Systolic, Diastolic, Heart rate, Respiratory rate).

Variable	15-30th min	30-60th min	60-120 min	15-120th	n min
	р	р	р	р	
Systolic0,59 <sup>a</sup>	0,001 <sup>*a</sup>	0,001 <sup>*a</sup>	$0,000^{*a}$		
Diastolic	0,196 <sup>a</sup>	$0,019^{*a}$	0,339 <sup>a</sup>	$0,044^{*a}$	
Heart rate	0,316 <sup>b</sup>	$0,000^{*a}$	$0,000^{*a}$	$0.000^{*a}$	
RR	0,621 <sup>a</sup>	0,358 <sup>a</sup>	$0,000^{*a}$	$0,000^{*a}$	

**Explanation**:  $a = wilcoxon \ test$  $b = pair \ T \ test$ 

\*= meaningful if p<0,05

Table 3 shows that there was no significant change of hemodynamic at minute 15-30, whereas at minute 30-60 there was haemodynamic change in systolic, diastolic and heart rate except RR. In minute 60-120 RR value started to happen significant change simultaneously with systolic as well as the pulse whereas diastolic does not change significantly. Overall at 15-120 minutes both systolic, diastolic, heart rate and RR were significant changes.

Table 4. Clinical Characteristic Patient Haem	odynamic.
---	-----------

Variable	Rerata ± SD	Median (min-max)
Systolic min 15	$144,7 \pm 30,3$	140 (54-215)
Systolic min 30	$146,1 \pm 30,2$	140 (65-229)
Systolic min 60	$140,5 \pm 27,8$	140 (59-235)
Systolic min 120	$131,4 \pm 27,6$	122,5 (61-218)
Diastolic min 15	$81,7 \pm 15,8$	80,50 (21-116)
Diastolic min 30	$82,88 \pm 13,9$	85 (29-120)
Diastolic min 60	$79,02 \pm 14,3$	80 (26-100)
Diastolic min 120	$77,6 \pm 15,2$	80 (33-132)
Heart rate min 15	$103,8 \pm 24,7$	106,5 (55-168)
Heart rate min 30	$102,2 \pm 24,7$	100 (49-168)
Heart rate min 60	$97,6 \pm 23,9$	91 (51-155)
Heart rate min 120	$89,6 \pm 21,7$	88 (52-139)
RR min15	$22,02 \pm 7,7$	22 (1-50)
RR min 30	$21,9 \pm 7,07$	22 (1-48)
RR min 60	$21,8 \pm 5,6$	22 (10-44)
RR min 120	$18,8 \pm 6,2$	18 (5-41)

Table 4 shows that, in minutes 15-120 there are significant differences in both systolic, diastolic, heart rate, and RR, although still within normal limits mean haemodynamic minutes 15-120 is still in a relatively stable state.

Table 5. The relationship between outcome and value  $PaCO_2$ .

PaCO <sub>2</sub>				
Outcome	n	Mean ± SD	р	
Passed Away	46	34,69 ± 3		
DOI: 10.9790/	0853-16	08033034	www.iosrjournals.org	32   Page

#### Survive 4 $36,07 \pm 2,64$ 0,379

Table 5 shows that, in the outcome, whether dead or alive, there is no significant difference with p > 0.05, although it has already reached the normal limit of PaCO2 value.

Table 6. The Relationship between outcome and value EtCO2.     EtCO2					
Passed Away Survive	46 4	$\begin{array}{c} 33,02\pm 3,9\\ 36,8\pm 1,8 \end{array}$	0,03		

Table 6 shows that at the outcome both dead or alive there was a significant difference with p < 0.05 even though the difference was close to the marginal normal margin of EtCO<sub>2</sub> value.

### IV. Discussion

Based on the results of research conducted at Emergency Installation RSSA, a total of 50 patients as a sample of the study. Of the 50 head injured patients taken as the study sample, most were male (72%), according to some previous studies at RSHS Bandung in 2013 (79.8%).

The data showed a correlation between clinical diagnosis with the value of  $PaCO_2$  with (p = 0.71) minute 15, (p = 0.76) minute 120 and between  $EtCO_2$  with  $PaCO_2$  with at minute 15 with (p = 0.072) and minute (P = 0.076), the results were all insignificant, for hemodynamic changes at 15-30 minutes there was no significant difference in both systolic, diastolic, heart rate and RR, while at 60-120 minutes there was a significant difference in both systolic and pulmonary When seen in minutes 15-120 there are significant differences in the 4 variables. In the 6th day outcome improvement based on the value of  $EtCO_2$  and  $PaCO_2$  at 120minutes either death or life there was a significant difference with p <0.05, although the difference was close to the marginal normal margin of  $EtCO_2$  value.

## V. Conclusions And Recommendations

Based on the results of the research that has been done, it can be concluded: there is no correlation between clinical diagnosis and the value of  $EtCO_2$  with the value of  $PaCO_2$  at minute 15 and 120, there is a match between the value of  $EtCO_2$  with the value of  $PaCO_2$  at minute 15 and 120, there is hemodynamic improvement in patient Severe brain injury at 15-120 minutes (systolic, diastolic, heart rate, RR), although RR variables begin to experience significant differences in the 60-120 minutes, the target value of  $PaCO_2$  in severe brain injury patients can improve the patient's haemodynamics at 120 minutes in Margin limit, no significant difference in outcome on day 6 of severe brain injury patients based on  $EtCO_2$  value and  $PaCO_2$  value at 120 minutes.

#### Thus, based on the discussion, it can be recommended as follows:

- 1. Patients who come to Emergency Installation RSUD Dr. Saiful Anwar (RSSA) with severe brain injury should check the value of  $EtCO_2$  without having to wait for the results of  $PaCO_2$  value from a blood gas analysis that takes a much longer time.
- 2. If the clinical state of a severe brain injury patient during which clinical observation worsens even though the value of EtCO<sub>2</sub> reaches the target, immediate intervention is necessary.
- **3.** Need further research outcome on severe brain injury patients who have done surgical intervention compared with non-surgery.

#### References

- [1]. A.H.A.-A.C.L.S 2010, 'Guidelines', Circulation, p. 135.JM Field, MF Hazinski.
- [2]. Alzheimer, 2002, 'The epidemiology and impact of traumatic brain injury:a brief overview, Journal of Head Trauma Rehabilitation, p. 375-378.
- [3]. Bazarian J, Mcclung J, Shah Manish N, Cheng YT, Flesher W, Krauss J, 2005 : Traumatic brain injury in the united state, p :85-91
- [4]. Bullock, Reagan, Mitchel, 2000 :Management and prognosis is of severe traumatic brain injury, part 1, Guidlines of the management of devere traumatic brain injury. J Neurotruma 17:451.
- [5]. Capless, SM & Hubmayr, RD 2003, 'Respiratory monitoring tools in the intensive care unit', Cur Opin Crit Care, vol. 9, no. 3, p. 230.
- [6]. Curry P, Viernes D, Sharma D, 2011 Periopperative management of traumatic brain injury. Int J Critt IIIn Inj Sci.
- [7]. Davis P, Davinson M, Patrick S, 2005: The impact of aeromedical response to patients with moderate to severe trumatic brain injury. Ann Emerg Med; 46:115-112.

- [8]. D Hanley, 2007, 'Intracranial hypertension; Intensive care manual', fourth edition, Butterworth Heinemann.p. 395-402.
- [9]. Ezri T, Warters D, 2007, 'Indications For Tracheal Intubation', in CA Hagberg (ed.), Hagberg: Benumof's Airway Management, 2 edn, Mosby Elsevier.
- [10]. Grifith, H & Johnson, G 1992, 'The use of curare in general anesthesia', Anesthesiology vol. 3, p. 418.
- [11]. Grmec, S & Klemen, P 2001, 'Does the end-tidal carbon dioxide (EtCO<sub>2</sub>) concentration have prognostic value during out-of-hospital cardiac arrest?', Eur J Emerg Med vol. 8, p. 263.
- [12]. Guyton, AC & Hall, JE, 2006, 'Energetics and Metabolic Rate', in Textbook of Medical Physiology, 10 edn, Elsevier Saunders, Philadelphia, pp. 882-3.
- [13]. Hatton J, 2001, 'The sequence of human genome in traumatic brain injury', vol.291, Issue 5507, pp.1304-1351.
- [14]. Http://www.cdc.gov/ncipc/pub-res/TBI\_US\_04/00\_preliminary.htm (Langlois JA Rutland-Brown W Thomas KE, 2014 :Traumatic brain injury in the United States:emergency visits, hospitalisation and deaths.CDC Publication:Centers for Disease Control and Prevention, nasional Center for injury Prevention and control.
- [15]. John AP. Paul JF. Harold KK, 1996 Pathophysiology of Traumatic Brain Injury, Neurosurgery, second edition, volume II. Mc Graw-Hill Companies ;2623-37
- [16]. Joynt, GM ,2009, 'Airway management and acuteupper-airway obstruction', in AD Bersten & N Soni (eds), Oh's Intensive Care Manual, Elsevier, p. 331.
- [17]. Marc Quinlan, M & Brian M. Jekich, M, 2016, 'Resuscitation', in M Barbara K. Blok (ed.), First Aid For The Emergency Medicine Boards, third edn, Mc Graw Hill Education, China, pp. 1-78.
- [18]. Mangat HS, 2012, Severe traumatic brain injury. American academy of neurology : 532-546.
- [19]. Menzel, M, Henze, D, Soukup, J, Engelbrecht, K, Senderreck, M, Clausen, T & Radke, J 2001, 'Experiences with continuous intraarterial blood gas monitoring', Minerva anestesiologica, vol. 67, no. 4, pp. 325-31.
- [20]. Mitchell, V & Patel, A 2005, "Tracheal Tubes', in I Calder & A Pearce (eds), Core Topics In Airway Management, Cambridge University Press, p. 57.
- [21]. Myers, C, 2007, 'Critical care monitoring in the emergency department', Emergency Medicine Practice, vol. 9, p. 7.
- [22]. Sastroasmoro, S & Ismael, S 1995, Dasar Dasar Metodologi Penelitian, Binarupa Aksara, Jakarta.
- [23]. Selladurai, VKompanje, E., Leebeek, F. Acta Neurochir (Wien) (2007) 150: 165, 'Coagulation disorders after traumatic brain injury.
- [24]. Steine, J & Grande, C,2000, 'Anesthesia for trauma', in R Miller (ed.), Anesthesia, 5 edn, Churchill Livingstone, p. 2157.
- [25]. Sorani MD, Manley GT, 2008, Dose-response relationship of mannitol and intracranial pressure: a meta-analysis.J Neurosurg.;108:80-7.
- [26]. Qureshi AI, Suarez JI, 2000 ' Hypertonic saline solutions for treatment of intracranial hypertension'. Curr Opinion Anaesthesiol;5:3301-3
- [27]. Vissers, RJ & Danzl, DF 2011, 'Tracheal Intubation and Mechanical Ventilation', in JE Tintinalli & JS Stapczynski (eds), Tintinalli Emergency Medicine, 7 edn, McGraw-Hill, New-York, p. 198.
- [28]. Wagstaff, ATJ 2009, 'Oxygen therapy', in AD Bersten & N Soni (eds), Oh's Intensive Care Manual, 6 edn, Elsevier, pp. 315-7.

\*Muhammad Isya Firmansyah. "Fitness Between The Value of Etco2 Rate with The Valance of Paco2 in Hemodynamic Repair on Severe Brain Injury Patients." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 16.8 (2017): 30-34.