A COMPARISON OF THE EFFECTS OF ENDOTRACHEAL SUCTIONING WITH AND WITHOUT NORMAL SALINE INSTILLATION ON PHYSIOLOGIC CHANGES IN PEDIATRIC PATIENTS WITH MECHANICAL VENTILATION

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ABSTRACT

This crossover-experimental research compared of the effects of endotracheal suctioning with and without normal saline instillation on physiologic changes in pediatric patients with mechanical ventilation. The conceptual framework was based on biomedical knowledge about pathophysiology of oxygenation and ventilation during endotracheal suctioning. Sixty-five suctionings from fifty-eight pediatric patients, who were admitted to the Pediatric Intensive Care Unit (PICU) in Ramathibodi Hospital during July 2005 to January 2006, were selected by purposive sampling. The crossover-experimental design was used to compare the mean changes in oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure between the subjects after endotracheal suctioning. Each subject was exposed to both methods: the endotracheal suctioning with and without normal saline instillation. The sequence of the experimental method for eligible subjects was assigned by drawing lots (number 1 to 65) without replacement. The duration between each suctioning method was 3 hours before starting another method. Data were analyzed using with SPSS/FW program in descriptive statistics, paired t-test, and wilcoxon signed ranks test.

The results revealed that oxygen saturation after endotracheal suctioning with normal saline instillation was decreased more than after the endotracheal suctioning without normal saline instillation over time with a statistically significant difference ($p<.05$). End-tidal CO_2, heart rate, and mean blood pressure after endotracheal suctioning with normal saline instillation were increased more than after the endotracheal suctioning without normal saline instillation over time with a statistically significant difference ($p<.05$).

This study suggests that the instillation of normal saline before endotracheal suctioning resulted in decrease of oxygen saturation, increase of end-tidal CO_2, heart rate and blood pressure. Thus, these data should be used to develop an endotracheal suctioning clinical nursing practice guideline.

KEY WORDS: ENDOTRACHEAL SUCTIONING / NORMAL SALINE INSTILLATION / ENDOTRACHEAL INTUBATION / PHYSIOLOGIC CHANGES

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การศึกษาเปรียบเทียบผลของการหยอดน้ำเกลือนอร์มัลผ่านท่อหลอดลมคอก่อนการดูดเสมหะต่อการเปลี่ยนแปลงทางด้านสรีรวิทยาในผู้ป่วยเด็กที่ต้องใช้เครื่องช่วยหายใจ (A COMPARISON OF THE EFFECTS OF ENDOTRACHEAL SUCTIONING WITH AND WITHOUT NORMAL SALINE INSTILLATION ON PHYSIOLOGIC CHANGES IN PEDIATRIC PATIENTS WITH MECHANICAL VENTILATION)

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บทคัดย่อ

การวิจัยครั้งนี้ ทำการศึกษาเปรียบเทียบผลของการหยอดและไม่หยอดน้ำเกลือนอร์มัลผ่านท่อหลอดลมคอก่อนการดูดเสมหะต่อการเปลี่ยนแปลงทางด้านสรีรวิทยาในผู้ป่วยเด็กที่ต้องใช้เครื่องช่วยหายใจ กรอบแนวคิดในการศึกษาครั้งนี้ คือ ความรู้ทางด้านพยาธิสรีรวิทยาเกี่ยวกับการได้รับออกซิเจนและการระบายอากาศระหว่างการดูดเสมหะ กลุ่มตัวอย่าง คือ ผู้ป่วยเด็กจำนวน 58 คน ที่รับเข้ารักษาในหอผู้ป่วยวิกฤตเด็กของโรงพยาบาลรามาธิบดี ระหว่างเดือนกรกฎาคม 2548 ถึง เดือนมกราคม 2549 กลุ่มตัวอย่างนี้ ได้รับการทดลองดูดเสมหะจำนวน 65 ครั้ง โดยเลือกกลุ่มตัวอย่างแบบเฉพาะเจาะจง การออกแบบงานวิจัยในครั้งนี้ผู้ป่วยทุกรายเป็นกลุ่มควบคุมในตนเองเพื่อเปรียบเทียบ ระดับความอิ่มตัวของออกซิเจน ระดับความดันก๊าซคาร์บอนไดออกไซด์ในลมหายใจออก อัตราการเต้นของหัวใจ และค่าความดันโลหิตเฉลี่ยที่เปลี่ยนแปลงไปของผู้ป่วยหลังการดูดเสมหะระหว่างการหยอดและไม่หยอดน้ำเกลือนอร์มัล เลือกลำดับการทดลองโดยการสุ่มจับฉลากแบบไม่แทนที่ตั้งแต่ 1 ถึง 65 ระหว่างการดูดเสมหะในการทดลองแต่ละครั้งจะต้องเว้นระยะห่างกันประมาณ 3 ชั่วโมง การวิจัยครั้งนี้วิเคราะห์ข้อมูลด้วยโปรแกรม SPSS/FW โดยใช้สถิติบรรยาย paired t-test และ wilcoxon signed ranks test

ผลการวิจัยพบว่า กลุ่มที่หยอดน้ำเกลือนอร์มัลมีระดับความอิ่มตัวของออกซิเจนลดลงมากกว่ากลุ่มที่ไม่ได้รับการหยอดน้ำเกลือนอร์มัลก่อนการดูดเสมหะอย่างมีนัยสำคัญทางสถิติ ($p<.05$) กลุ่มที่หยอดน้ำเกลือนอร์มัลมีระดับความดันก๊าซคาร์บอนไดออกไซด์ในลมหายใจออก อัตราการเต้นของหัวใจ และค่าความดันโลหิตเฉลี่ยเพิ่มขึ้นมากกว่ากลุ่มที่ไม่ได้รับการหยอดน้ำเกลือนอร์มัลก่อนการดูดเสมหะอย่างมีนัยสำคัญทางสถิติ ($p<.05$)

ผลที่ได้จากการศึกษาครั้งนี้สนับสนุนว่า การหยอดน้ำเกลือนอร์มัลก่อนการดูดเสมหะอาจก่อให้เกิดการเปลี่ยนแปลงในผู้ป่วยเด็ก ได้แก่ ระดับความอิ่มตัวของออกซิเจนในเลือดลดลง ระดับความดันก๊าซคาร์บอนไดออกไซด์ในลมหายใจออก อัตราการเต้นของหัวใจ และความดันโลหิตเฉลี่ยเพิ่มมากขึ้น ดังนั้น ควรนำความรู้ที่ได้จากการศึกษาครั้งนี้พัฒนาไปสู่แนวทางการปฏิบัติการพยาบาลในการดูดเสมหะ

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CHAPTER I
INTRODUCTION

Background and Rationale

Most of the pediatric patients under crisis condition often have problems concerning respiratory system. About 61.65% of hospitalized children's lives were threatening that they need monitoring and care in the Pediatric Intensive Care Unit (PICU) (The statistics of the Pediatric Intensive Care Unit of Faculty of Medicine, Ramathibodi Hospital, 2004). Most of them require the artificial airway to support their lives as the endotracheal intubation and mechanical ventilation can maintain the airway; thus, reduce work of breathing and lead to effective gas exchange and ventilation. However, the endotracheal intubation also presents a great threat to the integrity of the respiratory system as it bypasses the normal protection of body function such as warming and filtering inspired air. This may result in dryness and tenacious secretions. Moreover, the patients with endotracheal intubation are unable to cough due to the epiglottis does not close tightly which leads to retention and obstruction of secretion easily. These may cause atelectasis, pneumonia and possibly lead to death. Thus, endotracheal suctioning is necessary to remove respiratory secretions in order to maintain optimum ventilation and oxygenation.

In current nursing practice, nurses may arbitrarily decide when instillation of normal saline is appropriate. Some practitioners have been taught that routine instillation of normal saline before suctioning is necessary to thin and loosen tenacious secretions including stimulate cough. However, several researches find that normal saline may be harmful to the oxygenation status. In addition, normal saline instillation may retain in the airway; interrupt the ventilation and oxygenation which leading to hypoxia. When patients face with hypoxemia, they will experience headache, tiredness, and finally, unconsciousness or coma. Furthermore, immediately after hypoxia occurs, the myocardial will work very hard to maintain the cardiac output. Thus, the patients will undergo the tachycardia. In addition, blood pressure will be

higher in order to compensate the adequate oxygenation for various tissues. Besides, the retention of normal saline can reduce the ventilation and cause carbon dioxide retention.

Several researches studied the effects of instillation of normal saline before endotracheal suctioning on arterial oxygen tension and oxygen saturation, which tended to decrease more after instillation of normal saline (Ackerman & Gugerty, 1990; Ackerman, 1993; Ackerman & Mick, 1998; Bostick & Wendelgass, 1987; Ji, Kim & Park, 2002; Ridling, Martin & Bratton, 2003; Schwenker, 1998; Shorten et al., 1991). Ackerman (1993) studied 40 adult patients of post open-heart surgery, and found that oxygen saturation was decreased at 2, 3, 4, and 5 minutes after endotracheal suctioning with normal saline instillation. She recommended that the instillation of normal saline should not be treated as a routine or standard intervention and that it should be regarded as potentially hazardous. In the pediatric patient study, Ridling, Martin, and Bratton (2003) studied the effects of the endotracheal suctioning with and without normal saline instillation in 24 critically ill children, and found that oxygen saturation was decrease at 1 and 2 minutes after endotracheal suctioning with normal saline instillation. Kinloch (1999) studied 35 patients after coronary bypass grafting, and found that the recovery time of oxygen saturation to return to baseline after endotracheal suctioning without and with normal saline instillation were at the average of 3.78 minutes and 7.30 minutes respectively. Besides, several studies found that the normal saline instillation before suctioning tended to increase the heart rate and blood pressure (Ackerman & Mick, 1998; Beerman & Dhanireddy, 1992; Gray, MacIntryre & Kronenberger, 1990).

A study of the patient's perception on 40 patients experiencing open-heart surgery in intensive care unit was found that 37.5% perceived the suctioning as the dangerous procedure whilst 57.5% perceived the normal saline instillation as disturbance to a great extent (Karnchana Simajaruek, B.E. 2538). Moreover, instillation of normal saline before suctioning not only associated with a decrease in oxygen saturation but also may increase the risk of lower airway infection. The number of bacteria dislodged increased 5-fold with instillation of normal saline (Hargler & Traver, 1994). This result indicated the artificial airways were colonized with bacteria and that instillation of normal saline dislodged these bacteria and carried

the organisms to the lower airway. Thus, it was recommended that the practice of normal saline instillation be abandoned.

From the experience of the researcher, the instillation of normal saline before endotracheal suctioning affects additional discomfort, struggle and cry in pediatric patient. Besides, the retention of normal saline in airway may obstruct the ventilation and lead to retain of carbon dioxide, especially in pediatric patient with neurological problem as it may cause an increased intracranial pressure (Williams & Wilkins, 1996). Furthermore, the trachea of pediatric patients is smaller than the adult's. As a result, the normal saline instillation may be more dangerous, for instance, airway obstruction and hypoxia; it may cause hypoxia, reducing oxygenation and ventilation, and increase the risk of lower airway infection. These may cause suffering, prolong the endotracheal intubation, delay on recovery, and increase of hospital costs.

From the literature review and various researches, there have been few studies on the effects of normal saline instillation in endotracheal suctioning in pediatric patients. Most of them are concerned with adults and studied in foreign countries. For Thailand, there had been no study on the pediatric patient found yet. Consequently, the researcher is interested in studying a comparison of the effects of endotracheal suctioning with and without normal saline instillation in pediatric patients with mechanical ventilator on oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure. The results in this study are expected to be useful as a guideline for nursing practice in endotracheal suctioning appropriately and safely from complications.

Research Question

Are the oxygen saturation (SpO_2), end-tidal CO_2, heart rate, and mean blood pressure after suctioning over time different between with and without normal saline instillation before endotracheal suctioning in pediatric patients with mechanical ventilation?

Objectives of the Study

To compare the differences of oxygen saturation (SpO_2), end-tidal CO_2, heart rate, and mean blood pressure between with and without normal saline instillation before endotracheal suctioning in pediatric patients with mechanical ventilation.

Conceptual Framework

The researcher uses biomedical knowledge on pathophysiology of oxygenation and ventilation during endotracheal suctioning to be a conceptual framework of the study. The endotracheal suctioning induces physiologic responses by autonomic nervous system, coordinating neuroendocrine functions, and controlling homeostasis.

The autonomic nervous system (ANS) provides an external influence on myocardial contractility and rate. This involves adjusting the heart rate and contractility to the demands of the body. The autonomic nervous system has an enhancing or restraining effect on the inherent pacemaker system and can alter the automaticity of abnormal pacemaker systems. When the endotracheal suctioning leads to a generalized increase of central nervous system arousal, signals from the hypothalamus transmits to autonomic control centers of the brain stem. In early stage, the endotracheal suctioning affects reduction of oxygen saturation and stimulation of vagus nerve receptor. The body has been hypoxemia. Vagus nerve or parasympathetic nervous system is stimulated; it affects the bradycardia and hypotension immediately. Then, the body has been compensated by negative feedback.

Parasympathetic nervous system (PSNS)

The major effect of vagus stimulation is on the SA node, atrial muscle, and the AV node. The result of stimulation is a restraining influence on the conduction tissue, which only a slight decrease in ventricular contractility. Vagal stimulation slows the heart rate by restraining the rate of diastolic depolarization in the conduction tissue including low blood pressure immediately (Bullock, 2000).

In general, detection of inadequate blood pressure and a lack of oxygen result in activation of the sympathetic nervous system. Specialized sensory nerve endings, called baroreceptors, located in the aortic arch and carotid arteries respond to changes in blood pressure and transmit this information to the central nervous system (CNS). A

fall in blood pressure causes parasympathetic system inhibition and cardiac sympathetic nerve activation, resulting in a rise in heart rate.

Because blood pressure is influenced by arteriolar resistance, it is not always a good indicator of cardiac output; hence, other monitoring systems are utilized. Chemoreceptors provide additional information to the central nervous system about the adequacy of cardiac output. Chemoreceptor located in the carotid arteries, aortic arch, and hypothalamus responds to the levels of oxygen and carbon dioxide of in blood. Detection of indequate oxygen or excessive carbon dioxide results in sympathetic activation and an increase in heart rate. A reduction in carbon dioxide results in parasympathetic activation and decrease heart rate (Banasik, 1995).

Sympathetic nervous system (SNS)

Fibers from the sympathetic nervous system are present in the atrial wall, ventricles, and SA and VA nodes. When it is stimulated, these fibers release norepinephrine, which stimulate the rate of depolarization and the rate at which impulses are transmitted through the conduction tissue. Therefore, increased sympathetic tone improves cardiac rate and the contractility of myocardial muscle. The predominant effect is usually on the sinus node and causes a sinus tachycardia. Stimulation of the sympathetic nervous system can also increase the irritability of myocardial muscle cells, causing abnormal or early depolarization, such as, premature atria or ventricular contractions. These are usually referred to as ectopic foci because they are outside the SA node.

The effects of the sympathetic nervous system on the coronary arteries are somewhat more complex. Norepinephrine has been shown to cause coronary artery vasoconstriction and increased oxygen extraction by myocardial cell. Epinephrine is released from the adrenal glands and has a secondary dilating action on the coronary arteries. Normally, autoregulation of coronary blood flow appears to counteract the effects of neural stimulation. Then, the sympathetic nervous system is stimulated that results in increase heart rate and blood pressure in order to correct hypoxemia (Bullock, 2000).

Moreover, the normal saline instillation before endotracheal suctioning affects more physiologic response than the suctioning only, for example, higher decrease of oxygen saturation, higher increase of heart rate and blood pressure.

Normally, gas exchange requires the movement of gases into and out of the lungs. Various control mechanisms and forces interplay in the two phases of ventilation: inspiration and expiration. Inspiration is the process of moving air into the lungs whilst expiration moves it out. The endotracheal suctioning reduces the ventilation and oxygenation. Since the air in airway is suctioned; it may cause reduction of oxygen. Besides, the suction catheter can obstruct and interrupt the movement of the air into the lungs. These may result in a reduction of ventilation. Furthermore, the normal saline instillation may interrupt ventilation and diffusion of oxygen and carbon dioxide between alveolar and capillary of lungs leading to hypoxemia and hypoventilation (Figure 1).

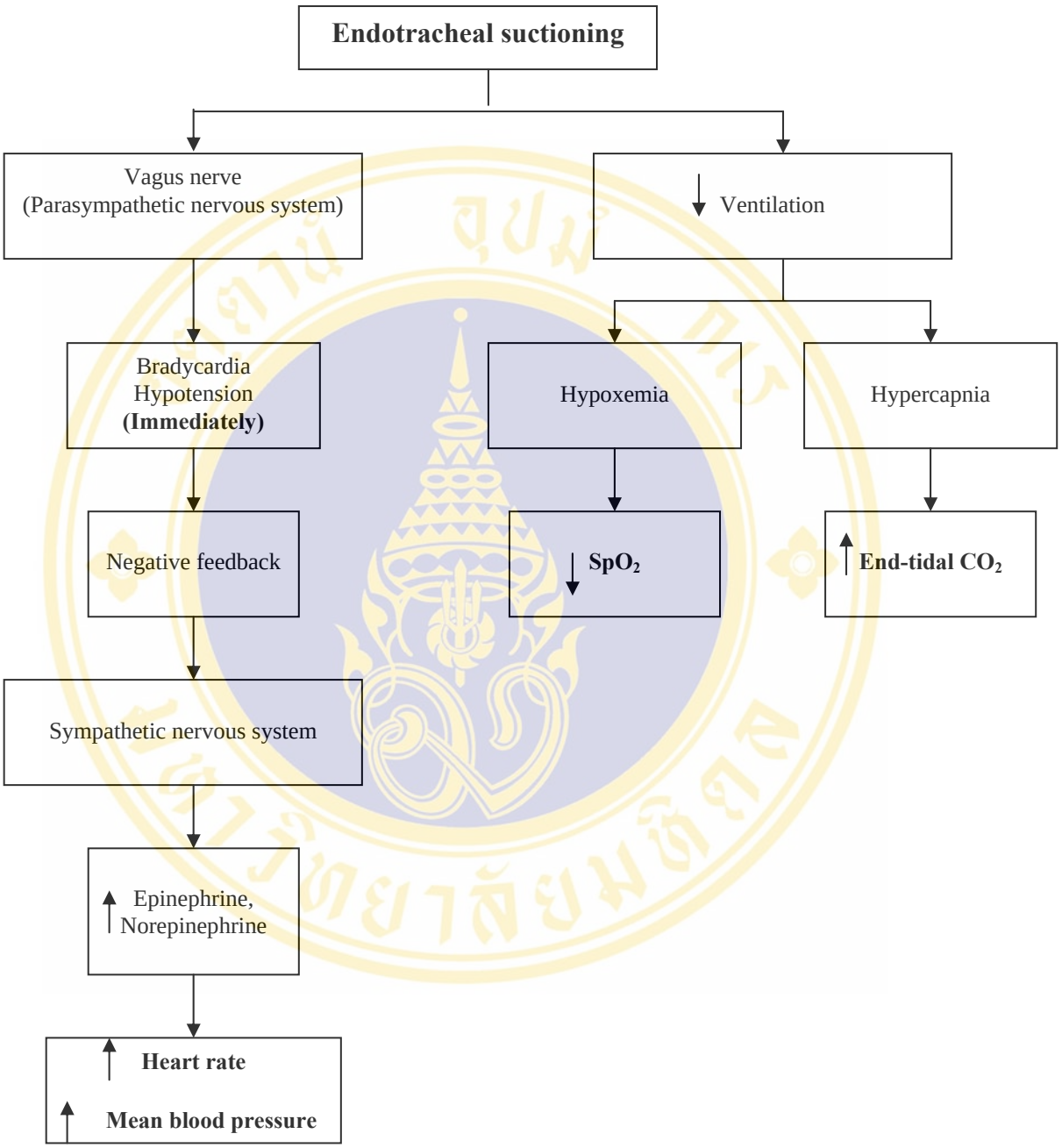

Figure 1: Conceptual framework of this study

Hypotheses

1. The mean of oxygen saturation of patients who had the endotracheal suctioning with normal saline instillation decrease more than the patients who had the endotracheal suctioning without normal saline instillation.

2. The mean of end-tidal CO_2 of patients who had the endotracheal suctioning with normal saline instillation increase more than the patients who had the endotracheal suctioning without normal saline instillation.

3. The mean of heart rate of patients who had the endotracheal suctioning with normal saline instillation increase more than the patients who had the endotracheal suctioning without normal saline instillation.

4. The mean of mean blood pressure of patients who had the endotracheal suctioning with normal saline instillation increase more than the patients who had the endotracheal suctioning without normal saline instillation.

Scope of the Study

The research aimed to compare the difference of oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure between with and without normal saline instillation before endotracheal suctioning. The endotracheal suctioning applies negative pressure of 80 to 100 mmHg, the diameter of suction catheter should not be bigger than a half of the internal diameter of the endotracheal tube, and the period of suctioning should not be longer than 10 seconds in pediatric patients with mechanical ventilation. The sample used in this study was fifty-eight pediatric patients who were admitted in the Pediatric Intensive Care Unit (PICU) of the Faculty of Medicine, Ramathibodi Hospital during July 2005 to January 2006.

Expected Outcomes and Benefit

1. These data should be used to develop an endotracheal suctioning clinical nursing practice guideline.

2. To provide knowledge to nurses and other staff for proper practice in endotracheal suctioning.

3. The patient can be safe from complications from endotracheal suctioning and normal saline instillation.

4. To serve as a direction for study and research that is beneficial in nursing the patient having endotracheal intubation, prevent tenacious secretions and other methods to reduce instillation of normal saline and reduce cost of using normal saline.

Definition of Terms

Endotracheal suctioning refers to the procedure applies the size of the suction catheter should not be bigger than a half of internal diameter of the endotracheal tube. The negative pressure should be limited between 80 to 100 mmHg. Duration of suctioning should take no longer than 10 seconds.

Normal saline instillation before endotracheal suctioning refers to instillation of sterilized normal saline in the endotracheal tube before suctioning, the amount of normal saline depends on patient's age: less than 1 year use 0.5 ml, 1-8 years use 1 ml, and more than 8 years use 2 ml.

Without normal saline instillation before endotracheal suctioning refers to no sterilized normal saline instillation in the endotracheal tube before suctioning.

Oxygen saturation (SpO_2) refers to the amount of oxygen bound to hemoglobin compared with hemoglobin's maximal ability for oxygen binding. It can be assessed by capnometer, BCI Capnockeck Plus (BCI-9004). The greater decrease of oxygen saturation from the baseline levels, the worse response.

End-tidal CO_2 refers to the partial pressure or maximal concentration of carbon dioxide at the end of an exhaled breath, which is expressed as mercury millimeters (mmHg). It can be assessed by the sidestream capnometer, BCI Capnocheck Plus (BCI-9004). The greater increase of end-tidal CO_2 from the baseline levels, the worse response.

Heart rate refers to the rate of heart contraction in 1 minute resulting from the pumping of left lower heart, which is expressed as beats/minute. It measured by BCI Capnocheck Plus (BCI-9004). The greater increase of heart rate from the baseline levels, the worse response.

Mean blood pressure refers to the average pressure in the system throughout the cardiac cycle. It is not the arithmetic average of the diastolic and systolic pressures, and estimated by diastolic blood pressure plus one-third of the pulse pressure. It is expressed as mercury millimeters (mmHg), and can be assessed by non-

invasive blood pressure monitor, Phillip (Model No. M1205A). The greater increase of mean blood pressure from the baseline levels, the worse response.

CHAPTER II
LITERATURE REVIEWS

This research is aimed to study the effects of endotracheal suctioning with and without normal saline instillation on physiologic changes in pediatric patients with mechanical ventilator. The literature reviews are presented in sequence as follows:

1. Mechanism of respiratory system
2. Artificial airway
3. Endotracheal suctioning, complications from suctioning and managements
4. Normal saline instillation before endotracheal suctioning
5. Impacts of endotracheal suctioning with normal saline instillation
6. Assessments of oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure

Mechanism of Respiratory System

The mechanism of respiratory systems consists of four processes: ventilation, perfusion, gas exchange, and oxygen transport.

1. Ventilation

Pulmonary ventilation or breathing is a process by which gases in an atmosphere and lung alveoli are exchanged (Hinchliff, et al.,1996). Inspiration moves air into the lungs, while expiration moves air out of the lungs. The mechanism that produces pulmonary ventilation is the one that establishes a gas pressure gradient between the atmosphere and the alveolar air. When the atmospheric pressure is greater than the alveolar, the air moves from the atmosphere into the lungs or an inspiration occurs. When the alveolar pressure becomes greater than atmospheric pressure, the air moves from the lungs to the atmosphere or an expiration occurs (Lumb, 2000; Sudpranom Samantawaekin, B.E. 2543).

Dead space

Other concepts important to ventilation are dead space, minute ventilation, and alveolar ventilation. Dead space includes three dimensions: anatomic dead space, alveolar dead space, and physiologic dead space.

Anatomic dead space includes the volume of non-usable gas (not used in gas exchange) in the conducting airways from the nose down to the respiratory bronchioles. The area between the nasal cavity and the terminal bronchioles is considered to be anatomic dead space because gas exchange does not occur in these areas. In the newborn and young child, the anatomic dead space is proportionately lager for its size.

Alveolar dead space is composed of the ventilated but unperfused or underperfused areas of the lung and is often referred to as wasted ventilation. Alveolar dead space increases with the development of pulmonary emboli owing to decreased perfusion.

Physiologic dead space is the sum of the anatomic dead space and alveolar dead space. Approximately one-third of each breath is wasted as dead space (Schumann, 1995).

Minute ventilation

Minute ventilation or expired volume (V_E) is the product of tidal volume time respiratory rate per minute (Schumann, 1995).

Alveolar ventilation

By comparison, alveolar ventilation (V_A) equals the difference between tidal volume (V_T) and anatomic dead space volume (V_D) multiplied by the respiratory rate (RR) per minute:

$$(V_T - V_D) \times RR = \text{Alveolar ventilation } (V_A)$$

Because alveolar ventilation is affected by both anatomic dead space and the respiratory rate, slow deep breathing yields greater alveolar ventilation than rapid shallow respiration (Schumann, 1995).

2. Perfusion

The function of the pulmonary circulation is to bring the entire cardiac output into contact with alveolar air so that gas exchange can occur. The total volume of blood in pulmonary circulation is approximately 500 ml but 80 ml occurs within the alveolar capillaries. Pulmonary capillary pressure is about 10 mmHg, where the oncotic pressure is 25 mmHg. There is a net force of about 15 mmHg that keeps the alveoli free of fluid (Ganong, 1995). The distribution of pulmonary blood flow can be considered in the three classic zones described by West (1978) (Figure 2).

Zone I. It presents those apical areas in which alveolar pressure is greater than arteriolar and venular pressure ($P_A > P_{art} > P_{ven}$). In this region there is no blood flow because the pulmonary capillaries are collapsed.

Zone II. In this region, pulmonary arteriolar pressure is greater than alveolar and venular pressure ($P_{art} > P_A > P_{ven}$). Blood flow will increase downwards this region because pulmonary arteriolar pressure is increasing as a result of gravitational effects.

Zone III. In this region, both the arteriolar and venular pressure exceed alveolar pressure ($P_{art} > P_{ven} > P_A$).

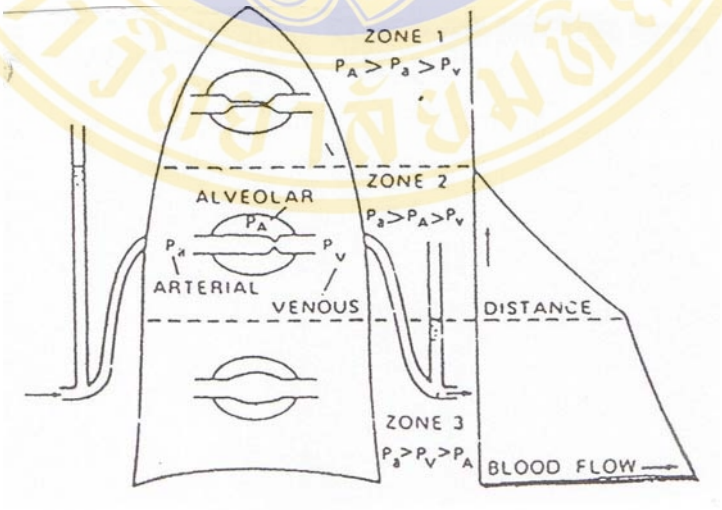

Figure 2: Distribution of blood flow in the lungs (West, 1978)

Ventilation-perfusion ratios

A factor important to the concepts of ventilation and perfusion is the matching of an adequate volume of air in the alveoli to adequate pulmonary blood flow. In the ideal state, 4 L/minute of alveolar ventilation is matched to 5 L/minute of capillary blood flow in the lungs, creating a normal V_A/Q ratio of 0.8. Two major factors impacting this normal V_A/Q ratio are right-to-left shunt and regional ventilation and perfusion changes. Other factors influencing the ratio are position changes, exercise, bed rest, and disease conditions of the lung (Schumann, 1995).

3. Gas exchange

The exchange of oxygen and carbon dioxide occurs in the alveolar. Carbon dioxide and oxygen diffuse through the alveolar capillary membranes. The pressure gradient for carbon dioxide causes carbon dioxide to diffuse from the blood into alveolar space, while the pressure gradient for oxygen causes oxygen to diffuse from the alveolar space to the blood. The amount of oxygen that diffuses through the alveolar-capillary membrane depends on the pressure gradient and on the amount of function of alveolar membrane (Liangchai Limlomwong, B.E. 2538).

Diffusion

Diffusion is the movement of gas from a high concentration area to a low concentration area. The alveolar-capillary membrane through which O_2 and CO_2 must diffuse consists of six barriers. For O_2 to reach the hemoglobin molecule, it must pass through surfactant, the alveolar membrane, interstitial fluid, the capillary membrane, plasma, and the red blood cell membrane. The rate of diffusion of a gas is proportional to the tissue area and the difference in gas partial pressure between the two sides of alveoli, and inversely proportional to the tissue thickness that the gas must move through. Oxygen diffuses into blood from the alveoli, and carbon dioxide diffuses out of the blood into the alveoli (Schumann, 1995).

4. Oxygen transport

The transport of oxygen to the body tissue and the removal of carbon dioxide is a complex process involving external respiration, hemoglobin concentration, hemoglobin-oxygen affinity, arterial oxygen saturation, cardiac output, blood viscosity, internal respiration, and changes in oxygen supply and demand (Kotter & Osguthorpe, 1995).

4.1 Oxygen delivery (Do_2) is the amount of oxygen delivered per minute to the tissues. It is calculated by multiplying the arterial oxygen content (Cao_2) by the cardiac output. Cardiac output depends on heart rate, preload, stroke volume, and afterload and is usually between 4 and 8 L/minute. Therefore, oxygen delivery is approximately 1,000 mL/minute.

$$\text{Oxygen delivery (mL/min)} = \text{cardiac output} \times Cao_2 \times 10$$

4.2 Oxygen demanded is the demand of oxygen of cell for aerobic metabolism. The oxygen demanded is the measurement of demand on metabolic oxygen of cell. It can be estimated roughly from the oxygen consumption.

4.3 Oxygen consumption (Vo_2) is the amount of oxygen consumed by the tissues, and it is measured in milliliters of oxygen per minute. Once the oxygen reaches the tissues, oxygen consumption is controlled by the rate of energy expenditure within the cells or the rate at which adenosine diphosphate (ADP) is formed from adenosine triphosphate (ATP) to provide energy. The increasing concentration of ADP enhances the metabolic utilization of oxygen and nutrients, which releases energy. Oxygen consumption can be determined by subtracting the oxygen remaining in the venous blood (venous transport) from the oxygen delivered to the tissues by the arteries (arterial transport), and is known as the Fick equation.

$$\text{Oxygen consumption} = \text{arterial oxygen transport} - \text{venous oxygen transport}$$

$$Vo_2 = CO \times (Cao_2 - Cvo_2) \times 10$$

Arterial blood oxygen content (Cao_2) and venous blood oxygen content (Cvo_2) can be calculated by considering the oxygen capacity of hemoglobin, the oxygen combined with hemoglobin, and the oxygen dissolved in plasma.

Carbon dioxide diffusion and transport

Carbon dioxide is the by-product of metabolic activity and travels a pathway opposite to oxygen. Tissue carbon dioxide diffuses rapidly into the venous end of the capillaries with a gradient of less than 1 mmHg. It diffuses about 20 times more readily than oxygen. The systemic venous blood eventually returns carbon

dioxide to the pulmonary capillary bed via the pulmonary artery and pulmonary arterioles. Following pressure gradients, carbon dioxide in pulmonary capillary blood diffuses across the alveolar-capillary interspaced and into the alveolus. The carbon dioxide is then removed from the alveolus during expiration. The movement of oxygen and carbon dioxide across the alveolar-capillary membrane due to ventilation, diffusion, and perfusion is depicted.

Carbon dioxide transport occurs in two forms: in RBCs (89%) and in the plasma (approximately 11%). In the RBC, the majority of the carbon dioxide is carried as bicarbonate (63%). It may also be carried as carbaminohemoglobin [carbon dioxide combined with hemoglobin (21%) or dissolved (5%)]. In the plasma, it is carried as bicarbonate (5%), as a carbamino compound (1%) or dissolved (5%).

The reaction of carbon dioxide as it is carried dissolved in the RBCs. It is important to note that this is a reversible reaction that occurs rapidly at the tissue level, where carbon dioxide is picked up, and in the lungs, where it is released. Most carbon dioxide diffuses into RBCs, is combined with water and, through the action of a catalyst called carbonic anhydrase, and forms carbonic acid (H_2CO_3). Carbonic acid immediately dissociates into hydrogen ions (H^+) and bicarbonate ions (HCO^-_3) that can diffuse back into the plasma or stay in combination with a positive ion in the RBCs. The excess hydrogen ion formed in this reaction usually binds with the hemoglobin molecule to form hydrogen hemoglobin. If significant amounts of bicarbonate ions diffuse into the plasma, a negative ion is drawn into the RBCs to equalize the electrochemical gradient. This ion is usually chloride, and the mechanism of its movement is called the chloride shift. This process can provide bicarbonate to the plasma when the pH is decreased. Normally, in the lungs a reverse process occurs very rapidly. Hydrogen is released from hemoglobin and recombines with bicarbonate to form carbonic acid, which dissociates into carbon dioxide and water. Carbon dioxide diffuses out of the RBCs into the alveolar-capillary interspaced and is blown off in the exhaled air.

Under normal alveolar ventilation conditions, alveolar carbon dioxide (P_ACO_2) of 40 mmHg is in equilibrium with the resulting arterial carbon dioxide (PaO_2) of 40 mmHg. The levels of both alveolar and arterial carbon dioxide

are directly and inversely proportional to the volume of alveolar ventilation. Thus, in alveolar hypoventilation, having the alveolar ventilation from 4 to 2 L/minute doubles the P_aCO_2 from 40 to 80 mmHg. In hyperventilation, doubling the alveolar ventilation from 4 to 8 L/minute halves the P_aCO_2 to 20 mmHg (Mackin & Bullock, 2000).

Consequently, the tissue receives enough oxygen depends on functioning of lungs and heart. If there is abnormality in the mechanism of respiratory may cause hypoxia. Besides, the endotracheal intubation affects the upper airway to dried, the secretion is tenacious and dried leading to the retention and obstruction of airway. So, the suctioning is necessary in order to widen airway, and well gas exchange between alveolar and capillary. This is to reduce and prevent hypoxemia. However, the nurse should be knowledgeable and expert in nursing in order to prevent danger from suctioning.

Artificial Airway

In the pediatric patients with respiratory problem or another disease that affects the functioning of respiratory system which leads to inability to breathe or insufficient breathing, they need help through endotracheal intubation with mechanical ventilation. The endotracheal intubation is beneficial (Champagne, 2000; Jammaree Theeratakulpisan, B.E. 2543; Unchalee Yuengsrikul, B.E. 2545) in order to:

1. Reduce work of breathing
2. Provide effective ventilation
3. Provide gas exchange and sufficient oxygen delivery
4. Prevent aspiration in unconscious patients
5. Reduce the expansion of stomach
6. Use as the medication administration

However, the endotracheal intubation may affect the body badly as follows (Mukda Suwankosit, B.E. 2533; Suwannee Jaroongjitarree, B.E. 2530):

1. Lose of defense on trachea, as the trachea is bypassed with endotrachael tube
2. Damage to airway membrane
3. Stimulate to create more secretion
4. Reduce the function of cilia

5. Inefficient cough due to epiglottis cannot close tightly

6. Make the swallowing abnormal

7. Risk of respiratory infection

Therefore, the endotracheal intubation may cause the airway obstruction, because of the secretion is retention. In addition, the patients may be atelectasis or respiratory infected resulting in the danger of lives. Consequently, the removal of secretion is necessary, which we can manage in various methods. For example paying attention to the patient's hydration status to ensure adequate pulmonary hydration, ensuring airway heat and humidity, using nebulizer treatments, and stimulating the patients to cough and dislodge secretion including suctioning.

Endotracheal Suctioning, Complications from Suctioning and Managements

Endotracheal suctioning

Endotracheal suctioning is very important for releasing secretion of the patients who had tenacious secretion and cannot cough it out by himself. It aims to (Kozier & Erb, 1987)

1. Open the airway and prevent the airway obstruction

2. Promote optimal ventilation and oxygenation

3. Prevent pneumonia and atelectasis from retention of secretion

Complications from suctioning and managements

The endotracheal suctioning may lead to complications. Mostly, the cause of complication is due to inappropriate suctioning, such as the suctioning takes very long time, the suction catheter has large size, or the suction catheter inserts too deeply. These may lead to damage or harmful to death.

1. Hypoxemia: When the suction catheter is placed in the airway and a vacuum applied, oxygen-enriched gas is sucked out of the lungs and replaced by room air that enters around the catheter. The concentration of oxygen in airway and alveolar has reduced considerably whilst the exchange of gas between alveolar and blood vessel has also reduced. The symptoms of patients are as follows (AARC, 1993; Dean, 1997; Wood, 1998; Young, 1995):

1.1 Central nervous system: When the body has the reduction of oxygen saturation, it will intervene with the perception in early stage leading to anxiety, fatigue, headache, unconscious, or faint at last.

1.2 Cardiovascular system: The cardiac muscle will increase functioning in order to compensate oxygen for tissues. In the early stage of hypoxemia, the body will experience tachycardia or arrhythmia, chest pain, and hypotension that may cause arrest.

1.3 Respiratory system: Hypoxemia stimulates the respiratory system leading to increase of respiration rate, pant, fatigue, and unconscious.

Managements

1. Expanse the volume of lungs by giving 3-4 breaths of the positive pressure ventilation manually through resuscitating bag or use 100% of oxygen in sigh mode before suctioning, during and after endotracheal suctioning.

2. The suction catheter should not be large than a half of the internal diameter of the endotracheal tube.

3. The period of suctioning should not be more than 10 – 15 minutes.

2. Dysrhythmia: Significant cardiac arrhythmias may occur during the suctioning process from two sources: (1) arterial hypoxemia leading to myocardial hypoxia or (2) vagal stimulation secondary to tracheal irritation. When vagal nerve is stimulated, it affects the bradycardia or dysrhythmia immediately. Then, the body is compensated by negative feedback in which the sympathetic nervous system is activated leading to increase heart rate (Wood, 1998).

Managements

1. The period of suctioning should not be more than 10 – 15 seconds.

2. The depth of suction catheter should be 1 centimeter above the carina and the suction catheter should not be inserted too deeply. It may stimulate the vagus nerves causing dysrhythmia.

3. The abnormal blood pressure: Hypotension may occur from either of the two circumstances: (1) profound bradycardia resulting from vagal stimulation; or (2) prolonged coughing maneuvers during the suctioning process. Tracheal irritation from

the suction catheter may stimulate tracheal and carinal reflexes resulting in paroxysmal cough-like maneuvers which interrupt ventilation. These coughing maneuvers, along with bradycardia, can have a severe effect on both venous return and cardiac output (Shapiro, Harrison & Trout, 1983).

Managements

1. The period of suctioning should not be more than 10 – 15 seconds.

2. The depth of the suction catheter should be 1 centimeter above the carina and the suction catheter should not be inserted too deeply.

4. Lung collapse: The insertion of a large suction catheter into a small diameter of the endotracheal tube results in inadequate space for air to readily entrain around the catheter. Thus, when a vacuum is applied, the lungs may collapse (Allen, 1998).

Managements

The suction catheter should not be larger than a half of the internal diameter of the endotracheal tube.

5. Mucosal damage: Tracheitis may arise from using too high negative pressure from suctioning or frequently suctioning. These mucosal areas immediately become hemorrhagic and may erode leading to the death of tissue and tracheal stenosis (Young, 1995).

Managements

1. The negative pressure in suctioning is appropriate for each age, not over 120 mmHg in children, and not over 140 mmHg in adult.

2. The end of the suction catheter should be smooth and round; there must be at least 2 holes in order to prevent mucosal damage.

6. Respiratory infection: If the personnel are careless on aseptic technique during suctioning and contaminated equipments, this shall lead to infection easily. Because of the endotracheal intubation is the bypass of defense mechanism, and this is the way for bacteria into lungs directly (Demers & Saklad, 1973).

Managements

Utilize aseptic technique for both method and equipment used in suctioning.

7. Create more secretion from mucus gland is stimulated by frequent suctioning leading to the increase of obstruction of airway.

Managements

Assess the lung sound in order to evaluate the necessity in suctioning.

8. Increase intracranial pressure: Normally, the blood circulation at the brain of pediatric patients, especially the infant will be sensitive to the change of blood pressure leading to the increase of intracranial pressure and it tends to have bleeding in brain more easily.

Managements

1. Assess the lung sound in order to evaluate the necessity in suctioning.

2. The period of suctioning should not be more than 10-15 seconds and the suction catheter should not be inserted too deeply.

9. Bronchospasm: Because of the interruption or stimulation of airway frequently, the incident depends on the period of suctioning (Demers & Saklad, 1973).

Managements

Assess the lung sound in order to evaluate the necessity in suctioning.

10. Psychological problem: Causing fear and anxiety (Bostick & Wendelgass, 1987) that relates to dysrhythmia.

Managements

1. Inform the patient before suctioning to reduce fear and anxiety.

2. Suctioning softly.

The indications of endotracheal suctioning (Day, Farnell & Wilson-Barnett, 2002; Glass & Grap, 1995; Griggs, 1998) are as follows:

1. Visible or audible secretions
2. Feeling secretions in the chest (by the patient) and restlessness
3. Increased airway pressure when ventilated and decreased tidal volume
4. Diminished air entry or altered chest movement

5. Decreased oxygen saturation levels and deteriorated arterial blood gas values

6. Altered hemodynamics, including increased blood pressure and tachycardia

Suctioning may frequently lead to hypoxemia, which can cause cardiac dysrhythmias, and even cardiac arrest and death. Strategies used to minimize these effects include hyperoxygenation or hyperinflation.

Hyperoxygenation involves the administration of oxygen at a greater percentage or fraction of inspired oxygen (FiO_2) than the patient has been currently receiving (Glass & Grap, 1995; Wood, 1998). This may be performed before (pre-oxygenation), during (insufflation) and/or after the procedure (post-oxygenation). Several researchers have examined this issue. The patient receives hyperoxygenation before suctioning so that the difference between the pressure of oxygen in alveolar and capillary increase; thus, leading the oxygen to move inside more. It is to reserve the amount of oxygen in the lungs in order to prevent the hypoxemia during and after suctioning by 100 %O_2 in sigh mode from ventilator or giving 3-5 breaths of the positive pressure ventilation manually through the resuscitating bag (Brown, 1983; Day, Farnell & Wilson-Barnett, 2002; Moore, 2003; Runton, 1992; Skelley, 1980). Besides, Mukda Suwankosit (B.E. 2533) studied to compare of oxygen saturation after suctioning between no preoxygenation via the ventilator, hyperinflation by manual resuscitating bag with oxygen 10 L/minute, and hyperoxygenation with 100% oxygen via the ventilator for 1 minute before suctioning in 30 critically ill patients in surgical-trauma intensive care and neuro-surgery intensive care setting. It was found that the hyperinflation by 3 breaths of the positive pressure ventilation manual by resuscitating bag results in the oxygen saturation after suctioning reduce less than no preoxygenation via the ventilator. Hyperoxygenation with 100% oxygen via the ventilator for 1 minute before suctioning results in the oxygen saturation levels increases after suctioning. That corresponds to Chanokporn Hanchanchaikul (B.E. 2534) who suggested that hyperoxygenation with 20% above the patient's baseline FiO_2 for 1 minute before suctioning and hyperoxygenation with 100% for 15 seconds can prevent hypoxemia too.

The duration of suctioning is important in reducing and preventing hypoxemia. Adlkofer and Powaser (1978) studied in 54 patients who underwent heart and capillary surgery to whom received non-hyperoxygenation before endotracheal suctioning and used negative pressure between 100 to 120 mmHg. It was found that 24 patients had arterial oxygen tension reduce less than 10 mmHg in suctioning for 10.1 ± 2.7 seconds, 14 patients had arterial oxygen tension reduce between 10 to 20 mmHg in suctioning for 11.5 ± 3.5 seconds, and 16 patients had arterial oxygen tension reduced for more than 20 mmHg in suctioning for 13.3 ± 6.1 seconds. The majority of researchers recommended that suctioning should take between 10 and 15 seconds to perform, as longer durations were associated with an increased risk of mucosal damage and hypoxemia (Day, Farnell & Wilson-Barnett, 2002; Moore, 2003; Young, 1995).

Besides, the depth of the suction catheter insertion that is necessary in suctioning. Kleiber, Krutzfield and Rose (1988) compared the histological evidence of acute mucosal damage caused by the calibrated catheter insertion method and by the commonly used resistance insertion method. The intubated kittens were subjected to one of two procedures: (1) insertion of a suction catheter to a predetermined distance and withdrawal with and without the application of suction or (2) insertion of the catheter until resistance was met and withdrawal with or without the application of suction. All of the animals in the predetermined distance groups had normal tissues. The animals in the resistance groups displayed multifocal areas of denuded epithelium and varying degrees of inflammation. Thus, several researchers recommended the suction catheter should be inserted 1 cm above the carina (Day, Farnell & Wilson-Barnett, 2002; Day, Wainwright & Wilson-Barnett, 2001; Dean, 1997; Runton, 1992; William, 2004; Wood, 1998).

The endotracheal suctioning techniques

1. Assess the lung sound to evaluate the necessity in suctioning.

2. Increase the pressure of oxygen in the alveolar by hyperoxygenation so order that the difference between the pressure of oxygen in alveolar and capillary increase lead to oxygen move inside more. It is to reserve the amount of oxygen in the lungs in order to prevent the hypoxemia during and after suctioning by 100 %O_2 in

sigh mode from ventilator or giving 3-5 breaths of the positive pressure ventilation manual through the resuscitating bag (Chanokporn Harncharnchaikul, B.E. 2534; Mukda Suwankosit, B.E. 2533; Day, Farnell & Wilson-Barnett, 2002; Moore, 2003; Runton, 1992).

The size of manual resuscitating bag (Suparat Viyacheta, B.E. 2544)

- Infant (premie to 1 year): 250-500 ml
- Small child (2-5 years): 500 ml
- School-age child (6-12 years): 500-1,000 ml
- Adolescent to adult: 1,000 ml

3. Use aseptic technique in suctioning. This entails the use of sterile glove for the hand that will handle the catheter and the use of sterile suction catheter (Boggs & King, 1993; Imle & Klemic, 1989; Mckillop, 2004).

4. The size of the suction catheter should not be more than a half of the internal diameter of the endotracheal tube (Mckillop, 2004; Runton, 1992).

Size of Endotracheal Tube (mm)	Size of Suction Catheter (Fr)
2.5	6
3.0	6.5
3.5	6.5
4.0	8
4.5	8
5.0	8
5.5	10
6-6.5	10-12
7-7.5	12-14

Besides, the depth of the suction catheter should be inserted 1 cm above the carina (Day, Farnell & Wilson-Barnett, 2002; Day, Wainwright & Wilson-Barnett, 2001; Dean, 1997; Runton, 1992; William, 2004; Wood, 1998) or inserting catheter with the end of suction catheter passing endortracheal tube around 0.25 – 0.5 inches and use the tape to make for appropriateness of the depth of the suction catheter insertion.

5. Apply negative pressures in children of various ages (Boggs & King, 1993; Celik & Elbas, 2000) as follows:

- 50 – 70 mmHg in the infant
- 70 – 90 mmHg in the early stage children
- 90 – 120 mmHg in the late stage children

6. The suction catheter should not be pulled down during suctioning.

7. Insert the suction catheter when the patient inhales and not sucking during insertion of the catheter into the endotracheal tube. Because the oxygen is sucked out of the airway, this may lead to hypoxemia and the mucosal damage (Moore, 2003).

8. The duration of suctioning should not be more than 10 – 15 seconds (Day, Farnell & Wilson-Barnett, 2002; Mckillop, 2004; Moore, 2003; Young, 1995).

9. Assess the lung sound in order to evaluate the effectiveness of suctioning. For each time of suctioning, the suction catheter should not be inserted for more than 3 times or too long. This can help protect hypoxemia and other complications from suctioning (Day, Wainwright & Wilson-Barnett, 2001; Glass & Grap, 1995; Moore, 2003).

10. During suctioning, there must be close observation and record of heart rate. If the patient experiences dysrhythmia or laryngospasm, the suctioning must be stopped.

Suctioning is very important and essential for the patient who had endotracheal intubation with the mechanical ventilation in order to help maintain the airway. The nurse should assess the necessity for suctioning and reasonable practice. For example, the instillation of normal saline in order to reduce tenacious secretion should be performed as necessary and suctioning should not be performed in routine.

Normal Saline Instillation before Endotracheal Suctioning

Normal saline instillation before endotracheal suctioning is aimed to (Ackerman, 1993; Bostick & Wendelgass, 1987; Demers & Saklad, 1973; Kinloch, 1999)

1. Loosen or thin secretion
2. Stimulate a cough
3. Lubricate the suction catheter

The amount of normal saline instillation depends on the age of the subject as follows:

1. The children less than 1 year old use 0.25 – 0.5 ml (Ridling, Martin & Bratton, 2003; Shorten, 1991).

Besides, using normal saline in infant can be calculated for 0.1 – 0.2 ml/kg (Hodge, 1991 referred in Young, 1995).

2. The children 1 – 8 years old use 0.5 ml (Ridling, Martin & Bratton, 2003).

3. The children more than 8 years old use 1 – 2 ml (Ridling, Martin & Bratton, 2003)

4. Adults use 2 – 5 ml (Tolles & Stone, 1990 referred in Blackwood, 1999; Schwenker, 1998).

Normal saline instillation is intended as a beneficial in removing secretions. On the contrary, normal saline instillation may cause potential adverse effects on physiological functioning and psychological wellbeing such as hypoxemia, the risk of lower airway infection, and patient discomfort (Ackerman, 1993; Bostick & Wendelgass, 1987; Blackwood, 1999; Demers & Saklad, 1973).

Thus, nurses should further develop and focus on alternative methods of dryness preventions and tenacious secretions in the mechanically ventilated patient. Alternative methods that controlling the tenacity of secretions should be considered (Blackwood, 1999) are as follows:

- Paying attention to the patient's hydration status in an effort to ensure adequate pulmonary hydration
- Ensuring airway heat and humidity
- Using mucolytic agents and nebulizer treatments

The patient with an artificial airway does not have the availability of the upper airway to heat and humidify the inspired air. This heat and humidity deficit must be provided to the tracheobronchial tree, unless otherwise provided. It is essential that the gas delivered to an artificial airway be 100% humidified at body temperature. Without appropriate humidification of the artificial airway, the incidence of obstruction caused by drying of secretions will be great. With appropriate heating and humidification of inspired gas, the incidence of crusting and obstructing of the airway in secretion is extremely rare.

Several researches suggested that an optimal level of humidity (core temperature 37°C) and 100% relative humidity (44 mg/L) offered significant therapeutic benefits to the patient such as improved mucocilliary clearance (Shapiro et al., 1983; Williams et al.,1996). Besides, Wattana Nampet (B.E. 2531) studied the effects of heated humidification on recovery time of body temperature and sputum viscosity in 30 postoperative open-heart patients. The result showed that the experimental group who had received heated humidification on recovery time of body had the temperature up to 37 °C less than the controlled group who had received normal humidification with statistical significance (<.05), and the sputum viscosity in experimental group was less than the controlled group with statistical significance (<.05). Therefore, we should use heated-humidifier in the mechanical ventilation as this may cause the function of airway to be normal.

Normal saline instillation before endotracheal suctioning is beneficial in removing secretions. However, the nurse should instill the normal saline, when tenacious secretions are present due to the disadvantages of the normal saline instillation which lead to hypoxemia, increase of carbon dioxide tension, patient discomfort including increase the risk of lower airway infection.

Impacts of Endotracheal Suctioning with Normal Saline Instillation

The instillation of normal saline before endotracheal suctioning has become a common practice in some areas (Ackerman, 1993, Ackerman et al., 1996). The rational is that saline will thin tenacious secretion and assist in removal of them. However, several researchers try to indicate the disadvantage of normal saline instillation such as hypoxemia, increase of carbon dioxide tension, the increate of heart rate and blood pressure, discomfort and increase the risk of lower airway infection as well (Ackerman & Mick, 1998; Akgul & Akyolcu, 2002; Blackwood, 1999; Bostick & Wendelgass, 1987; Briening, 1996; Gray, MacIntryre & Kronenberger, 1990; Hagler & Traver, 1993; Kinloch , 1999; Ridling, Martin & Bratton, 2003; Schwenker, 1998).

Normal saline in the form of drop of water with large particle, mostly it will mostly remain in the airway. The hyperinflation of patient's lungs by manual resuscitating bag for 1 time has inadequate pressure in making drop of water to be

broken in small particles and distributed around the lungs. All of the normal saline remained in the trachea and main bronchi, and none of it reached the periphery of the lung (Hanley, et al., 1978). Demers and Saklad (1973) suggested that the mucus and water in bulk form are immiscible, even after vigorous shaking. The form of an aerosol is offered because it can dissolve secretion in airway. Besides, the dangerous of normal saline instillation as precaution is airway obstruction due to secretion has large particle from water absorption and bronchospasm.

The endotracheal suctioning with normal saline instillation affects to oxygen saturation, carbon dioxide tension, heart rate, blood pressure, respiratory rate, and patient discomfort. In addition, these affect the change of various parameters such as lung compliance, tidal volume, minute volume, force vital capacity, positive inspiratory airway pressure, and airway resistance.

Oxygen saturation

Several researches studied the effects of instillation of normal saline before endotracheal suctioning on arterial oxygen tension and oxygen saturation. These tended to decrease after instillation of normal saline (Ackerman, 1993; Ackerman & Mick, 1998; Bostick & Wendelgass, 1987; Kinloch, 1999; Ridling, Martin & Bratton, 2003; Schwenker, 1998). Ackerman (1993) studied in 40 adult patients post open-heart surgery, and found that oxygen saturation was decreased at 2, 3, 4, and 5 minutes after endotracheal suctioning with normal saline instillation. Then she recommended that the instillation of normal saline not be routine or standard intervention and that it should be regarded as potentially hazardous. Ackerman and Mick (1998) evaluated the effect of instillation of normal saline before suctioning in 29 adult critically ill patients with pulmonary infections, and found that oxygen saturation was decreased at 4, 5, and 10 minutes after endotracheal suctioning with normal saline instillation. In the pediatric patient study, Ridling, Martin, and Bratton (2003) studied the effects of the endotracheal suctioning with and without normal saline instillation in 24 critically ill children, and found that oxygen saturation was decrease at 1 and 2 minutes after endotracheal suctioning with normal saline instillation. Kinloch (1999) studied in 35 patients after coronary bypass grafting, and found that the recovery time of oxygen saturation to return to baseline after endotracheal suctioning without and with normal saline instillation were average of 3.78 minutes and 7.30 minutes respectively. Besides,

Ji, Kim and Park (2002) studied the effects of normal saline instillation before suctioning on oxygen saturation in 16 critically ill patients with pneumonia. Their results showed that the recovery times for oxygen saturation to return to baseline after suctioning without normal saline instillation at 45 seconds and excess of 5 minutes in the case of normal saline instillation. They suggested a downward trend of arterial oxygen tension (PaO_2) in patients who received a larger amount of solution. Because of the amount of normal saline, this may be more obstructing to the exchange of oxygen between capillary and alveolar.

Thus, the endotracheal suctioning with normal saline instillation reduces the oxygen saturation due to that the normal saline instilled cannot mix with the secretion and cannot suction all. The remaining of normal saline causes the obstruction of the gas exchange.

Carbon dioxide tension

Several researches studied the effects of endotracheal suctioning with normal saline instillation on carbon dioxide tension. Gray, MacIntryre and Kronenberger (1990) studied the effects of endotracheal suctioning with normal saline instillation on carbon dioxide tension in 15 adult critically ill patients with lung problems. It was found that the carbon dioxide tension increased after suctioning immediately with statistically significant difference, but later the differences in carbon dioxide tension between the both methods were not statistically significant. This result was in according with the data from Akgul and Akyolcu (2000) studied the effects of endotracheal suctioning with and without normal saline instillation on carbon dioxide tension in 20 adult critically ill patients with pulmonary or cardiovascular problems. They found that carbon dioxide tension of endotracheal suctioning with normal saline instillation increased more than the endotracheal suctioning without normal saline immediately with statistically significant difference, but later the differences in carbon dioxide tension between the both methods were not statistically significant.

However, the carbon dioxide tension increases due to the normal saline is foreign agent that falls into the endotracheal tube. The normal saline cannot be suctioned to clear. If the airway has a lot of normal saline retention, the patient may experience discomfort in respiration. Because of the airway obstruction may interrupt the diffusion of oxygen and carbon dioxide between alveolar and capillary causing the

reduction of gas exchange and ventilation; these may affect the increase of carbon dioxide tension and hypoxemia.

Heart rate and blood pressure

Several researches reported the effects of endotracheal suctioning with normal saline instillation on heart rate and blood pressure. Gray, MacIntryre and Kronenberger (1990) studied the effects of endotracheal suctioning with normal saline instillation on heart rate and blood pressure in 15 adult critically ill patients with lung problems. They found that the heart rate and blood pressure increased immediately, but there were not statistically significant differences. Besides, there had been a study on the infant patients; the result was that the heart rate and blood pressure increased with no statistical significance (Beerman & Dhanireddy, 1992; Shorten, Byrne & Jones, 1991). Similarly, Ackerman and Mick (1998) studied the effects of instillation of normal saline before suctioning in 29 adult critically ill patients with pulmonary infections; it was found that the heart rate and systolic pressure increased although the differences between the groups were not significant.

Saowaluk Jiratamkul (B.E. 2534) studied the effects of endotracheal suctioning with or without normal saline instillation on the heart rate, systolic blood pressure, oxygen saturation, and sputum viscosity in 20 postoperative opened heart surgery patients with mechanical ventilator. It was found that the suctioning with normal saline instillation led to the higher heart rate than the suctioning without normal saline instillation was statistical significance ($P < .01$). Nevertheless, systolic blood pressure was not different. Akgul and Akyolcu (2000) studied the effects of endotracheal suctioning with and without normal saline instillation on heart rate in 20 adult critically ill patients with pulmonary or cardiovascular problems. They found that heart rate was increase at 4 and 5 minutes after endotracheal suctioning with normal saline instillation with a statistically significant difference.

Endotracheal suctioning with normal saline instillation result in the increase of heart rate and blood pressure. Due to its vagal stimulation, the body is compensated by negative feedback. Because these lead to a generalized increase of central nervous system arousal, signals from the hypothalamus transmit to autonomic control centers of the brain stem. In early stage, endotracheal suctioning with normal saline instillation affects to oxygen desaturation and vagal stimulation which result in

bradycardia and hypotension immediately. Then, the body has compensation by negative feedback. It affect directly to the increase of depolarization rate in SA node and stimulate the arterial chemoreceptor, the sympathetic nerve system is activities. Epinephrine and norepinephrine are related to stimulation of the sympathetic nerves. These hormone increases in the blood circulation. Thus, the heart rate and blood pressure increases in order to correct hypoxia.

Aforementioned, the effect of normal saline instillation before endotracheal suctioning affects to increase the risk of lower airway infection and patient discomfort. Hagler and Traver (1994) studied the effect of normal saline instillation before endotracheal suctioning on the lower airway infection in 10 critical patients who had intubated for at least 48 hours. The result of this study found that the suction catheter insertion dislodged up to 60,000 viable bacterial colonies. However, the use of 5 ml normal saline instillation dislodged up to 310,000 viable colonies. This evidence suggested that mechanically ventilated patients were at the risk of a nasocomial infection by dislodging bacteria into the lower airways. Besides, Wade (1982) suggested about patients' feeling a sensation of drowning or suffocation with normal saline instillation. Moreover, Gray, MacIntryre and Kronenberger (1990) attempted to measure the patient's discomfort during normal saline instillation on a scale of 1 (least uncomfortable) to 6 (most uncomfortable). However, the concept of discomfort was not operationally defined and no clarification was given regarding the design of the scale (i.e. verbal report, ruler scale) nor its reliability or validity. The findings suggested that patients found normal saline instillation more uncomfortable than without normal saline instillation (4 VS 3); the difference was not statistically significant.

However, the normal saline instillation before endotracheal suctioning cannot identify the advantage and disadvantage clearly. Hence, there should be further study on the subjected matter. Nowadays, many modern types of equipment are convenient, quick, and more efficiently use to assess the effects of normal saline instillation such as monitor, pulse oximeter, and capnometer. These equipments can assess the change of oxygen saturation, carbon dioxide tension, heart rate, and blood pressure without hurting patient.

Assessments of Oxygen Saturation, End-tidal CO_2, Heart rate, and Mean Blood pressure

Currently, monitoring is the permanent activity in continuously and evaluating the physiologic function of the patient in order to guide management decisions and to assess the impact of treatment. Monitoring may be invasive or noninvasive.

Invasive monitoring penetrates the body of the patient such as arterial blood pressure, central venous pressure (CVP), and pulmonary artery.

Noninvasive monitoring does not penetrate the body. They typically rest on the skin of the patient such as pulse oximeter, capnometer, noninvasive blood pressure (NIBP), and bedside monitoring. In this noninvasive monitoring, there is no pain, whilst reduce complications and reduce expenses including increase of comfort more than invasive monitoring.

Oxygen saturation: SpO_2

Basic principles of oxygenation are best explained in relation to the partial pressure of oxygen (PO_2) and the percentage of hemoglobin oxygen saturation (SpO_2).

Oxygen in the blood is carried in two ways: dissolved in the liquid part of the blood plasma and in chemical combination with hemoglobin. Most oxygen is transported in the second manner.

The amount of dissolved oxygen carried in the plasma is directly proportional to the partial pressure of oxygen (Henry's law). There is 0.003 ml of oxygen dissolved in each 100 ml of blood for each 1 mmHg partial pressure of oxygen. Thus at an ideal PaO_2 of 100 mmHg, only 0.3 ml of oxygen would be carried per 100 ml of plasma. The individual's normal resting cardiac output is 5 L/minute.

Most of oxygen in the body is transported to the cells in combination with hemoglobin. Oxygen combines loosely and reversibly with the heme portion of hemoglobin. When the PO_2 is low, as in the tissue capillaries, the oxygen is released from the hemoglobin.

The amount of oxygen carried in the blood by hemoglobin is directly dependent on the concentration of hemoglobin. The average individual has about 15 g of hemoglobin in each 100 ml in blood. Each gram of hemoglobin has the maximum capability to combine with 1.34 ml of oxygen. Therefore a hemoglobin level of 15

g/ 100 ml would result in 20.1 ml of oxygen combined with hemoglobin and saturation (Hazinski, 1992).

The percentage of hemoglobin saturation with oxygen maybe abbreviated as % HbO_2 saturation. In a clinical setting, oxygen saturation is defined as the amount of hemoglobin actually combined with oxygen and divided by the amount of hemoglobin available. The formula is written as follows:

$$\% \ HbO_2 \text{ saturation} = \frac{\text{Hb combined with oxygen}}{\text{Total hemoglobin}} \times 100\%$$

The relationship between the PaO_2 and the hemoglobin saturation is expressed by the oxyhemoglobin dissociation curve as shown in Figure 3. The curve is not linear; instead, it is S shaped, with a large plateau at the higher levels of PaO_2. There are several important parts of the oxyhemoglobin dissociation curve. The curve flattens when the PaO_2 exceed 80 to 100 mmHg. This means that although the PaO_2 continues to rise beyond 100 mmHg, the hemoglobin cannot become more saturated than 100%; it cannot carry any more oxygen. Thus, any further rise in the PaO_2 will result in only small increases in the amount of dissolved oxygen in the blood. Therefore a rise in PaO_2 from 100 to 700 torr does not mean the seven times more oxygen is carried in the blood. In fact, this rise is associated with only approximately a 10% increase in oxygen content. Because the hemoglobin is fully saturated once the PaO_2 reaches 100 mmHg, there is usually no advantage in maintaining the patient's PaO_2 any higher than this value.

The slope of the oxyhemoglobin dissociation curve becomes very steep once the PaO_2 is less than 60 mmHg. Thus when the patient's PaO_2 falls below 60 mmHg, even small decreases in the PaO_2 are associated with a significant fall in the hemoglobin saturation and the arterial oxygen content. Therefore, if possible, the patient's PaO_2 should be maintained above 60 mmHg.

The shape of the oxyhemoglobin curve may be altered by several factors. If the curve is shifted to the right, this means that hemoglobin binds less oxygen at any partial pressure of oxygen. Conversely, if the curve is shifted to the left, hemoglobin binds more oxygen at any given PaO_2. Factors that shift the curve to the right include

acidosis, hypercapnia, and hyperthermia. Under these conditions, less oxygen is bound at any given PO_2, but within the normal range the amount of oxygen released to tissues is enhanced. In contrast the oxyhemoglobin dissociation curve may be shifted to the left by alkalosis, hypocapnia, and hypothermia. While these factors increase hemoglobin saturation with oxygen at any given partial pressure of oxygen, hemoglobin release to tissues may be impaired (Hazinski, 1992).

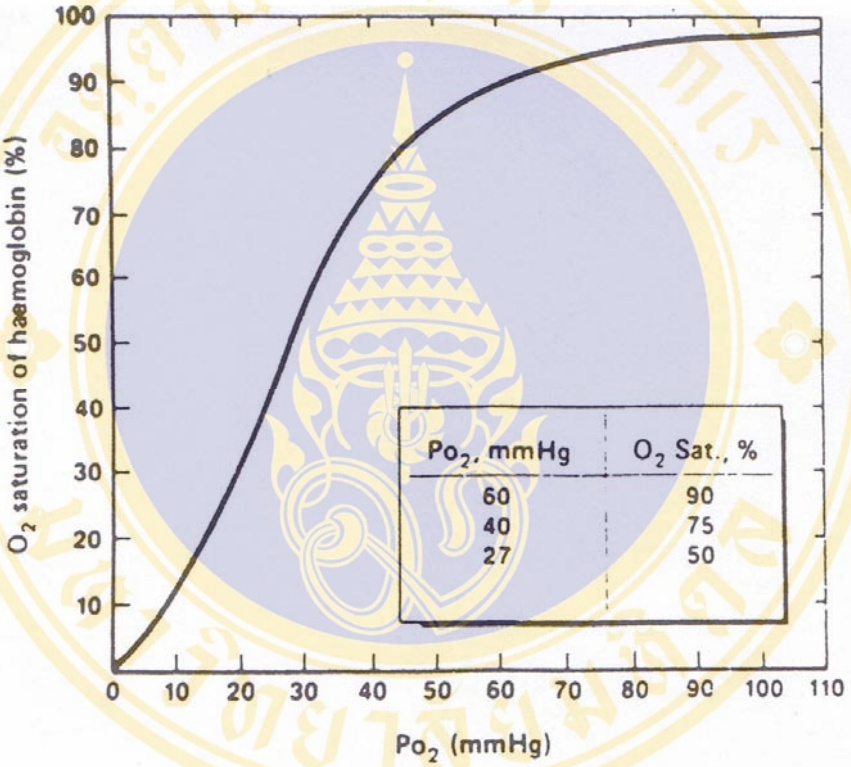

Figure 3: Oxygen-hemoglobin dissociation curve (Supitcha Sangchote, B.E. 2543: 109)

Pulse oximeter

Pulse oximeter is a device that provides continuous and noninvasive measurements of arterial oxygen saturation.

Pulse oximeter is based on two primary principles of light transmission and reception called spectrophotometry and photoplethysmography. It is used to measure arterial oxygen saturation non-invasively. Spectrophotometry measure the percentage of oxygenated hemoglobin in the blood and photoplethysmography is used to differentiate arterial from venous blood.

The measurements of hemoglobin and its derivatives by spectrophotometry are based on the Lambert-Beer laws of absorption. This law combines Lambert's law and Beer's law, both of which describe transmission of radiation through an absorbing substance. Lambert's law states that the optical density of a homogeneous medium is directly of a homogeneous absorbing substance. Lambert-Beer laws specify the relationship of the optical density to both path length and concentration of the absorbing substance.

Spectrophotometry can be used to accurately measure hemoglobin and its derivatives. Given the condition according to the Lumbert-Beer laws that the blood must be hemolyzed before analysis, it must be withdrawn from a subject. These in vitro measurements of hemolyzed blood by spectrophotometry are conducted by a device called co-oximeter or hemoximeter. Photoplethysmography uses reflection or transmission of light through vascular tissue to measure arterial pressure waveforms generated by the cardiac cycle (McCarthy, Decker, & Stoller, 1993).

Pulse oximeter estimates arterial oxygen saturation by measuring the absorption of light (of 2 wavelengths, approximately 660 nm and 940 nm) in human tissue beds. The wavelengths of red light (600 nm) is more absorbed by deoxygenated hemoglobin than by oxygenated hemoglobin, whereas the wavelengths of infrared light (940 nm) is more absorbed by oxygenated hemoglobin than by deoxygenated hemoglobin. As light passes through human tissue, it is absorbed in various degrees by tissue, bone, blood vessels, fluids, skin, venous blood, and arterial blood, including various types of hemoglobin. The light absorption changes as the amount of blood in the tissue bed changes and as the relative amounts of oxygenated and deoxygenated hemoglobin change. Measuring the changes in light absorption allows estimation of heart rate and arterial oxygen saturation. To measure accurately, the oximeter must distinguish between the background (or constant) absorption and the pulsatile changes in absorption caused by the changing blood volume with each heartbeat. The background absorption can change when there is a change in the shape or position of the tissues through the light passes, which can cause false readings (Salyer, 2003).

Accuracy of pulse oximeter

There are numerous studies of the accuracy and precision of pulse oximeters in various populations. The methods for describing accuracy are differed in those studies, making an overall summary of accuracy challenging. There are considerable performance differences among the various brands of pulse oximeter, which are probably due to differences in the signal processing software and calibration curves. The bias of the pulse oximeter is the mean difference between simultaneous pulse oximetric and co-oximetric measurements of oxygen saturation. Most manufactures claim confidence limits in any given pulse oximeter reading of ± 4% for readings above 70%. At the oxygen saturation below 80%, the accuracy is worse, although its clinical importance is still questionable (Salyer, 2003).

Limitations of pulse oximeter

Pulse oximeter has several well known limitations as follows (Aroonwan Pruettipan, B.E.2545; Salyer, 2003):

1. Poor perfusion

The accuracy of pulse oximeters depends on the function of pulsating vascular bed. Under the conditions of low blood flow such as cardiac arrest or severe peripheral vasoconstriction, pulse oximeter becomes unreliable.

2. Ambient light

If the finger probe of pulse oximeter does not fit correctly, light can be shunted directly from the LEDs to the photodetector. This will cause a falsely low or elevated SpO_2. High intensity of light from fluorescent lights and operation-room lights has been reported to interfere with pulse oximeter performance.

3. Dyshemoglobinemia

Because pulse oximeters are unable to distinguish between oxygenated hemoglobin and the various dysfunctional hemoglobins, such as methemoglobin and carboxyhemoglobin, they are unable to bind with and carry oxygen.

4. Skin pigmentation

As skin pigmentation darkens, pulse oximeter performance deteriorates. Several studies have shown that the accuracy and performance of pulse oximeters are affected by deeply pigmented skin.

5. Motion artifact

Motion of probe can produce unreliable and inaccurate pulse oximeter readings.

6. Anemia

Although pulse oximeters are generally reliable over a wide range hemoglobin levels, they become less accurate and reliable with conditions of severe anemia (Hb < 8 g/dl at low saturations, and hematocrit < 10% at all saturation).

End-tidal CO_2

Carbon dioxide tension (PCO_2) can identify the alveolar ventilation level.

Capnometry is the measurement of carbon dioxide on the patient's airway during the ventilatory cycle. The capnometer provides a numeric display of inhaled PCO_2. Capnography is the graphic waveform display of carbon dioxide. (Supitcha Sangchote, B.E. 2540). Most bedside capnometers used in respiratory care measure carbon dioxide by infrared absorption. Capnometer has two types as follow:

1. The mainstream capnometer, the measurement chamber is placed directly at the airway. This results in a very crisp capnogram that is generated almost instantaneously. However, some problems are associated with this configuration. The mainstream sensor is heavy which causes it to kink of endotracheal tube easily.

2. The sidestream capnometer, gas from the airway is aspirated through fine-bore tubing to the measurement chamber inside the device. This elimination may be the problems associated with the mainstream sensor. However, it introduces other problems related to the aspiration of gas from airway. The sample tubing tends to become obstructed with secretions and water, and a delay in airway to the measurement chamber (Aroonwan Pruettipan, B.E. 2545).

The normal capnogram is illustrated in Figure 4. During inspiration, PCO_2 is zero. At the beginning of exhalation, PCO_2 remains zero as gas from the anatomic dead-space leaves the airway (Part 1). The capnogram then rises sharply as alveolar gas mixes with dead-space gas (Part 2). The curve then levels and forms a plateau during most of exhalation (Part 3), which represents gas flow from alvoli and is; thus, called the alveolar plateau. The PCO_2 at the end of the alveolar plateau is called the end-tidal CO_2 or $PetCO_2$ (Aroonwan Pruettipan, B.E. 2545).

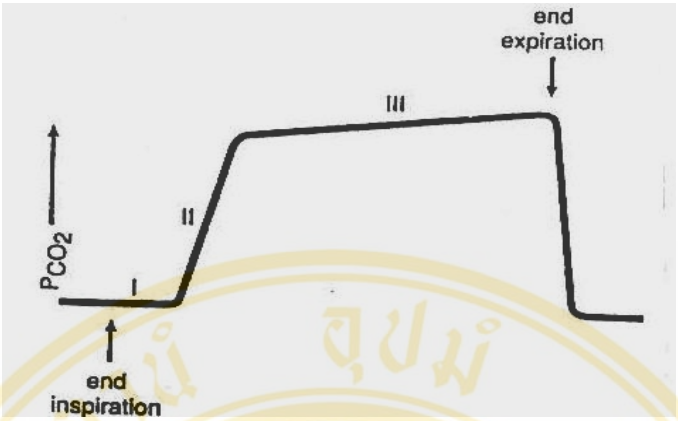

Figure 4: Normal capnogram (Aroonwan Pruettipan, B.E. 2545: 63)

An increase or decrease in end-tidal CO_2 can be the result of changes in CO_2 production and delivery to the lungs, changes in alveolar ventilation, or an equipment malfunction (Hess, 1993) (See Figure 5 and 6).

Increased CO_2 production and delivery to the lungs

- Fever
- Sepsis
- Bicarbonate administration
- Increased metabolic rate
- Seizures

Decreased alveolar ventilation

- Respiratory center depression
- Muscular paralysis
- Hypoventilation
- COPD

Equipment malfunction

- Rebreathing
- Exhausted CO_2 absorber
- Leak in ventilator circuit

Figure 5: Causes of increased end-tidal CO_2 (Hess, 1993: 383)

Decreased CO_2 production and delivery to the lungs
- Hypothermia
- Pulmonary hypoperfusion
- Cardiac arrest
- Pulmonery emboli
- Hemorrhage
- Hypotension

Increased alveolar ventilation
- Hyperventilation

Equipment malfunction
- Ventilator disconnect
- Esophageal intubation
- Complete airway obstruction
- Poor sampling
- Leak around endotracheal tube cuff

Figure 6: Causes of decreased end-tidal CO_2 (Hess, 1993: 383)

The benefits of capnometer (Aroonwan Pruettipan, B.E. 2545; Hess, 1993; Supitcha Sangchote, B.E. 2543) are as follows:

1. Capnometer in the patient with no lung's disease on ventilator, there need to an adaptation of level of PCO_2 such as head injured patients. The end-tidal CO_2 is often near the PCO_2 making it is not necessary to penetrate blood to check and analyze arterial blood gas frequently.

Besides, the other benefit is for the patients who uses ventilator such as weaning from the mechanical ventilator, and determine PEEP levels.

2. Capnometer in assessing the tissue perfusion and the cardiac output during cardiac arrest.

The end-tidal CO_2 is useful in the evaluation of the effectiveness of cardiopulmonary resuscitation. The end-tidal CO_2 correlates with cardiac output during CPR, as well as coronary perfusion pressure. The onset of cardiac arrest results in a drop of end-tidal CO_2, and who are successfully resuscitated have a higher end-tidal CO_2 during resucitation.

3. Capnometer in assessing the dead space ventilation is for the patient who has changes of dead space such as acute pulmonary embolism

4. Capnometer to identify the characteristics of respiration in the patient on ventilator by observing waveform and end-tidal CO_2, for example, detection of esophageal intubation. Evaluation of the capnographic wave form is useful in the detection of partial obstruction of the endotracheal tube. Monitoring of esophageal PCO_2 produces very low levels of end-tidal CO_2.

Heart rate

The function of the heart is considered in terms of the electrical and mechanical activities of the myocardial muscle. The cellular aspects provide a better understanding of the process. The electrical events precede and initiate the mechanical response and require certain properties inherent to cardiac muscle cells: automaticity, rhythmicity, excitability, and conductivity. The mechanical events of cardiac contraction are the result of four major determinants: preload, afterload, contractility, and distensibility. The heart rate, normally an extrinsic influence, helps to determine cardiac output (Bullock, 2000).

Autonomic influence on cardiac activity

The divisions of the autonomic nervous system (ANS) are consisted of: sympathetic and parasympathetic, exert external influences on myocardial contractility and rate. The cardiovascular center in the medulla receives input from other areas of the brain and relays messages throughout the body. In the heart, such messages include the autonomic nervous system adjustment of the heart rate and contractility to the body's demands. The autonomic nervous system has an enhancing or restraining effect on the inherent pacemaker system and can alter the automaticity of abnormal pacemaker systems.

Sympathetic nervous system (SNS)

The chemical mediators of the sympathetic nervous system are norepinephrine and epinephrine, collectively called catecholamines. The SNS releases these mediators during the stress reaction. These mediators stimulate α- and β-adrenergic receptors on target cells, which cause specific effects. Receptors from the sympathetic nervous system are present in the atrial wall, ventricles, and SA and AV nodes. When stimulated, these cardioaccelerator fiber release norepinephrine, which

stimulates β_1-receptors to increase the rate of depolarization and impulse transmission through the conduction tissue. Increased sympathetic tone increases cardiac rate and the contractility of myocardial muscle. The increased contractility is due to enhanced calcium entry through the myocardial muscle calcium channels. The sympathetic nervous system stimulation predominantly affects the SA node and causes a sinus tachycardia.

The effects of the sympathetic nervous system on the coronary arteries are somewhat more complex. Norepinephrine causes coronary artery vasoconstriction and increased oxygen extraction by the myocardial cell. Some individuals have a hyperactive response to norepinephrine and exhibit coronary artery vasospasm during stressful situations. Epinephrine, which is released from the adrenal glands, has a secondary dilating action on the coronary arteries. Its main actions are to produce tachycardia and increase contractility (a positive inotropic effect).

Prostaglandins, a group of chemically related substances, may be stimulated secondarily in the sympathetic stress response. They are synthesized by the myocardial cells and arteries and usually dilate the coronary arteries.

Normally, autoregulation of coronary blood flow appears to counteract the effects of neural stimulation. The changes of perfusion pressure are counteracted by changes in vascular resistance, so that blood flow remains rather constant. Autoregulation is probably due to the response of smooth muscle in the arterioles to stretch, so that when the local blood pressure rises the vessels constrict, and when it falls, they dilate. When SNS stimulation induces coronary vasoconstriction, coronary autoregulation usually overrides the mechanism and ischemia is prevented.

Parasympathetic nervous system (PSNS)

The parasympathetic nervous system is mediated through the chemical transmitter acetylcholine, which is released from vagal fibers. The major effects of vagal stimulation are a restraining or allowing influence on the SA node, atrial muscle, and AV node. Vagal stimulation slows the heart rate by restraining the rate of diastolic depolarization in the conduction tissue. It causes only a slight decrease in ventricular contractility.

A balance exists between sympathetic nervous system and parasympathetic nervous system stimulation of the heart, but the predominant system appears to be the parasympathetic nervous system. Evidence supporting this is that resting heart rate is usually lower than the inherent automatic SA pacing rate (Banasik, 1995; Bullock, 2000).

In addition to baroreceptors and chemoreceptors, other sensory fibers that detect pressure are located within the cardiac chambers. These sensory receptors respond to changes in the intrachamber pressure, which reflect the volume of blood in the chamber. Atrial or ventricular overdistention activates the sympathetic system and increases heart rate. Heart rate may also be influenced by higher central nervous system activities that do not involve reflex pathways.

In general, an increase in heart rate will result in an increase in cardiac output. However, at very high heart rates, cardiac output may actually fall. At high heart rates, the time for diastolic ventricular filling is significantly reduced, resulting in a low stroke volume. The benefit of increased heart rate is; therefore, undermined by impaired pumping efficiency (Banasik, 1995).

The endotracheal suctioning or normal saline instillation stimulates the vagal nerves leads to decrease heart rate immediately. Because of the oxygen is sucked out of the lungs together with the suctioning, and the normal saline instillation interrupted ventilation and oxygenation lead to hypoxia. The sympathetic nervous system is activated cause to tachycardia. (Saowaluk Jiratamkun, B.E. 2534).

In the ICU, the measurement of heart rate can be monitored by the bedside monitoring that shows electrical wave of heart (Decker, 1987 referred in Saowaluk Jiritamkun, B.E. 2534).

In this study, the researcher selected capnometer (BCI Capnochek Plus Model BCI-9004) as an instrument to measure the oxygen saturation, end-tidal CO_2, and heart rate parameters. This device continuously monitors and displays arterial blood oxygen saturation (SpO_2), heart rate, and plethysmogram. This capnometer determines oxygen saturation and heart rate by passing two low intensity wavelengths of light, one red and one infrared, through body tissue to a photodetector. Heart rate identification is accomplished by using plethysmographic techniques and oxygen saturation measurements which are determined by using spectrophotometric oximetry

principles. A few seconds after the patient is attached, the oxygen saturation measurement and heart rate measurement should be shown. Because the pediatric patients often cries, struggles and distress; endotracheal tube may esily kink. The researcher then selects the sidestream capnometer as this device is light; thus, uneasy to kink off the endotracheal tube.

Mean blood pressure

Systolic blood pressure reflects the maximum pressure in the aorta and major arteries during ventricular ejection of blood. Ventricular contraction forces blood into the distensible aorta where much of the stroke volume of the contraction is retained. The volume in the aorta and the large arteries exerts pressure on arterial walls; this pressure is the systolic arterial pressure. An increase or decrease in volume can produce a corresponding elevation or reduction in systolic blood pressure, assuming no other changes in the system.

During diastole, passive elastic recoil of the arterial walls ejects blood out of the aorta and into the peripheral arteries (Henshaw, 1995).

Diastolic pressure reflects the minimum pressure in these vessels during the pre ejection rest period, just prior to the subsequent ventricular contraction. Because the next contraction occurs before all the blood is ejected, the pressure never falls to zero. The minimum pressure diastolic pressure) is determined in part by the diameter of the arterioles, which is also the major determinant of systemic vascular resistance (SVR). Therefore, the diastolic blood pressure reading provides an estimate of SVR. Vasoconstriction, a narrowing of vessel diameter, increases SVR and diastolic blood pressure. Vasodilation reduces SVR and diastolic blood pressure (Henshaw, 1995).

Pulse pressure is determined by stroke volume (the amount of blood ejected with each heart beat), the speed at which the stroke volume is ejected, and arterial distensibility. Pulse pressure is the difference between the systolic and diastolic blood pressure (Henshaw, 1995).

Mean blood pressure is the average pressure in the system throughout the cardiac cycle. Mean blood pressure is not the arithmetic average of the diastolic and systolic pressures. Clinically, the formula used to estimate mean blood pressure is diastolic pressure plus one-third of the pulse pressure (Henshaw, 1995; Pipat Joedrangsee, B.E. 2545; Supanee Wasinamon B.E. 2537).

$$mBP = 1/3\ [systolic + (2 \times diastolic)]$$
$$= Diastolic + 1/3\ (pulse\ pressure)$$

Besides, the determinants of mean blood pressure include the cardiac output (CO) and the total peripheral resistance (TPR) as in the equation:

$$mBP = CO \times TPR$$

Control of blood pressure

Regulation of blood pressure is achieved primarily by regulating the determinants of blood pressure: cardiac output and total peripheral resistance. Because arterioles offer the greatest amount of resistance to blood flow, understanding the mechanisms that regulate arteriolar diameter is essential.

Arterioles are innervated by both the sympathetic and parasympathetic divisions of the autonomic nervous system. A tonic level of sympathetic discharge produces a baseline vasoconstriction. The parasympathetic system does not play a significant role in blood pressure.

Baroreceptors in the carotid sinus and in the arch of the aorta are sensitive to changes in mean arterial pressure and help regulate blood pressure by influencing both cardiac output and peripheral resistance. The baroreceptors send impulses to a cardiovascular control center in the medulla of the brain stem. The baroreceptors alter their rate of discharge in response to changes in mean arterial pressure. An increase in pressure causes an increase in impulse receptor firing rate causing a decrease in sympathetic outflow and an increase in parasympathetic outflow to the heart and vascular tree, causing slowing of the heart rate and vasodilation. These changes result in a decrease in arterial blood pressure. A decrease in arterial blood pressure causes similar changes in the opposite direction. In the face of persistent elevations in arterial blood pressure, the baroreceptors adapt and rest at the new, higher level, while still opposing immediate changes in blood pressure.

Circulating hormones can also affect blood pressure. For example, epinephrine and norepinephrine are released from the adrenal medulla in response to low mean arterial pressure. The effects of these hormones are the same as direct

sympathetic nervous system stimulation: vasoconstriction, increased heart rate, and increased myocardial contractility. These mechanisms work to raise blood pressure.

The rennin-angiotensin-aldosterone system is also involved in blood pressure control. Any reduction in renal perfusion stimulates release of the enzyme rennin, which acts on angiotensinogen to produce angiotensin I, an inactive substance. Angiotensin-converting enzyme (ACE), found primarily in the vasculature of the lungs, converts angiotensin I to angiotensin II, one of the most potent vasoconstrictors known. In addition, angiotensin II promotes sodium and water retention by the kidney, both directly and indirectly, by stimulating the release of aldosterone from the adrenal medullae. The resulting expansion of volume along with the vasoconstriction increases blood pressure.

Arterial natriuretic peptide (ANP) is released from artial myocytes in response to stimulation of stretch receptors by excess volume. Release of ANP results in an increased glomerular filtration rate, excretion sodium and water, and vasodilation. In addition, ANP inhibits the secretion of rennin, aldosterone, and vasopressin.

Antidiuretic hormone (ADH), released from the posterior pituitary gland, has a direct vasoconstriction effect. In addition, it helps regulate volume by preventing excretion of water by the kidneys.

Other substances, such as histamines, kinins, prostacyclin, endothelin-1, and various metabolites, have the ability to produce local vasoconstriction. The precise mechanisms of action and the clinical significance of these agents are being investigated (Henshaw, 1995) (Figure 7).

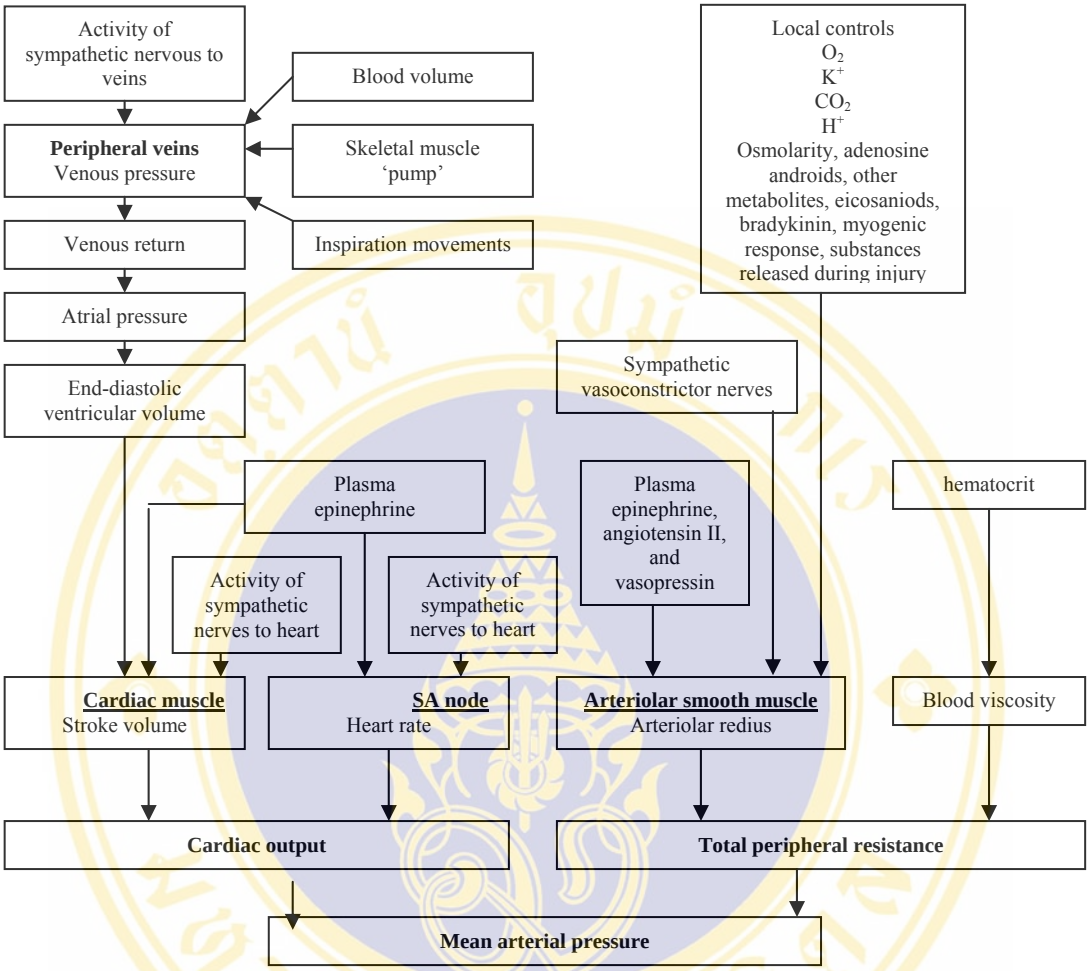

Figure 5: Control of blood pressure (Henshaw, 1995: 331-332)

Measurement of blood pressure

The measurement of blood pressure has the direct and indirect methods as follows (Henshaw, 1995).

Indirect methods

Blood pressure is most commonly measured indirectly using a sphygmomanometer. A hollow bladder within a cuff, the sphygmomanometer is wrapped around a limb, usually the upper arm. As the brachial or radial artery is palpated the cuff is inflated. Cuff inflation is continued for another 30 mmHg, the cuff is then gradually deflated until the pulse reappears. The return of blood flow through the artery is signaled by the Korotkoff sounds.

Another indirect method combines the Doppler technique with the use of a sphygmomanometer. Ultrasonic impulses are transmitted from a transducer placed over an artery to the blood cells flowing through the artery. The impulses are reflected back to the transducer, which translates the impulses into audible sound. As the blood pressure cuff is deflated, the transducer is placed over an artery. The onset of sounds from the Doppler instrument is equivalent to phase I of the Korotkoff sounds and signals systolic blood pressure.

Direct methods

Direct measurement of arterial blood pressure is also possible. A catheter is introduced into a peripheral artery, such as the radial, brachial, or femoral artery, and is connected to a pressure transducer. Pressure from the artery is transmitted to and exerts pressure on the diaphragm at the air-fluid interface in a dome attached to the transducer. The transducer converts the pressure on the diaphragm to electrical signals. The electrical signals are amplified, filtered, and displayed on a monitor in waveforms. Digital readings often are displayed.

Factors that affect blood pressure measurement

A number of factors can affect blood pressure measurements. These factors are of two types: technical factors and factors involving the subjective influences of the observer (Henshaw, 1995).

Technical aspects include such things as body positioning and size of the cuff used. If the arm used for blood pressure measurement is positioned above the level of the heart, the blood pressure may be artificially lowered. If the arm is below the heart, blood pressure may be artificially raised. The American Heart Association (1993) recommends a cuff with a bladder width is 40 percent of the circumference of the midpoint of the limb. Cuffs with bladders that are too narrow will result in falsely elevated blood pressure readings. The bladders that are too wide result in erroneously low readings.

From the mean blood pressure (mBP) is the value between systolic and diastolic pressure that have relationship with the amount of blood that is pumped out of the left ventricle within 1 minute (cardiac output: CO) and the total peripheral resistance: TPR. Consequently, this research selected to use mean blood pressure in

studying normal saline instillation before suctioning and use the measurement of blood indirectly that connected to the monitor to measure noninvasive blood pressure (NIBP).

In this study, the researcher has selected blood pressure monitor, Phillip (Model No. M1205A). Because of this device is noninvasive monitor and automatically detected, it can monitor and record parameter at one minute interval or setting intervals.

Normal of oxygen saturation (Adtapol Aeam-udomkal, B.E. 2549)

Oxygen saturation 95 to 98%

Normal of end-tidal CO_2 (Adtapol Aeam-udomkal, B.E. 2549)

end-tidal CO_2 (mmHg)	status	alveolar ventilation level
< 35	hypocapnia	hyperventilation
35-45	eucapnia	normal ventilation
> 45	hypercapnia	hypoventilation

Normal of heart rates in children (Hazinski, 1992)

Age	Awake heart rate (per min)	Sleeping heart rate (per min)
Neonate	100-180	80-160
Infant	100-160	75-160
Toddler	80-110	60-90
Preschool	70-110	60-90
School-age child	65-110	60-90
Adolescent	60-90	50-90

Normal blood pressure in children (Hazinski, 1992)

Age	Sytolic pressure (mmHg)	Diastolic pressure (mmHg)
Birth (12 hr, <1,000 g)	39-59	16-36
Birth (12 hr, 3 kg)	50-70	25-45
Neonate	60-90	20-60
Infant	87-105	53-66
Toddler	95-105	53-66
School age	97-112	57-71
Adolescent	112-128	66-80

From the abovementioned literature reviews, the normal saline instillation via endotracheal tube before suctioning from the researches have no clear benefit, but the danger of suctioning is found. The danger will increase after normal saline instillation leads to hypoxemia, the level of carbon dioxide tension increases, the heart rate increases, the blood pressure increases, and the patient's discomfort increases. In addition, the study in pediatric patient is limited; thus, the researcher is interested in studying a comparison of the effects of endotracheal suctioning with and without normal saline instillation on oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure in pediatric patients with mechanical ventilation.

CHAPTER III
RESEARCH METHODOLOGY

Research Design

This study was a crossover-experimental design, aimed to compare the effects of endotracheal suctioning between with and without normal saline instillation on physiologic changes in pediatric patients with mechanical ventilation.

Population and Sampling

The population of this study was pediatric patients who were admitted in Pediatric Intensive Care Unit (PICU) at Faculty of Medicine, Ramathibodi Hospital. The sample was selected by purposive sampling under the inclusion criteria as follows:

1. Male or female newborn whose ages are up to 15 years old.

2. Being intubated with the endotracheal tube and supported by mechanical ventilation with a heated humidifier. The temperature control of the non-heated wire humidifier was set at level 2 to 3 whilst the heated wire humidifier was set at 39°C and at level -2 of the control chamber since these settings could provide the optimal humidity for the patient.

3. Having a hemodynamic stable longer than 6 hours before endotracheal suctioning, i.e.,

Heart rate was in a range of:

- 100-180 beats/min in newborn
- 100-160 beats/min in patient aged 1 week-3 months
- 80- 150 beats/min in patient aged 3 months-2 years
- 70-110 beats/min in patient aged 2-10 years old
- 60-90 beats/min in patient aged 10-15 years old

Mean arterial blood pressure was between:

- 50-60 mmHg in newborn
- 50-65 mmHg in patient aged 1 week-3 months
- 50-80 mmHg in patient aged 2-10 years old
- 70-95 mmHg in patient aged 10-15 years old

Respiratory rate was in a range of:

- 30-60 beats/min in newborn
- 24-40 beats/min in patient aged 1 week-3 months
- 22-34 beats/min in patient aged 3 months-2 years
- 18-30 beats/min in patient aged 2-10 years old
- 12-20 beats/minute in patient aged 10-15 years old

Body temperature was between 36-37°C (Axillary)

4. Had a hemoglobin level in a range of 10-16 g/dl.
5. Their parents agreed to participate in this study.

The exclusion criterion as follows:

During the data collection, if there are the irregular heart beats during suctioning such as PVC more than twice per minute or heart beats lower than 60 beats per minute, the researcher would stop the suction and notify the physician immediately and, thus; unable to continue the research on the subject.

Sample Size

The sample size used in this study was estimated by calculation based on the power analysis. Since the main statistics was the paired t-test and wilcoxon signed ranks test, and used alpha = .05, power of the test = .8, and effect size as a medium effect size = .5, the sample size of the study was 63 subjects in each study group (Polit & Beck, 2003: 496-498). The researcher considered using 65 subjects, in which all subjects served as their own control in this study.

The sampling used in this study was based on the above criteria where they were assigned with 2 alternate methods under cross-over design. One of them was method A (the endotracheal suctioning without normal saline instillation) and the other was method B (the endotracheal suctioning with normal saline instillation). The sequence of two different conditions was randomly assigned by drawing lots from

1-65 without replacement. When the subjects had received an odd number (1, 3, 5, …, 65), the method was A then B. On the other hand, an even number (2, 4, 6, …, 64) had the sequence of method B then A.

Settings

The study was conducted in Pediatric Intensive Care Unit (PICU) at the Faculty of Medicine, Ramathibodi Hospital. Within the PICU, the nurse station was located in the middle, separated from the patient unit. Each room was designed as a closed environment for the infectious patients wherein sliding glass doors were installed to separate each of the six rooms. Moreover, there were four positive-pressure rooms for the immuno-compromised patients.

Under normal settings, the day to day process of this unit was taken care by a group of staff, which comprised of registered nurses, pediatricians and residents. Sometimes, there were medical and nursing students practicing in the PICU. Most of the nursing and treatment activities were taken in the daytime whilst many doctors had bedside conferences on regular weekdays. Common activities during each shift were taking vital signs, suctioning, turning position, feeding, and medical administration, whereas other activities, i.e., resuscitation, treatment and admission a new patient were occasionally required. The pediatric patients were routinely weighed and linen changed at 8.00 a.m. on the day shift. Visiting hours were allowed from 8.00 a.m. to 8.00 p.m. providing two visitors for each visit. Presently, visiting hours and the number of visitors were more flexible.

The pediatric patients admitted to PICU were ranged from newborn to fifteen years with critical conditions. Respiratory failure was the most common problem of the critical illness which resulted in sudden hospitalization. The pediatric patients with respiratory failure usually required the endotracheal intubations and supported by the mechanical ventilation with heated humidifier. The temperature control of the non-heated wire humidifier was set at level 2 to 3, and the heated wire humidifier was set at 39°C and at level -2 of the control chamber, since these setting could provide the optimal humidity for the patient. Thus, the suctioning was considered an important component of the mechanically ventilated patient's care. This procedure was performed every 3 hours as the pediatric patients required suctioning before feeding

every 3 hours to prevent aspiration. This procedure depended on the child's age, muscular and neurological status, activities level, ability to cough, and the quantity and viscosity of secretions.

Instrumentation

The instruments of this study were composed of two parts as follows:

1. Experimental Instruments included:

1.1 The mechanical ventilator with a heated humidifier.

1.2 The sterile normal saline for instillation via endotracheal tube.

1.3 The suctioning equipment:

1.3.1 Vacuum pressure on wall suction by Omeda with negative pressure about 0 to 200 mmHg.

1.3.2 The size of the suction catheter was between 6-12 French (Fr) or should not be bigger than a half of the internal diameter of the endotracheal tube.

Size of endotracheal tube (mm)	Size of suction catheter (Fr)
2.5 - 3.5	6
4.0 - 5.0	8
5.5 - 6.0	10
6.5 - 7.5	12

1.4 Sterile right glove for suctioning.

1.5 A 500 ml -1,000 ml resuscitating bag used by these following criteria:

- Infant: use a 500 ml resuscitating bag
- Small child (2-5 years): use a 500 ml resuscitating bag
- School-age child (6-12 years): use a 500 ml-1,000 ml resuscitating bag
- Adolescent to adult: use a 1,000 ml resuscitating bag

1.6 100% Oxygen supply valve.

1.7 Positive end expiratory (PEEP) valve device.

1.8 Pressure gauge.

2. Data Collection Instruments included:

2.1 Capnometer (BCI Capnocheck Plus Model BCI – 9004) was used to measure an oxygen saturation, end-tidal CO_2 and heart rate parameters. This Capnometer was calibrated by E for L International Company before using in this study to ensure the accuracy of the instrument.

2.2 Monitor of Phillip (Model No. M 1205A), being noninvasive, automatic device and could be present for measurements at specific time intervals. Mean blood pressure parameter was recoded on a bedside monitor. This monitor was calibrated by Xovic Company to ensure the accuracy of the instrument.

2.3 Timer (Model sport timer) was used to measure the taken time. This timer was calibrated to the standard time by Navy.

2.4 Stethoscope was used to assess the lung sound.

2.5 Data record forms included:

2.5.1 Demographic data record form, which included data of gender, age, body weight, diagnosis, treatment, total fluid, x-ray, arterial blood gas (recently), duration of endotracheal tube usage, frequency of suctioning in 24 hours, duration of each suctioning time, type of mechanical ventilator and the sequence of experimental method (Appendix B, Part I).

2.5.2 Physiological data record form (during suctioning), which included data of oxygen saturation, end-tidal CO_2 and heart rate. These parameters were recorded as baseline (before data collection), immediately connecting the mechanical ventilator and after suctioning at minute 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, and 15. The mean blood pressure parameter was recorded as baseline, immediately connecting the mechanical ventilator and after suctioning at minute 5, 10, and 15 (Appendix B, Part II).

Protection of Human Subjects

The research proposal was approved by the Committee on Human Rights related to Research Involving Human Subjects of the Faculty of Medicine, Ramathibodi Hospital, before the data were collected. The parents of eligible subjects

were approached and asked to participate in the study. The researcher then introduced herself to the parents' subjects and asked for their participation by explaining the objectives of the study, the data collection process, and expected research outcomes. The parents' subjects were informed that they had the rights to refuse or agree to participate in the study (Appendix A). In addition, the parents' subjects were assured that their participation in this study or refusal would not affect their relationship with the personnel in the hospital and the treatment they received. They were also assured that the data collection from them would be kept confidential.

Procedure

The eligible subjects were randomly assigned by drawing lots without replacement. Substitution of odd number (1, 3, 5,…, 65) was method of A then B and even number (2, 4, 6, …, 64) would be method of B then A respectively.

1. The researcher used a stethoscope to assess the lung sound every ½ hour. The first researcher assistant arranged and recorded baseline parameters of subjects during stable condition, such as oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure.

2. The second research assistant (who performed the suction) arranged the suctioning equipment to set appropriate pressure of vacuum for each subjects such as: 80 mmHg for infant and 100 mmHg for children age older than 1 year.

Subsequently, the second research assistant wore the sterile right glove and held the suction catheter (selected an appropriate suction catheter as following criteria) connected to the finger tip to the suction catheter and another end to the wall suction.

Size of endotracheal tube (mm)	Size of suction catheter (Fr)
2.5 - 3.5	6
4.0 - 5.0	8
5.5 - 6.0	10
6.5 - 7.5	12

3. The researcher then gave manual positive pressure ventilation with the resuscitating bag, arranged the appropriate size of the resuscitating bag for each subject such as size 500 ml for infant, small child and school-age child and 1,000 ml

for adolescence. She connected it with oxygen supply valve for 100% oxygen with PEEP valve device to adjust the positive end-expiratory pressure that the patient received from the mechanical ventilator. The researcher disconnected the mechanical ventilator from the subject's endotracheal tube and connected it with the resuscitating bag and then, pressed the resuscitating bag evenly to the peak of inspiratory pressure of the mechanical ventilator by using the pressure gauge to monitor the pressure.

4. The experiment was performed as follows:

Method A or endotracheal suctioning without normal saline instillation, the researcher gave 5 breaths of the manual positive pressure ventilation by the resuscitating bag.

Method B or endotracheal suctioning with normal saline instillation, the researcher instilled normal saline via the endotracheal tube: 0.5 ml for children age younger than 1 year, 1ml for children of 1 – 8 years old and 2 ml for children age more than 8 years; and gave 5 breaths of the manual positive pressure ventilation by the resuscitating bag.

5. The second research assistant who performed the suction measured the depth of the suction catheter to be equal with the depth of endotracheal tube plus the length of partial endotracheal tube from the subject's mouth to slip joint along with the length of suction catheter beyond the endotracheal tube 1 cm. Then, the second research assistant used the tape to mark the suction catheter length at the head of the subject's bed to measure the depth of the suction catheter before suctioning. Each subject was suctioned no longer than 10 seconds (count 1 to 10).

6. The researcher gave 5 breaths of the manual positive pressure ventilation with the resuscitating bag.

7. Suctioned and gave 5 breaths of the manual positive pressure ventilation with the resuscitating bag twice and assessed the lung sound once more to evaluate the efficiency of suctioning.

8. The researcher connected the mechanical ventilator to the subject's endotracheal tube. The first research assistant recorded the oxygen saturation, end-tidal CO_2, and heart rate parameters. These parameters were recorded immediately after connecting the mechanical ventilation and suctioning at minute 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, and 15. The mean blood pressure parameter was recorded as baseline,

immediately after connecting the mechanical ventilator and suctioning at minute 5, 10, and 15.

Data Collection

The data collection was conducted by the researcher in the following steps:

1. The data collection procedure commenced once the approval was obtained from the Dean of the Faculty of Graduate Studies, Mahidol University; the head of Department of the Faculty of Medicine; the Director of Nursing Department of the Faculty of Medicine; and the Head of Pediatric Nursing Department of the Faculty of Medicine, Ramathibodi Hospital to allow data collection at the Pediatric Intensive Care Unit (PICU). Regarding the human subject protection procedures, all parents of eligible subjects were approached by the researcher. The informed consents were, thus; obtained from the parents' subjects to ensure that they were willing to participate in the study.

2. The head of the PICU was contacted whilst the involved nurses in this study were asked for their cooperation.

3. Two registered nurses from the PICU were selected as the research assistant in this study where they were informed of the details and practice procedure on the suction and other related matters. The first researcher assistant acted as a data collector whilst the second researcher assistant performed the suction. The researcher was responsible for providing the positive pressure ventilation manual with the resuscitating bag and instilled of normal saline via endotracheal tube.

4. The sequence of experimental method of eligible subjects was randomly assigned by drawing lots (number 1 to 65) without replacement from the researcher.

- Odd number (1, 3, 5,.., 65): an experimental method was A B respectively.
- Even number (2, 4, 6,.., 64): an experimental method was B A respectively.

Note: A was endotracheal suctioning without normal saline instillation.

B was endotracheal suctioning with normal saline instillation.

The time between suctioning of each method would be separated by 3 hours prior to the beginning of the other method.

5. The researcher used stethoscope to assess the lung sound every ½ hour. The first researcher assistant arranged the data collection instruments such as prepared

a cuff to measure blood pressure, applied the sensor probe of pulse oximeter to the subject's finger, connected the capnometer (BCI Capnocheck Plus) with endotracheal tube, and recorded of oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure parameters in data record forms with the baseline parameters of the subjects having stable condition.

6. The researcher and the second researcher assistant performed the suction following the suctioning guideline (See detailed procedure in Figure 8, Appendix B, and Figure17) on each subject. Additionally, the first researcher assistant recorded oxygen saturation, end-tidal CO_2, and heart rate parameters. These parameters were recorded immediately after connecting the mechanical ventilation and suctioning at minute 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, and 15. The mean blood pressure parameter was recorded as baseline, immediately after connecting the mechanical ventilator and suctioning at minute 5, 10, and 15.

7. After connecting the mechanical ventilation, the researcher should separate each method by 3 hours whereas each subject would be given the methods according to the drawing accordingly. The researcher selected to separate each method by 3 hours, because of the pediatric patients required suctioning before feeding every 3 hours to prevent aspiration.

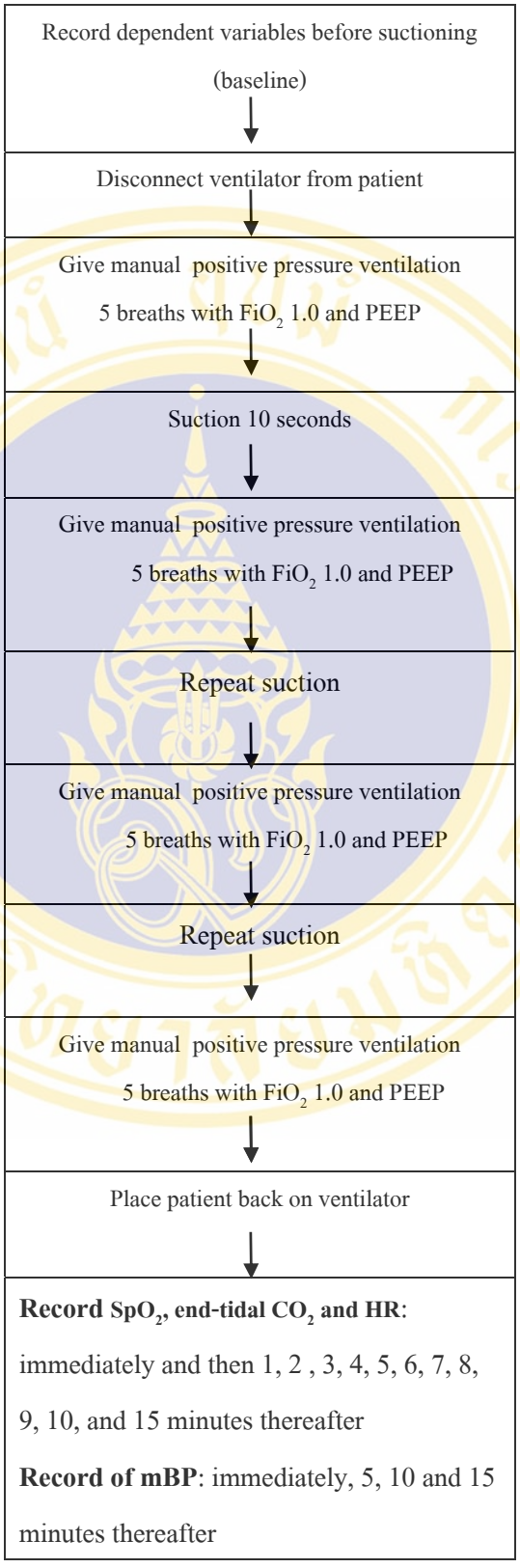

Figure 8: Step of suctioning and recording

Data analysis

The data was calculated by computer using the Statistical Package for Social Science for Window Version 12 (SPSS/FW) as follows:

1. Frequency and percentage were applied to describe the demographics data of the samples such as gender, age, diagnosis, and total fluid intake, type of mechanical ventilator, duration of using endotracheal tube, frequency of suctioning per day, and period of suctioning time in each episode.

2. Mean and standard deviation were conducted to describe the baseline and after suctioning parameters of oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure between the endotracheal suctioning with and without normal saline instillation.

3. Mean and standard deviation were applied to analyze duration of oxygen saturation reduced to minimum, duration of end-tidal CO_2, heart rate, and mean blood pressure increased to maximum and all parameters returned to baseline.

4. Paired t-test and wilcoxon signed rank test were conducted to compare the mean change of oxygen saturation, end-tidal CO_2, heart rate, and mean blood pressure between the endotracheal suctioning with and without normal saline instillation.

CHAPTER IV
RESULTS

This research was a crossover-experimental design to study the effects of endotracheal suctioning with and without normal saline instillation on physiologic changes in the pediatric patients with mechanical ventilation. In this chapter the result of the data analysis were presented in two parts; the descriptive data of the study sample and major study variables.

Part I. Descriptive Data of the Study Sample

Demographic Characteristics

The endotracheal suctioning was performed with 58 subjects, and yielded 65 observed suctioning. According to the crossover-experimental design in this study, each subject was required to complete both methods: the endotracheal suctioning with and without normal saline instillation which were randomly assigned. The sample consisted of 36 males (62.1%) and 22 females (37.9%) with the ages ranged from 5 days to 14 years and 6 months. The common ages ranged from 1 to 12 months (n=21, 36.2%). The majority of primary diagnoses were neurological problem (n=20, 34.5%), such as seizure disorder, AVM rupture, and hydrocephalus (Table 1). Most subjects received with 80% maintenance of total fluid intake (n=37, 56.9%). Most of the mechanical ventilators were used humidifier and a heated wired humidifier (n=45, 69.2%). The majority of subjects were intubated with the oral endotracheal tube size 4 to 5 mm (n=38, 60.0%), and suctioned catheter No. 8 Fr (n=38, 60.0%). Duration of using the endotracheal tube ranged from 1 to 15 days, and the most of duration of using the endotracheal tube ranged from 1 to 7 days (n=57, 87.7%). The samples received endotracheal suctioning ranged from 5 to 15 times per day, and most samples received endotracheal suctioning ranged from 6 to 10 times per day (n=54, 83.1%). The period of suctioning time in each episode ranged from 4 to 7 minutes, and the

most period of suctioning time in each episode was from 1 to 5 minutes (n=59, 90.8%) (Table 2).

Table 1: Frequency and Percentage of Sample Characteristics (n=58)

Characteristics	Frequency	Percentage
Gender		
Male	36	62.1
Female	22	37.9
Age		
< 1 month	2	3.4
1-12 months	21	36.2
1-3 years	13	22.4
3-6 years	8	13.8
6-12 years	12	20.4
12-15 years	2	3.4
Diagnosis		
Neurological problem	20	34.5
Respiratory problem	10	17.2
Others (liver, kidney & accident)	9	15.5
Cardiovascular with Respiratory problem	8	13.8
Hematology problem	5	8.6
Cardiovascular problem	4	6.9
Gastro-intestinal problem	2	3.4

Table 2: Frequency and Percentage of Sample Baseline Characteristics (n=65)

Characteristics	Frequency	Percentage
Total fluid intake		
60% maintenance	1	1.5
70% maintenance	3	4.6
80% maintenance	37	56.9
90% maintenance	2	3.1
100% maintenance	22	33.8
Type of the mechanical ventilator		
Non-heated wired humidifier	20	30.8
Bear cub	15	23.1
Bear 750	6	9.2
Bear 1000	4	6.2
Heated wired humidifier	45	69.2
Bennette7200	1	1.5
Evita4	5	7.7
Servo300	5	7.7
Servo900	9	13.8
Servo i	20	30.8
Size of the endotracheal tube (mm)		
2.5-3.5	8	12.3
4.0-5.0	39	60.0
5.5-6.0	12	18.5
6.5-7.5	6	9.2

Table 2: Frequency and Percentage of Sample Baseline Characteristics (n=65) (continued)

Characteristics	Frequency	Percentage
Size of the suction catheter (Fr)		
6	8	12.3
8	39	60.0
10	12	18.5
12	6	9.2
Duration of using the endotracheal tube (days)		
1-7	57	87.7
8-14	4	6.2
15-21	3	4.6
22-28	1	1.5
(mean = 3.68, median = 2.00, mode = 1.00, SD = 4.43)		
Frequency of the suctioning per day (times/day)		
1-5	1	1.5
6-10	54	83.1
11-15	10	15.4
(mean = 7.72, median = 6.00, mode = 6.00, SD = 2.64)		
Period of suctioning time in each episode (minute)		
1-5	59	90.8
5.01-10	6	9.2
(mean = 5.12, median = 5.00, mode = 5.00, SD = 0.44)		

Part II. Hypothesis Testing

To test the effects of endotracheal suctioning with and without normal saline instillation on physiologic changes in pediatric patients with mechanical ventilation. The dependent variables in hypotheses were the mean change of physiologic changes (i.e., oxygen saturation, end-tidal CO_2, heart rate and mean blood pressure).

The Kolmogorov-Smirnov test was used to test the normality of the dependent variables. In general, the non-significant result ($p>.05$) indicated normal distribution. The mean change of the heart rate and mean blood pressure met the normality assumption whilst the mean change of oxygen saturation and end-tidal CO_2 did not meet the assumption of normality in this study. Thus, the paired t-test and wilcoxon signed ranks test were used in this study.

The results of hypothesis testing were presented as follows:

Hypothesis 1: The mean of oxygen saturation of patients who had the endotracheal suctioning with normal saline instillation decrease more than those of the patients who had the endotracheal suctioning without normal saline instillation.

Oxygen saturation referred to the amount of oxygen bound to hemoglobin compared with hemoglobin's maximal ability for oxygen binding as measured by pulse oximeter, BCI Capnocheck (BCI-9004), which was a noninvasive device.

The mean of oxygen saturation displayed to reduce after endotracheal suctioning in both methods. The mean of oxygen saturation of the endotracheal suctioning with normal saline instillation decreased more than the endotracheal suctioning without normal saline instillation, and the recovery time to return to the baseline was more than the endotracheal suctioning without normal saline instillation (at 5 minutes and 4 minutes, respectively) (See Table 3 and Figure 9).

Table 3: Mean of Oxygen Saturation Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

Oxygen Saturation	Method A(without NSS)		Method B(with NSS)	
	M (%)	SD	M (%)	SD
Before suctioning (baseline)	98.57	2.17	98.62	2.07
Immediately after	94.83	2.92	93.89	3.06
1 min after	95.92	3.00	95.05	3.14
2 mins after	97.09	2.92	95.83	3.15
3 mins after	97.97	2.84	96.85	3.21
4 mins after	98.57*	2.45	97.69	3.10
5 mins after	98.89	2.22	98.38*	2.83
6 mins after	98.97	2.02	98.65	2.53
7 mins after	98.95	2.03	98.94	2.16
8 mins after	99.09	1.79	99.03	1.86
9 mins after	99.14	1.84	98.98	1.88
10 mins after	99.08	1.74	99.08	1.74
15 mins after	99.15	1.91	99.02	2.00

Note: * = The recovery time to return to baseline

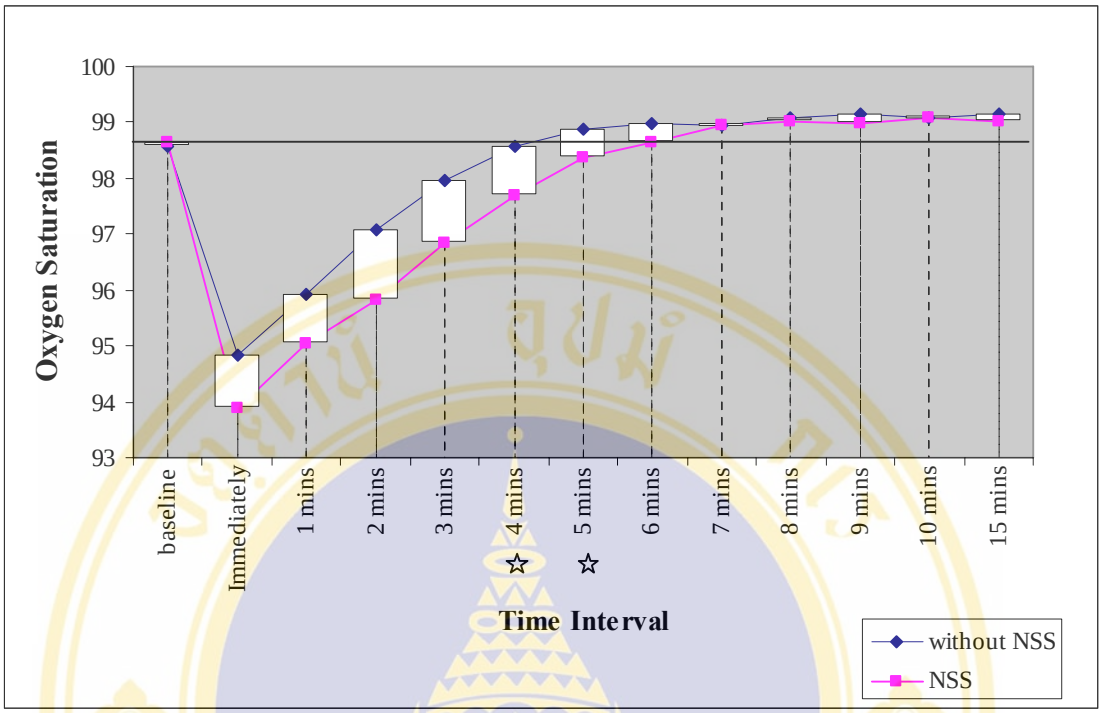

Figure 9: Mean of oxygen saturation over time

Wilcoxon signed ranks test was used to detect the difference mean change of oxygen saturation of the subjects after the endotracheal suctioning with and without normal saline instillation. The analysis showed that there were statistically significant differences in the mean change of oxygen saturation over time among the subjects in both methods ($p<.05$), except for the mean change of oxygen saturation at 7, 8, and 10 minutes after suctioning that there were no statistically significant differences ($p>.05$). The mean change of oxygen saturation in the endotracheal suctioning without normal saline instillation ranged from -3.74% to 0.58%, whereas it ranged from -4.72% to 0.46% in the endotracheal suctioning with normal saline instillation (See Table 4 and Figure 10).

Table 4: A Comparison by Wilcoxon Signed Ranks Test of the Mean Change of Oxygen Saturation Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

Oxygen Saturation	Method A (without NSS)		Method B (with NSS)		z-value	p-value
	Mean change (%)	SD	Mean change (%)	SD		
Immediately after	-3.74	1.40	-4.72	1.63	-6.87	0.00
1 min after	-2.65	1.34	-3.57	1.81	-5.94	0.00
2 mins after	-1.48	1.24	-2.78	1.54	-6.74	0.00
3 mins after	-0.60	1.07	-1.77	1.59	-6.11	0.00
4 mins after	0.00	0.75	-0.92	1.52	-5.25	0.00
5 mins after	0.32	0.66	-0.23	1.11	-4.72	0.00
6 mins after	0.40	0.63	0.03	1.09	-3.16	0.002
7 mins after	0.38	0.58	0.32	0.73	-0.21	0.834^{ns}
8 mins after	0.52	0.83	0.42	0.63	-1.33	0.183^{ns}
9 mins after	0.51	0.73	0.37	0.67	-2.29	0.022
10 mins after	0.56	0.75	0.46	0.73	-0.60	0.552^{ns}
15 mins after	0.58	0.79	0.40	0.81	-2.23	0.026

Note: Mean change = (Mean of oxygen saturation each point over time – Mean of oxygen saturation at baseline); ns = non significant

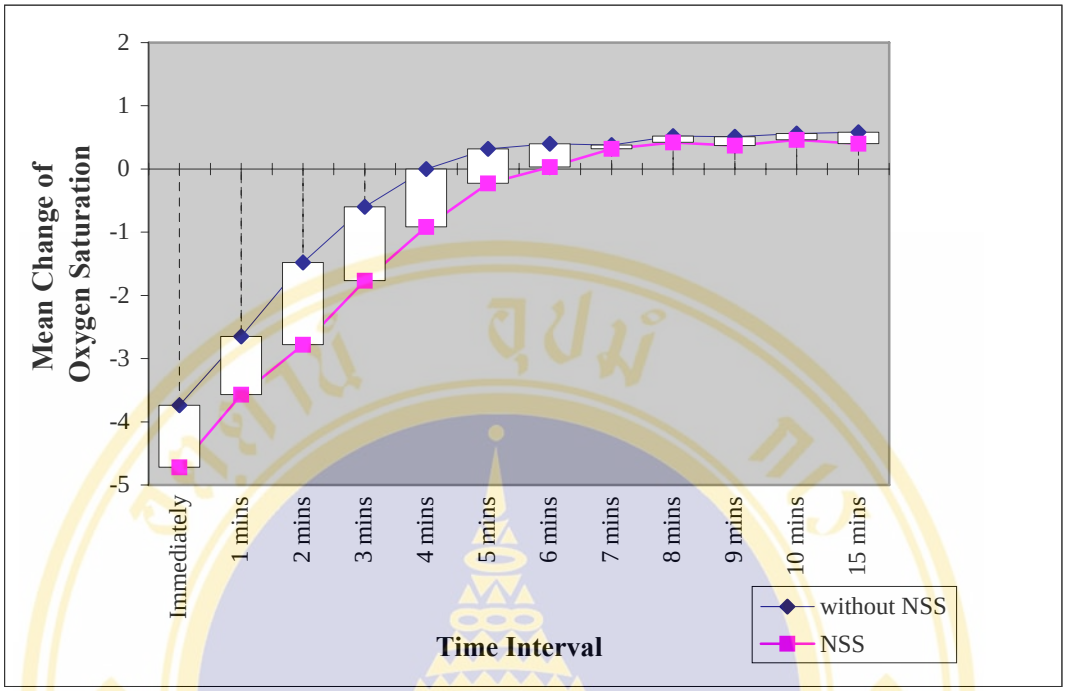

Figure 10: Mean change of oxygen saturation over time

Hypothesis 2: The mean of end-tidal CO_2 of patients who had the endotracheal suctioning with normal saline instillation increase more than those of the patients who had the endotracheal suctioning without normal saline instillation.

End-tidal CO_2 referred to the partial pressure or maximum concentration of carbon dioxide at the end of an exhaled breath as measured by the sidestream capnometer, BCI Capnocheck Plus (BCI-9004), which was a noninvasive device.

The means of end-tidal CO_2 showed to the increasing trend after endotracheal suctioning both the two methods, but the endotracheal suctioning with normal saline instillation increased more than the endotracheal suctioning without normal saline instillation. The mean of end-tidal CO_2 returned to baseline at 9 minutes after endotracheal suctioning without normal saline instillation (See Table 5 and Figure 11).

Table 5: Mean of End-tidal CO_2 Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

End-tidal CO_2	Method A(without NSS)		Method B(with NSS)	
	M (mmHg)	SD	M (mmHg)	SD
Before suctioning (baseline)	39.23	8.50	39.25	8.42
Immediately after	42.09	9.00	43.06	9.08
1 mins after	41.38	9.08	42.28	9.12
2 mins after	40.69	9.01	41.57	9.15
3 mins after	40.18	8.85	41.02	9.10
4 mins after	39.65	9.00	40.38	9.07
5 mins after	39.52	8.90	39.98	8.76
6 mins after	39.38	8.76	39.71	8.94
7 mins after	39.31	8.61	39.65	8.95
8 mins after	39.37	8.56	39.63	8.67
9 mins after	39.22*	8.70	39.66	8.60
10 mins after	39.28	8.52	39.82	8.51
15 mins after	40.18	8.34	40.65	8.51

Note: * = The recovery time to return to baseline

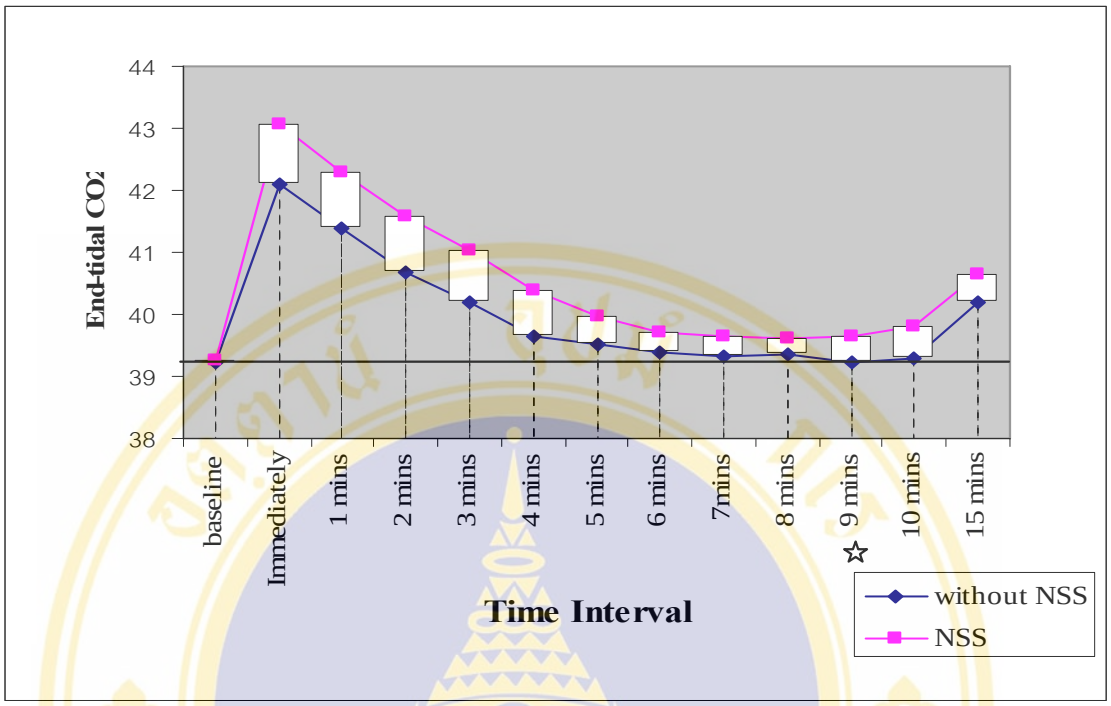

Figure 11: Mean of end-tidal CO_2 over time

As shown in Table 6, wilcoxon signed ranks test was used to detect the differences in the mean change of end-tidal CO_2 of the endotracheal suctioning with and without normal saline instillation. The result showed that there were statistically significant differences in the mean change of end-tidal CO_2 over time between the two methods ($p<.05$), except for the mean change of end-tidal CO_2 at 8 minutes after suctioning that there were no statistically significant differences ($p>.05$). The mean change of end-tidal CO_2 in the endotracheal suctioning without normal saline instillation ranged from-0.02 to 2.86 mmHg, whereas it ranged from 0.38 to 3.82 mmHg in the endotracheal suctioning with normal saline instillation (See Figure 12).

Table 6: A Comparison by Wilcoxon Signed Ranks Test of the Mean Change of end-tidal CO_2 Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

end-tidal CO_2	Method A (without NSS) Mean change (mmHg)	SD	Method B (with NSS) Mean change (mmHg)	SD	z-value	p-value
Immediately after	2.86	1.78	3.82	2.08	-5.727	0.00
1 min after	2.15	1.77	3.03	2.08	-4.578	0.00
2 mins after	1.46	1.63	2.32	2.11	-4.369	0.00
3 mins after	0.95	1.57	1.77	1.93	-4.004	0.00
4 mins after	0.42	1.24	1.14	1.73	-4.194	0.00
5 mins after	0.29	1.23	0.74	1.40	-2.726	0.006
6 mins after	0.15	1.12	0.46	1.26	-1.942	0.052
7 mins after	0.08	1.07	0.40	1.21	-2.128	0.033
8 mins after	0.14	1.21	0.38	1.13	-1.833	0.067^{ns}
9 mins after	-0.02	1.48	0.42	1.17	-2.168	0.030
10 mins after	0.05	1.12	0.57	1.16	-3.398	0.001
15 mins after	0.95	0.99	1.40	1.40	-2.785	0.005

Note: Mean change = (Mean of end-tidal CO_2 each point over time – Mean of end-tidal CO_2 at baseline); ns = non significant

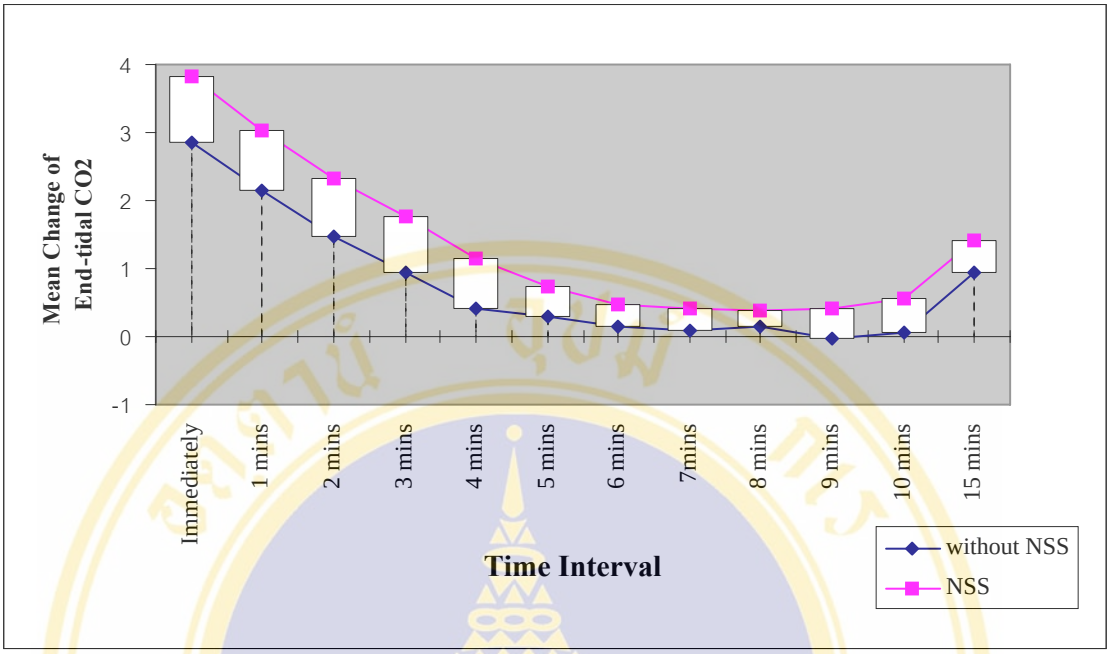

Figure 12: Mean change of end-tidal CO_2 over time

Hypothesis 3: The mean of heart rate of patients who had the endotracheal suctioning with normal saline instillation increase more than those of the patients who had the endotracheal suctioning without normal saline instillation.

Heart rate was measured by BCI Capnocheck Plus (BCI-9004), which was a noninvasive device. From Table 7 and Figure 13, the mean of heart rate in the endotracheal suctioning with and without normal saline instillation displayed the increasing trend. The mean of heart rate of endotracheal suctioning without normal saline instillation returned to baseline at 10 minutes after suctioning.

Table 7: Mean of Heart Rate Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

Heart Rate	Method A(without NSS)		Method B(with NSS)	
	M (BPM)	SD	M (BPM)	SD
Before suctioning (baseline)	114.43	19.42	114.25	19.70
Immediately after	139.14	17.10	141.98	16.77
1 min after	136.38	17.19	139.51	16.87
2 mins after	133.49	17.30	137.06	17.10
3 mins after	130.60	17.28	133.94	17.37
4 mins after	127.68	17.51	131.48	17.63
5 mins after	124.38	17.82	128.68	17.96
6 mins after	121.83	18.21	125.97	18.09
7 mins after	119.26	18.55	123.20	18.39
8 mins after	116.78	18.86	120.60	18.48
9 mins after	115.23	19.37	118.18	18.99
10 mins after	114.25*	19.33	115.91	19.33
15 mins after	113.97	19.17	114.86	19.39

Note: BPM = beats per minute; * = The recovery time to return to baseline

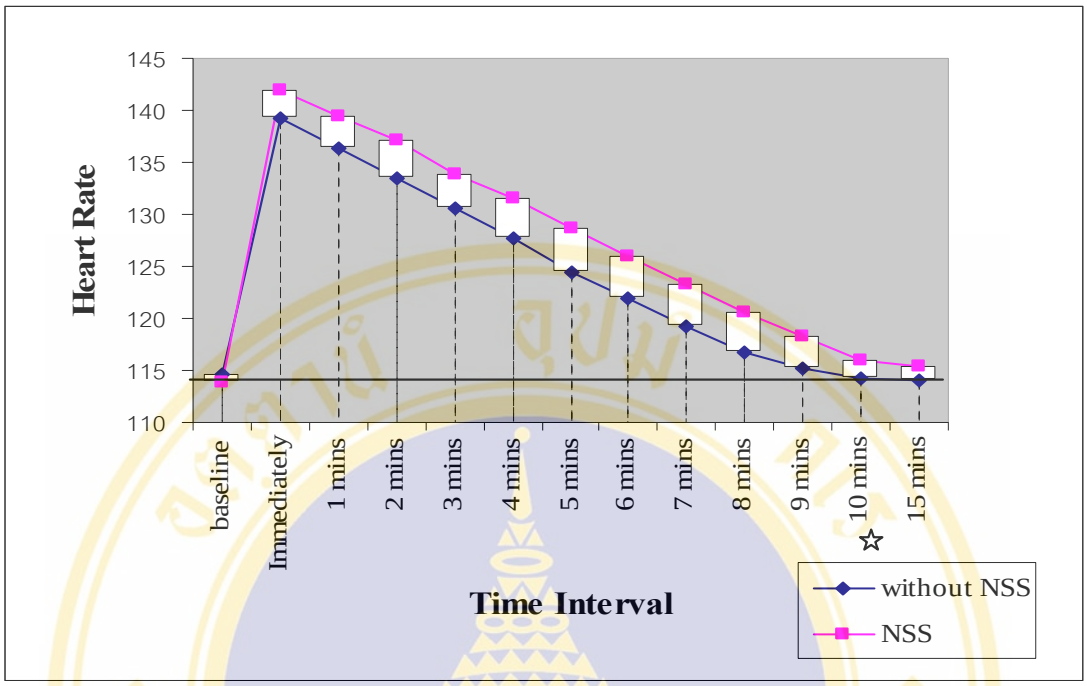

Figure 13: Mean of heart rate over time

To examine the differences in the mean change of heart rate of endotracheal suctioning with and without normal saline instillation, paired t-test was used. The result indicated that the mean change of heart rate of subjects in the endotracheal suctioning with and without normal saline instillation over time were statistically significant differences ($p<.05$). The mean change of heart rate in the endotracheal suctioning without normal saline instillation ranged from -0.18 to 24.71 beats per minute, whereas it ranged from 0.62 to 27.74 beats per minute in the endotracheal suctioning with normal saline instillation (See Table 8 and Figure 14).

Table 8: A Comparison by Paired t-test of the Mean Change of Heart rate Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

Heart Rate	Method A (without NSS) Mean change (BPM)	SD	Method B (with NSS) Mean change (BPM)	SD	t-value	p-value
Immediately after	24.71	8.64	27.74	9.31	-9.750	0.00
1 min after	21.95	8.15	25.26	8.91	-9.760	0.00
2 mins after	19.06	8.00	22.82	8.77	-7.493	0.00
3 mins after	16.17	7.96	19.69	8.19	-10.080	0.00
4 mins after	13.25	7.61	17.23	8.14	-11.459	0.00
5 mins after	9.95	6.72	14.43	7.90	-10.351	0.00
6 mins after	7.40	6.20	11.72	7.72	-9.832	0.00
7 mins after	4.83	5.43	8.95	6.76	-8.970	0.00
8 mins after	2.35	5.22	6.35	6.20	-8.284	0.00
9 mins after	0.80	4.52	6.35	6.20	-10.264	0.00
10 mins after	-0.18	4.08	1.66	4.81	-4.985	0.00
15 mins after	-0.46	3.61	0.62	3.96	-3.523	0.001

Note: Mean change = (Mean of heart rate each point over time – Mean of heart rate at baseline);
BPM = beats per minute

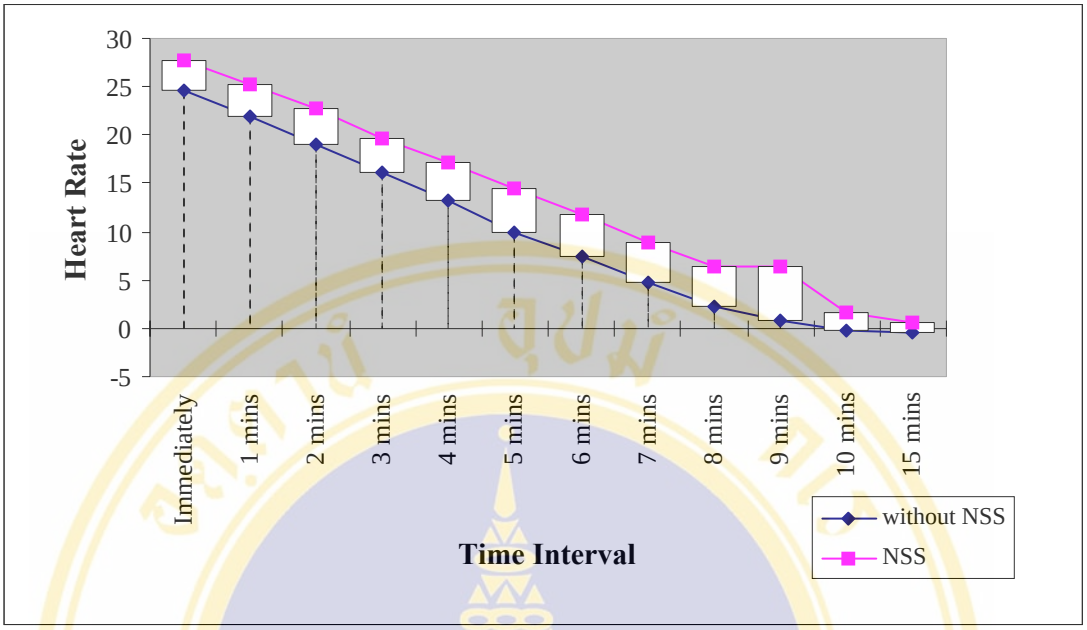

Figure 14: Mean change of heart rate over time

Hypothesis 4: The mean of mean blood pressure of patients who had the endotracheal suctioning with normal saline instillation increase more than those of the patients who had the endotracheal suctioning without normal saline instillation.

Mean blood pressure referred to the average pressure in the system throughout the cardiac cycle. It was not the arithmetic average of the diastolic and systolic pressures, and estimated by diastolic blood pressure plus one-third of the pulse pressure as measured by Phillip (Model No. M 1205A), which was a noninvasive device.

The mean of mean blood pressure showed the increase after endotracheal suctioning in both methods. The mean of mean blood pressure of the endotracheal suctioning with normal saline instillation increased more than the endotracheal suctioning without normal saline instillation, and returned to baseline on both methods at 15 and 10 minutes after suctioning, respectively (See Table 9 and Figure 15).

Table 9: Mean of Mean Blood Pressure Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

Mean blood pressure	Method A(without NSS)		Method B(with NSS)	
	M (mmHg)	SD	M (mmHg)	SD
Before suctioning (baseline)	69.74	10.24	70.46	9.88
Immediately after	83.77	11.03	86.48	10.77
5 mins after	74.23	11.02	77.46	10.48
10 mins after	67.48*	9.20	70.66	10.18
15 mins after	67.77	8.96	68.65*	9.18

Note: * = The recovery time to return to baseline

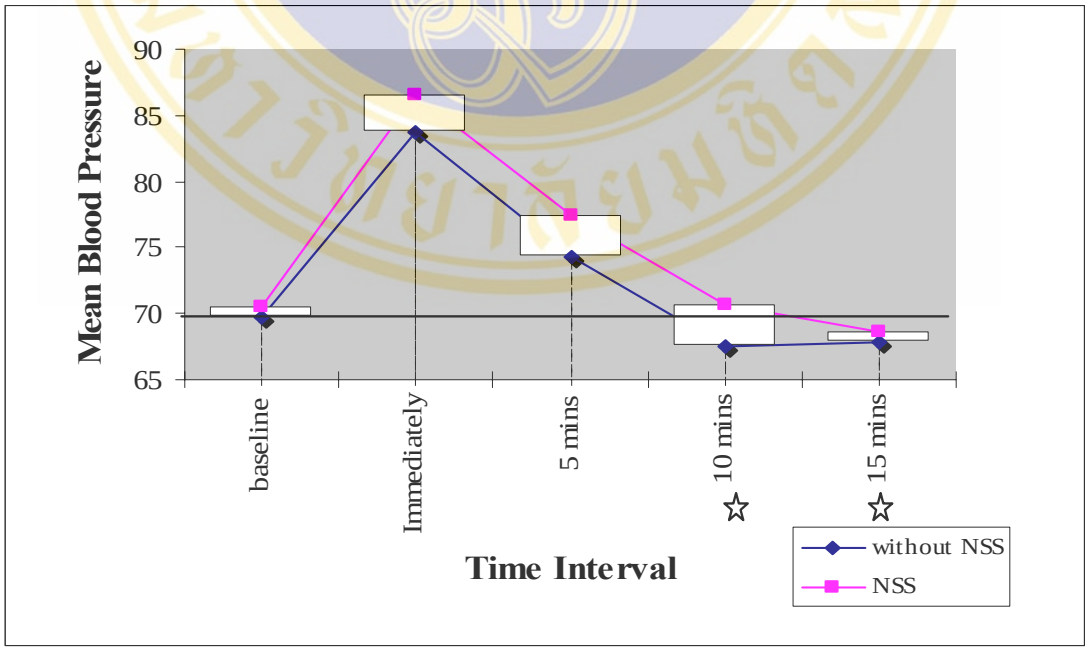

Figure 15: Mean of mean blood pressure over time

Paired t-test was used to detect the difference mean change of mean blood pressure in endotracheal suctioning with and without normal saline. The analysis showed that there were statistically significant differences in the mean change of mean blood pressure over time between the subjects in both methods ($p<.05$), except for the mean change of mean blood pressure at 15 minutes after suctioning that there was no statistically significant differences ($p>.05$). The mean change of mean blood pressure of subjects in the endotracheal suctioning without normal saline ranged from -2.26 to 14.03 mmHg, whereas it range from -1.82 to -16.02 mmHg in the endotracheal suctioning without normal saline instillation (See Table 10 and Figure 16).

Table 10: A Comparison by Paired t-test of the Mean Change of Mean Blood Pressure Over Time between the Endotracheal Suctioning with and without Normal Saline Instillation (n=65)

Mean Blood Pressure	Method A (without NSS) Mean change (mmHg)	SD	Method B (with NSS) Mean change (mmHg)	SD	t-value	p-value
Immediately after	14.03	8.85	16.02	8.82	-4.026	.00
5 mins after	4.49	6.65	7.00	6.15	-3.472	.00
10 mins after	-2.26	5.13	0.20	6.47	-4.261	.00
15 mins after	-1.97	6.06	-1.82	5.17	-2.229	.82^{ns}

Note: Mean change = (Mean of mean blood pressure each point over time – Mean of mean blood pressure at baseline); ns = non significant

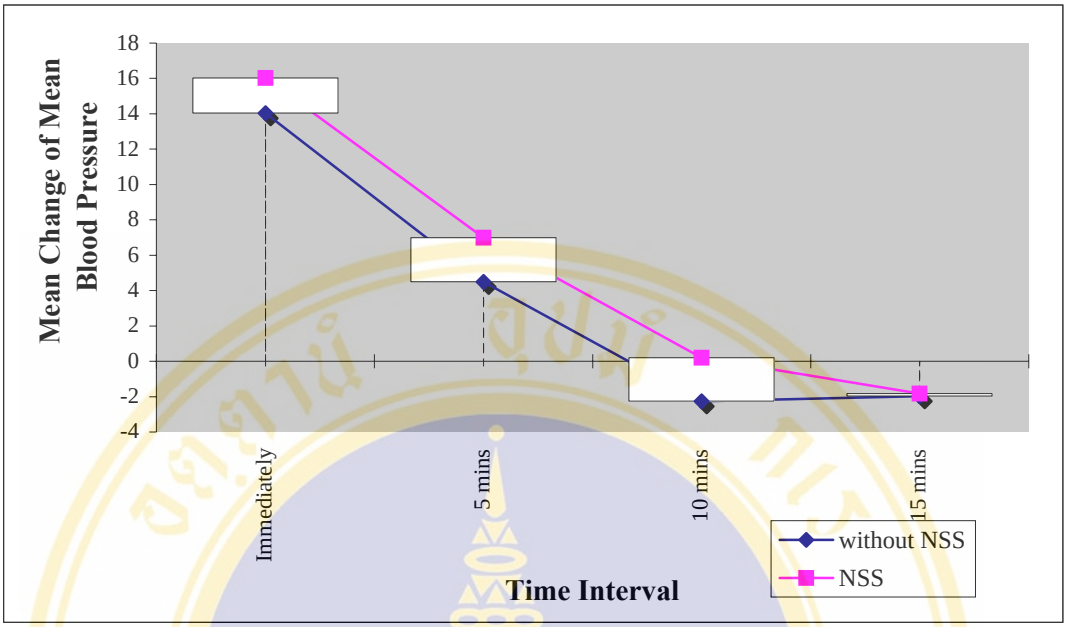

Figure 16: Mean change of mean blood pressure over time

CHAPTER V
DISCUSSION

The research was designed to study the effects of endotracheal suctioning with and without normal saline instillation on physiologic changes in pediatric patients with mechanical ventilation. The finding was discussed according to the results of the hypothesis testing.

Characteristics of the Sample

The endotracheal suctioning was performed with 58 subjects yielded 65 observed suctioning. Most of subjects' age ranged between 1 to 12 months (36.2%) and were male (62.1%). The majority of primary diagnoses were neurological problems (34.5%) (Table 1). Most of mechanical ventilators were used with heated wired humidifier (69.2%). The subject received with 80% maintenance of total fluid intake (56.9%). The most subjects were intubated with of the oral endotracheal tube size 4 to 5 mm (60.0%), and suctioned with the catheter No. 8 Fr (60.0%). Duration of using the endotracheal tube ranged from 1 to 7 days (87.7%). The sample received the endotracheal suctioning ranged from 6 to 10 times per day (83.1%), and the most period of suctioning time in each episode was 1 to 5 minutes (90.8%) (Table2).

Hypotheses Testing

Hypothesis 1: The mean of oxygen saturation of patients who had the endotracheal suctioning with normal saline instillation decrease more than the patients who had the endotracheal suctioning without normal saline instillation.

The findings in this study showed significant support for hypothesis 1. The mean of oxygen saturation of subjects after the endotracheal suctioning with normal saline instillation decreased more significantly when compared with the endotracheal suctioning without normal saline instillation (Table 3). The mean change of oxygen saturation over time between the subjects in both methods showed statistically

significant differences, whilst the mean change of oxygen saturation at 7, 8, and 10 minutes after suctioning showed no statistically significant differences (Table 4). This could be explained as follows:

The function of the respiratory system is to supply the blood with oxygen in order that the blood delivers oxygen to all parts of the body through breathing. When we breathe, we inhale oxygen and exhale carbon dioxide. This exchange of gases is the respiratory system's means of getting oxygen to the blood. This takes four major processes of mechanism of the respiratory system which consist of ventilation, perfusion, gas exchange, and oxygen transport (Liangchai Limlomwong, B.E. 2538; Sudpranom Samantawaekin, B.E. 2543; West & Wagner, 1997). The endotracheal suctioning leads to oxygen desaturation. Because of the suction catheter is placed in the airway and a vacuum applied, oxygen is sucked out of the lungs. The concentration of oxygen in airway and alveolar has reduced considerably which causing hypoventilation and oxygen desaturation. Besides, the endotracheal suctioning with normal saline instillation lead to more hypoxemia and oxygen desaturation. Because of the normal saline has large particle, it will retain in the airway mostly. The hyperinflation using a manual resuscitating bag for 1 time produces inadequate pressure in making solution to be broken into small particles. All of the normal saline remained in the trachea and main bronchi, and none of it reached the periphery of the lung (Hanley, et al., 1978). Demers and Saklad (1973) suggested that the mucus and water in bulk form were immiscible, even after vigorous shaking. Morever, the danger of normal saline instillation as precaution is airway obstruction due to secretion having large particle from solution absorption and bronchospasm. The remaining of normal saline instillation can interrupt the ventilation and oxygenation leading to hypoxemia and oxygen desaturation. When considering the characteristics of the sample in this study, it was found that the ages ranged between 1 to 12 months (36.2%) (Table 1). Most subjects were intubated with a size of the oral endotracheal tube from 4 to 5 mm (60.0%), and size of the suction catheter was 8 Fr (60.0%) (Table 2). It could be seen that the pediatric patients had smaller trachea than the adult's, and used the small size of oral endotracheal tubes. Thus, the normal saline instillation leads more airway obstruction and hypoxemia, which reduce the oxygenation and ventilation resulting in oxygen desaturation. Normally, the airway provides an important conduit for air to

travel to the alveoli, but also offer some resistance to ventilation. Airway resistance is influenced by many factors such as mucus or inflammation of the airway. In the pediatric patients, if the size of trachea has diminished less than 2 mm, it may possible to increase airway resistance to 16-fold. Whereas, the size of trachea has diminished less than 2 mm in adult patients, it may increase airway resistance to 3-fold (Aroonwan Pruettipan, B.E.2545). Aforementioned, the finding in this study showed significant support for hypothesis 1.

The significant results of this study were consistent with the previous studies. Ackerman and Gugerty (1990) studied the effects of normal saline instillation before suctioning on oxygen saturation in 26 critically ill adult patients. They found that a greater decrease in oxygen saturation and delayed improvement in oxygen saturation after suctioning with normal saline instillation. Ackerman (1993) studied the effects of endotracheal suctioning with and without normal saline instillation on oxygen saturation in 40 adult patients post operation open heart surgery, and found that oxygen saturation decreased at 2, 3, 4, and 5 minutes after endotracheal suctioning with normal saline instillation. Ackerman and Mick (1998) evaluated the effects of the endotracheal suctioning with and without normal saline instillation on oxygen saturation in 29 adult critically ill patients with pulmonary infections. They found that oxygen saturation decreased at 4, 5, and 10 minutes after suctioning with normal saline instillation. In the pediatric patient study, Ridling, Martin, and Bratton (2003) studied the effects of the endotracheal suctioning with and without normal saline instillation in 24 critically ill children, and found that oxygen saturation decreased at 1 and 2 minutes after endotracheal suctioning with normal saline instillation. Besides, in this study, the results showed the mean of oxygen saturation of the endotracheal suctioning with normal saline instillation returned to baseline longer than the endotracheal suctioning without normal saline instillation at 5 minutes and 4 minutes, respectively (See Table 3 and Figure 9). Because of the remaining of normal saline instillation obstructed the ventilation. This result was in accordance with the data from Kinloch (1999) who studied 35 patients after coronary bypass grafting, and found that the recovery times of oxygen saturation to return to baseline after endotracheal suctioning without and with normal saline instillation were average of 3.78 minutes and 7.30 minutes, respectively. Ji, Kim and Park (2002) studied the effects of normal

saline instillation before suctioning on oxygen saturation in 16 critically ill patients with pneumonia. Their results showed that the recovery times for oxygen saturation to return to baseline after suctioning without normal saline instillation at 45 seconds and excess of 5 minutes in the case of normal saline instillation.

Hypothesis 2: The mean of end-tidal CO_2 of patients who had the endotracheal suctioning with normal saline instillation increase more than the patients who had the endotracheal suctioning without normal saline instillation.

The findings in this study showed significant support for hypothesis 2. The mean of end-tidal CO_2 of subjects after the endotracheal suctioning with normal saline instillation more significantly increased when compared with the endotracheal suctioning without normal saline instillation (Table 5). The mean change of end-tidal CO_2 over time between the subjects in both methods showed statistically significant differences, whilst the mean change of end-tidal CO_2 at 8 minutes after suctioning showed no statistically significant difference (Table 6). This could be explained as follows:

Normally, gas exchange requires the movement of gases into and out of the lungs. Various control mechanisms and forces interplay in the two phases of ventilation: inspiration and expiration. Inspiration is the process of moving air into the lungs; expiration moves it out. Besides, the ventilation is the process of moving air into lungs (alveoli) and distributing air within the lungs to gas exchange units for maintenance of oxygenation and removal of carbon dioxide (Schumann, 1988). The endotracheal suctioning reduces the ventilation. Because of the air in airway is suctioned, it may cause reduction of oxygen. Besides, the suction catheter can obstruct and interrupt moving air into the lungs. These may result in reduction of ventilation and retention of carbon dioxide. Furthermore, the endotracheal suctioning with normal saline instillation leads more hypoventilation. Because of the normal saline is foreign body that falling into the endotracheal tube and airway, the results are airway obstruction, interruption of ventilation and diffusion of oxygen and carbon dioxide between alveolar and capillary of lungs. When considering the characteristics of the sample in this study, it was found that the majority of common diagnoses were neurological problem (34.5%) such as seizure disorder, AVM rupture, and

hydrocephalus (Table 1). The pediatric patients with neurological problems should be carefully observed in nursing care, especially during endotracheal suctioning or normal saline instillation, as these patients often have hypoventilation and unstable to release of carbon dioxide leading to retention of carbon dioxide. Moreover, the retention of normal saline in airway will obstruct the ventilation and diffusion of oxygen and carbon dioxide resulting in more hypoxemia, hypoventilation, and carbon dioxide retention. Besides, the result in this study found that the mean of end-tidal CO_2 returned to baseline at 9 minutes after endotracheal suctioning without normal saline instillation, but the mean of end-tidal CO_2 of endotracheal suctioning with normal saline instillation did not return to baseline within 15 minutes after connecting the mechanical ventilator (See Table 5 and Figure 11). Because of the most of sample in this study was the pediatric patient with neurological problems, the remaining of normal saline instillation affected on the recovery time of end-tidal CO_2 of endotracheal suctioning with normal saline instillation did not return to baseline within 15 minutes. Thus, the findings in this study showed significant support for hypothesis 2.

This finding was partly consistent with the finding of Gray, MacIntryre and Kronenberger (1990). They studied the effects of endotracheal suctioning with and without normal saline instillation on carbon dioxide tension in 15 adult critically patients with lung problem. They found that the carbon dioxide tension of endotracheal suctioning with normal saline instillation increased more than the endotracheal suctioning without normal saline immediately with statistically significant difference, but later found the differences in carbon dioxide tension between the both methods had no statistically significant. This result was in accordance with the data from Akgul and Akyolcu (2002) which studied the effects of endotracheal suctioning with and without normal saline instillation on carbon dioxide tension in 20 adult critically ill patients with pulmonary or cardiovascular problems. They found that carbon dioxide tension of endotracheal suctioning with normal saline instillation increased more than the endotracheal suctioning without normal saline immediately with statistically significant difference, but later found the differences in carbon dioxide tension between the both methods showed no statistically significant.

Hypothesis 3: The mean of heart rate of patients who had the endotracheal suctioning with normal saline instillation increase more than the patients who had the endotracheal suctioning without normal saline instillation.

The findings in this study showed significant support for hypothesis 3. The mean of heart rate of subjects after the endotracheal suctioning with normal saline instillation more significantly increased when compared with that the endotracheal suctioning without normal saline instillation (Table 7). The mean change of heart rate over time between the subjects in both methods showed statistically significant differences (Table 8). This could be explained as follows:

The body has adapted to various stimulations, and the control is based on the coordination of cardiovascular center for adaptation to equilibrium. Heart rate has controlling by automatic nervous system, hormone and heart itself. The autonomic nervous system (ANS) provides an external influence on myocardial contractility and heart rate. This involves adjusting the heart rate and contractility to the body's demands. The endotracheal suctioning with normal saline instillation lead to a generalized increase of central nervous system arousal, signals from the hypothalamus transmits to autonomic control centers of the brain stem. These affect oxygen desaturation, hypoxemia and vagal stimulation. Parasympathetic nervous system or vagus nerve is stimulated which affect bradycardia immediately. The major effects of vagal stimulation are restraining or slowing influence on the SA node, atrial muscle, and AV node. Vagal stimulation slows the heart rate by restraining the rate of diastolic depolarization in the conduction tissue. When the body has bradycardia, the sympathetic nervous system is stimulated immediately. Receptors from the sympathetic nervous system are present in the atrial wall, ventricles, and SA and AV nodes. When stimulated, these cardioaccelerator fibers release norepinephrine, which stimulates β_1-receptors to increase the rate of depolarization and impulse transmission through the conduction tissue. Stimulation of sympathetic nervous system affects the tachycardia and increase the contractility (Banasik, 1995). The finding of this study showed that the mean of heart rate of endotracheal suctioning without normal saline instillation returned to baseline at 10 minutes after suctioning, but the mean of heart rate of endotracheal suctioning did not return to baseline within 15 minutes (See Table 7 and Figure 13). When considering the characteristics of the sample in this study, it

was found that the most sample was the pediatric patient with neurological problems (34.5%) (See Table 1). The normal saline instillation stimulated parasympathetic nervous system and sympathetic nervous system affected to tachycardia. The heart rate of the sample in this study is changed easier than the other patients.

The significant result of this study was consistent with the previous studies. Saowaluk Jiratamkul (B.E. 2534) studied the effects of endotracheal suctioning with or without normal saline instillation on the heart rate in 20 postoperative opened heart surgery patients with mechanical ventilator. It was found that the suctioning with normal saline instillation lead to the higher heart rate than the suctioning without normal saline instillation with statistically significant difference. Akgul and Akyolcu (2002) studied the effects of endotracheal suctioning with and without normal saline instillation on heart rate in 20 adult critically ill patients with pulmonary or cardiovascular problems. They found that the heart rate increased at 4 and 5 minutes after endotracheal suctioning with normal saline instillation. The difference was statistically significant.

The finding in this study was partly consistent with the finding of MacIntryre and Kronenberger (1990) who studied the effects of endotracheal suctioning with and without normal saline instillation on heart rate in 15 adult critically patients with lung problem. They found that the heart rate of endotracheal suctioning with normal saline instillation increased more than the endotracheal suctioning without normal saline immediately with statistically significant difference, but later found the differences in heart rate between the both methods showed no statistically significant. Besides, this finding study was inconsistent with the finding of Ackerman and Mick (1998) who studied the effects of the suctioning with and without normal saline instillation on heart rate in 29 adult critically ill patients with pulmonary infections. They found that heart rate was slightly increased for both groups and the differences between the groups were not statistically significant. Although the changes in heart rate in their study were small and insignificant, clinical interpretation of the data suggested that normal saline instillation was a source of stress for patients and could lead to many complications.

Hypothesis 4: The mean of mean blood pressure of patients who had the endotracheal suctioning with normal saline instillation increase more than the patients who had the endotracheal suctioning without normal saline instillation.

The findings in this study showed significant support for hypothesis 4. The mean of mean blood pressure levels of subjects after the endotracheal suctioning with normal saline instillation increased more significantly when compared with the endotracheal suctioning without normal saline instillation (Table 9). The mean change of mean blood pressure over time between the subjects in both methods were statistically significant differences, whilst the mean change of mean blood pressure at 15 minutes after suctioning showed no statistically significant differences (Table 10). This result could be explained as follows:

The control of arterial blood pressure is classified as homeostasis of body by negative feedback. Blood pressure is a reflection on circulation of the body. The nervous system almost entirely control circulation through the autonomic nervous system, which is capable of changing blood pressure immediately. The sympathetic nervous system is a major regulatory part of the circulation. In early stage of oxygen desaturation that the heart rate will decrease immediately, the body is compensated by negative feedback in stimulating the function of the sympathetic nerve system to increase which lead to an increase of vasoconstriction, cardiac vigor, and heart rate. The vasoconstriction hormones are epinephrine and norepinephrine. These two hormones act directly on blood vessels and vasoconstriction (Pipat Joedrangsee, B.E. 2545). Endotracheal suctioning with normal saline instillation leads to increase central nervous system arousal, which increases the sympathetic activity. When epinephrine and norepinephrine are release in the blood circulation, the blood pressure is thus increase. When considering the characteristics of the sample in this study, it was found most subjects were intubated with the oral endotracheal tube size 4 to 5 mm (60%), and suctioned with the catheter No. 8 Fr (60%) (Table 2). The pediatric patients had smaller trachea than the adult's, and used the small size of oral endotracheal tubes. Thus, the normal saline instillation interrupts the ventilation and more hypoxemia. The body has compensated by negative feedback to correct hypoxemia. Sympathetic nervous system is the activity resulting in an increase of blood pressure, in which the pediatric patients with neurological problems are sensitive to changing. Thus, the

mean of mean blood pressure of endotracheal suctioning with normal saline instillation increased more than endotracheal suctioning without normal saline instillation. The mean of mean blood pressure of endotracheal suctioning with normal saline instillation returned to baseline longer than endotracheal suctioning without normal saline instillation at 15 and 10 minutes after suctioning, respectively (See Table 9 and Figure 15).

The finding in this study was partly consistent with the previous study. MacIntryre and Kronenberger (1990) studied the effects of endotracheal suctioning with and without normal saline instillation on blood pressure in 15 adult critically patients with lung problem. They found that systolic and diastolic blood pressure of endotracheal suctioning with normal saline instillation increased more than the endotracheal suctioning without normal saline immediately with statistically significant difference, but later found the differences in blood pressure between the both methods showed no statistically significant. This result was in accordance with Ackerman and Mick (1998) who studied the effects of the suctioning with and without normal saline instillation on systolic blood pressure in 29 adult critically ill patients with pulmonary infections. They found that systolic blood pressure of suctioning with normal saline instillation increased more than the suctioning without normal saline instillation immediately with statistically significance, and differences between the groups were not significant at any point of the time points.

In summary, the findings of this study indicated that the mean of oxygen saturation of subjects in the endotracheal suctioning with normal saline instillation more significantly decrease when compared with the endotracheal suctioning without normal saline instillation. The mean of end-tidal CO_2, heart rate, and mean blood pressure of subjects in the endotracheal suctioning with normal saline instillation more significantly increase when compared with the endotracheal suctioning without normal saline instillation.

In the study, it was found that instillation of normal saline before endotracheal suctioning results in a decrease of oxygen saturation, increase of carbon dioxide tension, heart rate and blood pressure, which agrees with the current literature. Some evidence, it is has been shown that this does not represent a danger to these

subjects. However, the nurse should be careful when endotracheal suctioning with normal saline instillation, to monitor patient status.

CHAPTER VI
CONCLUSION

The conclusion of this study is presented as follows: summary of the study, limitations of the study, recommendations for nursing practice, and recommendations for further study.

Summary of the Study

This study was crossover-experimental design that aimed to determine the effects of endotracheal suctioning with and without normal saline instillation on physiologic changes in the pediatric patients with mechanical ventilator. The subject in this study was selected by purposive sampling with consisted of 58 pediatric patients yield 65 observed suctioning in the Pediatric Intensive Care Unit (PICU) at Faculty of Medicine Ramathibodi Hospital. The samples were pediatric patients who were newborn to 15 years providing their hemodynamic condition were stable longer than 6 hours before the endotracheal suctioning. Whereas the data were collected during the period of July 2005 to January 2006.

The instruments used in this study included experimental instruments and data collection instruments. The experimental instruments included the mechanical ventilation with a heated humidifier; sterilized normal saline for instillation; suctioning equipments; resuscitating bag such as size 500 ml or 1,000 ml with 100% oxygen supply valve with PEEP valve device, and pressure gauge. The data collection instruments included sidestream capnometer (BCI Capnocheck Plus model BCI-9004) was used to measure oxygen saturation, end-tidal CO_2 and heart rate parameters, the bedside monitor (model No. M1205A, a non-invasive and automatic device) was used to measure blood pressure, timer, stethoscope, and the data record forms (a demographic data record form, and a physiological data record form).

A crossover-experimental design was used in this study, in which each subjects served as their own control. Each subject was exposed to both methods: the

endotracheal suctioning with and without normal saline instillation. The sequence of experimental method of eligible subjects was assigned by drawing lots (number 1 to 65) without replacement. The subjects were recorded on oxygen saturation, end-tidal CO_2, and heart rate parameters as baseline, immediately connecting the mechanical ventilator and after suctioning at minute 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, and 15. The mean blood pressure parameter was recorded as baseline, immediately connecting the mechanical ventilator and after suctioning at minute 5, 10, and 15. The duration between each suctioning method was 3 hours before starting the other method.

Fifty-eight pediatric patients on the endotracheal tube with mechanical ventilation were purposively selected for the study according to the inclusion criteria. The subject consisted of 36 males and 22 females; ages ranged between 1 to 12 months for the most part. The majority of primary diagnoses were neurological problems where most of the mechanical ventilators used were heated wired humidifier.

Data were analyzed by using the paired t-test and wilcoxon signed ranks test wherein the results were recorded as follows:

1. A comparison of the mean change of oxygen saturation over time between the endotracheal suctioning with and without normal saline instillation showed a higher decrease in the subjects after the endotracheal suctioning with normal saline instillation than after the endotracheal suctioning without normal saline instillation with a statistical significant difference ($p<.05$). The mean of oxygen saturation of the endotracheal suctioning with normal saline instillation returned to baseline longer than the endotracheal suctioning without normal saline at 5 minutes and 4 minutes, respectively.

2. A comparison of the mean change of end-tidal CO_2 over time between the endotracheal suctioning with and without normal saline instillation indicated a higher increase in the subjects after the endotracheal suctioning with normal saline instillation than after the endotracheal suctioning without normal saline instillation with a statistical significant difference ($p<.05$). The mean of end-tidal CO_2 returned to baseline 9 minutes after the endotracheal suctioning without normal saline instillation.

3. A comparison of the mean change of heart rate over time between the endotracheal suctioning with and without normal saline instillation indicated a higher

increase in the subjects after the endotracheal suctioning with normal saline instillation than after the endotracheal suctioning without normal saline instillation with a statistical significant difference ($p<.05$). The mean of heart rate returned to baseline 10 minutes after the endotracheal suctioning without normal saline instillation.

4. A comparison of the mean change of mean blood pressure over time between the endotracheal suctioning with and without normal saline instillation revealed a higher increase in the subjects after the endotracheal suctioning with normal saline instillation than after the endotracheal suctioning without normal saline instillation with a statistical significant difference ($p<.05$). The mean of mean blood pressure of the endotracheal suctioning with normal saline instillation returned to baseline longer than the endotracheal suctioning without normal saline at 15 minutes and 10 minutes, respectively.

Limitations of the Study

The study limitation was not overlooked because it might interfere with the outcome variables. The limitation in this study was medications received such as mucolytic drugs and bronchodilators. The researcher tried to control the equivalent conditions regarding the medication received (e.g., the time interval before receiving the medication, the time schedule for starting the experiment, and the kind of medications used) of both the methods.

Recommendations for Nursing Practice

The results of this research showed that the endotracheal suctioning with normal saline instillation had a decrease of oxygen saturation, increase of end-tidal CO_2, increase of heart rate and blood pressure more than the endotracheal suctioning without normal saline instillation.

Thus, the nursing practice should be altered to the best interest of the patients as follows:

1. These data should be used to develop an endotracheal suctioning clinical nursing pratice guideline.

2. To assess the lung sound for evaluation of the necessity in endotracheal suctioning as this procedure may lead to many complications such as decrease of oxygen saturation, increase of end-tidal CO_2, heart rate, and blood pressure.

3. Instillation of normal saline should not be routine and that each pediatric patient should be individually assessed to determine whether or not normal saline instillation in required.

4. The use of normal saline instillation such as tenacity secretion is necessary. The amount of normal saline usage varies with the ages of the pediatric patients: if less than 1 year use 0.5 ml; 1-8 years use 1 ml; and older than 8 years use 2 ml (Ridling, Martin & Bratton, 2003).

5. To be more careful on endotracheal suctioning with normal saline instillation in the pediatric patient with neurological problem due to the retention of normal saline in airway may obstruct the ventilation, lead to retain carbon dioxide more, and cause higher increase intracranial pressure.

In addition, the nursing organization should provide knowledge to nurses in the endotracheal suctioning appropriately providing a supportive reason for normal saline instillation.

Recommendations for Further Research

1. In this study, the researcher found that the mean of heart rate and mean blood pressure of endotracheal suctioning with normal saline instillation did not return to baseline within 15 minutes after connecting the mechanical ventilator. Further study should increase the period of times on data collection, which consisted of oxygen saturation, end-tidal CO_2, heart rate and mean blood pressure until these variables return to the baseline.

2. Additional research should study other variables as follows:

- The comparison of the endotracheal suctioning with and without normal saline instillation on discomfort in the pediatric patients with mechanical ventilation.

- The comparison the effects of the endotracheal suctioning with and without normal saline on the amounts of secretion suctioned in the pediatric patients with mechanical ventilation.

- The effects of normal saline instillation before endotracheal suctioning on lower airway infection in the patients with mechanical ventilation.

- The comparison of the effects of the endotracheal suctioning with and without normal saline instillation on the cost in the patients with mechanical ventilation.

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APPENDIX

APPENDIX A

คณะแพทยศาสตร์ โรงพยาบาลรามาธิบดี มหาวิทยาลัยมหิดล
ถนนพระราม 6 กทม. 10400
โทร. (662) 354-7275, 201-1296 โทรสาร (662) 354-7233

Faculty of Medicine, Ramathibodi Hospital, Mahidol University
Rama VI Road, Bangkok 10400, Thailand
Tel. (662) 354-7275, 201-1296 Fax (662) 354-7233

Documentary Proof of Ethical Clearance Committee on Human Rights Related to Researches Involving Human Subjects Faculty of Medicine, Ramathibodi Hospital, Mahidol University

No. 0540/2005

Title of Project	A Comparison of the Effects of Endotracheal Suctioning with and without Normal Saline Instillation on Physiologic Changes in Pediatric Patients with Mechanical Ventilator
Protocol Number	ID 05 – 48 – 06
Principal Investigator	Miss Jiraporn Punyoo
Official Address	Department of Nursing Faculty of Medicine, Ramathibodi Hospital Mahidol University

The aforementioned project has been reviewed and approved by Committee on Human Rights Related to Researches Involving Human Subjects, based on the Declaration of Helsinki.

Signature of Chairman
Committee on Human Rights Related to
Researches Involving Human Subjects ..... Prof. Krisada Ratana-olarn, M.D., FRCST, FICS.

Signature of Dean ..... Prof. Rajata Rajatanavin, M.D., F.A.C.E.

Date of Approval May 18, 2005

คณะแพทยศาสตร์ โรงพยาบาลรามาธิบดี มหาวิทยาลัยมหิดล
ถนนพระราม 6 กทม. 10400
โทร. (662) 354-7275, 201-1296 โทรสาร (662) 354-7233
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Rama VI Road, Bangkok 10400, Thailand
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เอกสารรับรองโดยคณะกรรมการจริยธรรมการวิจัยในคน
คณะแพทยศาสตร์โรงพยาบาลรามาธิบดี
มหาวิทยาลัยมหิดล

เลขที่ ๐๕๔๐/๒๕๔๘

ชื่อโครงการ การศึกษาเปรียบเทียบผลของการหยอดและไม่หยอดน้ำเกลือนอร์มัลผ่านท่อหลอดลมคอก่อนการดูดเสมหะต่อการเปลี่ยนแปลงทางด้านสรีรวิทยาในผู้ป่วยเด็กที่ต้องใช้เครื่องช่วยหายใจ

เลขที่โครงการ/รหัส ID ๐๕ – ๔๘ – ๐๖ ว

ชื่อหัวหน้าโครงการ นางสาวจิราภรณ์ ปั้นอยู่

ที่ทำงาน ภาควิชาพยาบาลศาสตร์
คณะแพทยศาสตร์โรงพยาบาลรามาธิบดี
มหาวิทยาลัยมหิดล

ขอรับรองว่าโครงการดังกล่าวข้างต้นได้ผ่านการพิจารณาเห็นชอบโดยสอดคล้องกับแนวปฏิญญาเฮลซิงกิ จากคณะกรรมการจริยธรรมการวิจัยในคน คณะแพทยศาสตร์โรงพยาบาลรามาธิบดี

ลงนาม _____
ประธานกรรมการจริยธรรมการวิจัยในคน (ศาสตราจารย์ นายแพทย์กฤษฎา รัตนโอพาร)

ลงนาม _____
คณบดีคณะแพทยศาสตร์โรงพยาบาลรามาธิบดี (ศาสตราจารย์ นายแพทย์รัชตะ รัชตะนาวิน)

วันที่รับรอง ๑๘ พฤษภาคม ๒๕๔๘

คณะแพทยศาสตร์ โรงพยาบาลรามาธิบดี มหาวิทยาลัยมหิดล
ถนนพระราม 6 กทม. 10400
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Rama VI Road, Bangkok 10400, Thailand
Tel. (662) 354-7275, 201-1296 Fax (662) 354-7233

ที่ จวค ๑๔๖๙/๒๕๔๘ คณะกรรมการจริยธรรมการวิจัยในคน

วันที่ ๖ กรกฎาคม ๒๕๔๘

เรื่อง แจ้งผลการพิจารณาของคณะกรรมการจริยธรรมการวิจัยในคน

เรียน นางสาวจิราภรณ์ ปิ่นอยู่

อ้างถึงโครงการวิจัยเรื่อง การศึกษาเปรียบเทียบผลของการหยอดและไม่หยอดน้ำเกลือนอร์มัลผ่านท่อหลอดลมคอก่อนการดูดเสมหะต่อการเปลี่ยนแปลงทางด้านสรีรวิทยาในผู้ป่วยเด็กที่ต้องใช้เครื่องช่วยหายใจ

หมายเลขโครงการวิจัย ID ๐๕ - ๔๘ - ๐๖ ว

ในนามของคณะกรรมการจริยธรรมการวิจัยในคน ผมขอแสดงความยินดีที่โครงการวิจัยดังกล่าวข้างต้นของท่านได้ผ่านความเห็นชอบ จากคณะกรรมการฯแล้ว

เพื่อให้สอดคล้องกับระเบียบปฏิบัติคณะแพทยศาสตร์โรงพยาบาลรามาธิบดี ว่าด้วยการศึกษาวิจัยและการทดลองในมนุษย์ พ.ศ. ๒๕๔๔ คณะกรรมการฯ ขอให้ท่านถือปฏิบัติโดยเป็นไปตามข้อแนะนำดังต่อไปนี้

๑. การดำเนินการวิจัยจะต้องเป็นไปตามโครงร่างวิจัยล่าสุดที่ผ่านการพิจารณาจากคณะกรรมการจริยธรรมการวิจัยในคนแล้ว
๒. การดำเนินการวิจัยจะต้องไม่เบี่ยงเบนไปจากโครงร่างวิจัยหรือมีการเปลี่ยนโครงร่างการวิจัยก่อนที่การแก้ไขเพิ่มเติมโครงร่างวิจัยนั้นจะได้รับการอนุมัติและเห็นชอบจากคณะกรรมการจริยธรรมการวิจัยในคนก่อน ยกเว้นในกรณีจำเป็นที่จะต้องกระทำไปก่อนเพื่อขจัดอันตรายเฉพาะหน้าที่เกิดขึ้นกับผู้ยินยอมตนให้ทำวิจัย
๓. ในกรณีที่มีการเปลี่ยนแปลงชื่อโครงการไปจากชื่อเดิมที่เสนอไว้ ต่อคณะกรรมการฯ ต้องแจ้งชื่อมายังคณะกรรมฯ เพื่อออกหนังสือรับรองให้เสมอ
๔. ผู้ยินยอมตนให้ทำวิจัยจะต้องได้รับเอกสารชี้แจงข้อมูล/คำแนะนำแก่ผู้ยินยอมตนให้ทำวิจัย (Patient/Participant Information Sheet) และลงนามในหนังสือยินยอมโดยได้รับการบอกกล่าวและเต็มใจ (Informed Consent Form) ก่อนเริ่มดำเนินการวิจัย
๕. ในเอกสารชี้แจงข้อมูล/คำแนะนำแก่ผู้ยินยอมตนให้ทำวิจัย (Patient's Information Sheet) จะต้องพิมพ์ข้อความดังต่อไปนี้ไว้ ด้วยทุกครั้ง

" ถ้าท่านมีข้อข้องใจหรือมีความกังวลใจเกี่ยวกับวิธีดำเนินการวิจัยของโครงการวิจัยนี้ ท่านสามารถติดต่อได้ที่ ประธานกรรมการ จริยธรรมการวิจัยในคน คณะแพทยศาสตร์โรงพยาบาลรามาธิบดี หน่วยจริยธรรมการวิจัยในคน สำนักงานวิจัยคณะฯ อาคารวิจัยและสวัสดิการ ชั้น ๓ (ห้อง ๓) โทรศัพท์ ๐๒-๒๐๑ ๑๕๔๔ ในเวลาราชการ"

๖. ความลับของผู้ยินยอมตนให้ทำวิจัย จะต้องถูกปกปิดไว้ตลอดเวลา ยกเว้นถ้าเป็นคำสั่งตามกฎหมาย

สุดท้ายนี้ ขอให้โครงการวิจัยของท่านประสบผลสำเร็จตามความมุ่งหมายอันจะนำมาซึ่งความเจริญก้าวหน้าทางวิชาการและเพื่อประโยชน์ของมนุษยชาติสืบต่อไป

ขอแสดงความนับถือ

(ศาสตราจารย์คลินิกเกียรติคุณเกียรวิทย์ ตันติแพทยางกูร)
รักษาการแทนประธานกรรมการจริยธรรมการวิจัยในคน

INFORMED CONSENT FORM (THAI VERSION)

หนังสือยินยอมโดยได้รับการบอกกล่าวและเต็มใจ

สำหรับผู้เข้าร่วมการวิจัยที่ไม่สามารถแสดงความยินยอมได้ด้วยตนเอง

ชื่อโครงการ การศึกษาเปรียบเทียบผลของการหยอด และไม่หยอดน้ำเกลือนอร์มัลผ่านท่อหลอดลมคอ ก่อนการดูดเสมหะต่อการเปลี่ยนแปลงทางด้านสรีรวิทยาในผู้ป่วยเด็กที่ต้องใช้เครื่องช่วยหายใจ

ชื่อผู้วิจัย นางสาว จิราภรณ์ ปั้นอยู่

*ชื่อผู้เข้าร่วมการวิจัย……..

อายุ……เลขที่เวชระเบียน…….

คำยินยอมของผู้มีอำนาจกระทำการแทนผู้เข้าร่วมการวิจัย

ข้าพเจ้า นาย/นาง/นางสาว……ซึ่งเป็นผู้มีอำนาจกระทำการแทนนาย/นาง/นางสาว/ด.ช./ด.ญ……ในฐานะ……ได้ทราบรายละเอียดของโครงการการวิจัย ตลอดจนประโยชน์ และข้อเสี่ยงที่จะเกิดขึ้นต่อผู้เข้าร่วมการวิจัยจากผู้วิจัยแล้วอย่าง ชัดเจนไม่สิ่งใดปิดบังซ่อนเร้นและยินยอมให้ทำการวิจัยในโครงการที่มีชื่อข้างต้น และข้าพเจ้ารู้ว่าถ้ามีปัญหาหรือ ข้อสงสัยเกิดขึ้นข้าพเจ้าสามารถสอบถามผู้วิจัยได้ และข้าพเจ้าสามารถไม่ให้ผู้เข้าร่วมการวิจัยเข้าร่วมโครงการวิจัยนี้เมื่อใดก็ได้ โดยไม่มีผลกระทบต่อการรักษาที่ผู้เข้าร่วมการวิจัยได้รับ นอกจากนี้ผู้วิจัยจะเก็บข้อมูลเฉพาะเกี่ยวกับ ตัวผู้เข้าร่วมการวิจัยเป็นความลับและจะเปิดเผยได้เฉพาะในรูปที่เป็นสรุปผลการวิจัย การเปิดเผยข้อมูลเกี่ยวกับตัวผู้เข้าร่วมการวิจัยต่อหน่วยงานต่างๆที่เกี่ยวข้อง กระทำได้เฉพาะกรณีจำเป็นด้วยเหตุผลทางวิชาการเท่านั้น

ลงชื่อ…… (ผู้มีอำนาจกระทำการแทน)

…….. (พยาน)

…….. (พยาน)

วันที่ ……

คำอธิบายของแพทย์หรือผู้ทำวิจัย

ข้าพเจ้าได้อธิบายรายละเอียดของโครงการ ตลอดจนประโยชน์ของการวิจัย รวมทั้งข้อเสี่ยงที่อาจจะเกิดขึ้นแก่ผู้เข้าร่วมการวิจัยให้ผู้มีอำนาจกระทำการแทนทราบแล้วอย่างชัดเจนโดยไม่มีสิ่งใดปิดบังซ่อนเร้น

ลงชื่อ…… (แพทย์หรือผู้วิจัย)

วันที่……

---

* ผู้เข้าร่วมการวิจัย หมายถึง ผู้ยินยอมตนให้ทำวิจัย

APPENDIX B

แบบบันทึกข้อมูล

<u>ส่วนที่ 1</u> (Part I) ข้อมูลทั่วไป

ผู้ป่วยรายที่ ……. อายุ ……. ปี……. เดือน

น้ำหนัก……กิโลกรัม

เพศ O หญิง O ชาย

การวินิจฉัยโรค O โรคหัวใจ ระบุ ……..

O โรคระบบทางเดินหายใจ ระบุ ……

O โรคหัวใจร่วมกับโรคระบบทางเดินหายใจ ระบุ ……

O โรคระบบประสาท ระบุ …….

O โรคระบบทางเดินอาหาร ระบุ ……...

O โรคเลือด ระบุ ……

O โรคอื่นๆ ระบุ ……

การรักษาที่ผู้ป่วยได้รับ……..…

……

……....

ปริมาณสารน้ำที่ผู้ป่วยได้รับ .....cc / kg / day (……% maintenance)

ผล x-ray ……

ผล arterial blood gas ครั้งสุดท้าย pH ……. , PCO_2 ……. mmHg, PO_2 ……. mmHg,

HCO_3 ……. mmol/L, Actual BE ……. mmol/L

ลักษณะของเสมหะ……..

Ventilator ที่ใช้ : ……. Setting mode ……

FiO_2 ……., flow…../min , PIP/PEEP…….. cmH_2O , IMV……. /min ,Ti……sec

Humidifier ของ Ventilator O non-heated wire humidifier เบอร์…….

O heated wire humidifier

temperature control ……C, chamber control ……C

ET-Tube No. …….. (stab …….. cms) , **สายดูดเสมหะ** เบอร์……

ใส่สายดูดเสมหะลึก …….cms

ระยะเวลาการใส่ ET-Tube ……วัน

ความถี่ของการดูดเสมหะ ……...ครั้ง/วัน

ระยะเวลาในการดูดเสมหะแต่ละครั้งเฉลี่ยนาน……นาที

ลำดับวิธีการทดลอง O ฉลากเลขคี่ (1, 3, 5,….., 65) ลำดับวิธีการทดลองเป็น A B

O ฉลากเลขคู่ (2, 4, 6,….., 64) ลำดับวิธีการทดลองเป็น B A

หมายเหตุ A = การดูดเสมหะที่ไม่มีการหยอดน้ำเกลือนอร์มัล

B = การดูดเสมหะร่วมกับการหยอดน้ำเกลือนอร์มัล

<u>ส่วนที่ 2</u> (Part II) แบบบันทึกข้อมูลการดูดเสมหะ

O **วิธีการทดลอง A (การดูดเสมหะที่ไม่มีการหยอดน้ำเกลือนอร์มัล)**

O **วิธีการทดลอง B (การดูดเสมหะร่วมกับการหยอดน้ำเกลือนอร์มัล)**

เวลาที่บันทึกข้อมูล	**ข้อมูลที่บันทึก**							
	ครั้งที่ 1				**ครั้งที่ 2**			
	SpO_2	end-tidal CO_2	HR	mABP	SpO_2	end-tidal CO_2	HR	mABP
***ค่าพื้นฐาน (Baseline)** (บันทึกก่อนปลดเครื่องช่วยหายใจ)								
หลังการดูดเสมหะ และต่อเครื่องช่วยหายใจทันที (Immediately after)								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 1								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 2								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 3								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 4								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 5								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 6								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 7								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 8								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 9								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 10								
หลังการต่อเครื่องช่วยหายใจ นาทีที่ 15								

ลักษณะของเสมหะ (ขณะทำการทดลอง) ……..……

The Suctioning Guideline

1. Assess the pediatric patient before endotracheal suctioning. The indications of endotracheal suctioning are as follows:

- Visible or audible secretions
- Feeling secretions in the chest (by the patient) and restlessness
- Increased airway pressure when ventilated and decreased tidal volume
- Diminished air entry or altered chest movement
- Decreased oxygen saturation levels

2. Tell the pediatric patient before endotracheal suctioning.

3. The first nurse (who acted to suction) arranged the suctioning equipment to set appropriate pressure of vacuum for each subjects such as:

- 80 mmHg for infant
- 100 mmHg for children age more than 1 year

After that she inserted the sterile right glove and held the suction catheter (selected an appropriate suction catheter that should not be bigger than a half of the internal diameter of the endotracheal tube) connected the finger tip to the suction catheter and another end to the wall suction.

Size of endotracheal tube (mm)	Size of suction catheter (Fr)
2.5 - 3.5	6
4.0 - 5.0	8
5.5 - 6.0	10
6.5 - 7.5	12

4. The second nurse, who gave manual positive pressure ventilation with the resuscitating bag. She connected it with oxygen supply valve for 100% oxygen and with PEEP valve device for adjust the positive end-expiratory pressure that the patient received from the mechanical ventilator. She disconnected the mechanical ventilator

from the pediatric patient's endotracheal tube and connected it with the resuscitating bag and then, pressed the resuscitating bag as equally to the peak of inspiratory pressure of mechanical ventilator by using the pressure gauge to monitor the pressure.

The sizes of resuscitating bag are as follows:

- 250-500 ml for infant (premie to 1 year)
- 500 ml for small child (2-5 years)
- 500-1,000 ml for school-age child (6-12 years)
- 1,000 ml for adolescent to adult

5. The first nurse who acted to suction, measured the depth of the suction catheter to equal with the depth of endotracheal tube plus the length of part of endoracheal tube from the pediatric patient' mouth to slip joint plus the length of suction catheter beyond the endotracheal tube 1 cm, and used tape to remark of the suction catheter length at the head of patient's bed to measured the depth of suction catheter before suctioning. Each subject was suctioned not be longer than 10 seconds (count 1 to 10).

6. The second nurse gave 5 breaths of the positive pressure ventilation manual by the resuscitating bag. Between suctioning, the nurse should observe vital signs of the pediatric patient. If they had vital signs unstable, the physician and nurse would manage it immediately.

7. Suctioned and gave 5 breaths of the positive pressure ventilation manual by the resuscitating bag again two times and assessed the lung sound again to evaluate the efficiency of suctioning.

8. The second nurse connected the mechanical ventilator to the pediatric patient's endotracheal tube.

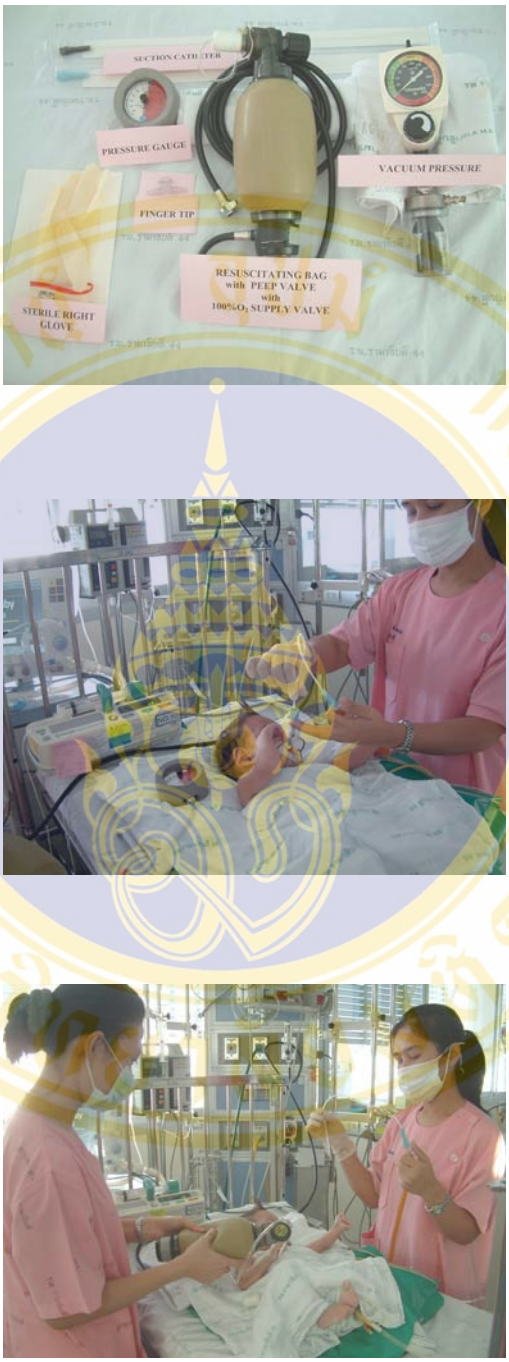

Figure 17: Suctioning procedure

APPENDIX C

Table 11: One-Sample Kolmogorov-Smirnov Test
(Oxygen Saturation of Endotracheal Suctioning without Normal Saline Instillation)

		baseline	immediately	1 mins	2 mins	3 mins	4 mins	5 mins
N		65	65	65	65	65	65	65
Normal Parameters[a,b]	Mean	98.57	94.83	95.92	97.09	97.97	98.57	98.89
	Std. Deviation	2.172	2.919	3.002	2.919	2.839	2.449	2.216
Most Extreme Differences	Absolute	.255	.265	.241	.272	.243	.280	.309
	Positive	.255	.183	.183	.211	.237	.280	.309
	Negative	-.243	-.265	-.241	-.272	-.243	-.247	-.273
Kolmogorov-Smirnov Z		2.056	2.136	1.941	2.193	1.957	2.254	2.488
Asymp. Sig. (2-tailed)		.000	.000	.001	.000	.001	.000	.000

		6 mins	7 mins	8 mins	9 mins	10 mins	15 mins
N		65	65	65	65	65	65
Normal Parameters[a,b]	Mean	98.97	98.95	99.09	99.14	99.08	99.15
	Std. Deviation	2.023	2.034	1.792	1.836	1.744	1.906
Most Extreme Differences	Absolute	.305	.304	.310	.319	.298	.329
	Positive	.305	.304	.306	.319	.298	.329
	Negative	-.275	-.263	-.310	-.301	-.282	-.314
Kolmogorov-Smirnov Z		2.461	2.447	2.501	2.576	2.405	2.649
Asymp. Sig. (2-tailed)		.000	.000	.000	.000	.000	.000

a. Test distribution is normal.

b. Calculated from data

Table 12: One-Sample Kolmogorov-Smirnov Test

(Oxygen Saturation of Endotracheal Suctioning with Normal Saline Instillation)

		baseline	immediately	1 mins	2 mins	3 mins	4 mins	5 mins
N		65	65	65	65	65	65	65
Normal Parameters[a,b]	Mean	98.62	93.89	95.05	95.83	96.85	97.69	98.38
	Std. Deviation	2.067	3.062	3.135	3.150	3.208	3.094	2.827
Most Extreme Differences	Absolute	.251	.299	.263	.211	.242	.228	.284
	Positive	.251	.174	.174	.184	.174	.228	.284
	Negative	-.244	-.299	-.263	-.211	-.242	-.231	-.231
Kolmogorov-Smirnov Z		2.027	2.408	2.123	1.704	1.952	2.288	2.288
Asymp. Sig. (2-tailed)		.001	.000	.000	.006	.001	0.002	.000

		6 mins	7 mins	8 mins	9 mins	10 mins	15 mins
N		65	65	65	65	65	65
Normal Parameters[a,b]	Mean	98.65	98.94	99.03	98.98	99.08	99.02
	Std. Deviation	2.528	2.164	1.862	1.883	1.744	1.996
Most Extreme Differences	Absolute	.296	.312	.301	.295	.313	.311
	Positive	.296	.312	.301	.295	.298	.311
	Negative	-.263	-.296	-.293	-.244	-.313	-.282
Kolmogorov-Smirnov Z		2.387	2.515	2.430	2.377	2.525	2.507
Asymp. Sig. (2-tailed)		.000	.000	.000	.000	.000	.000

a. Test distribution is normal.

b. Calculated from data

Table 13: One-Sample Kolmogorov-Smirnov Test

(End-tidal CO_2 of Endotracheal Suctioning without Normal Saline Instillation)

		baseline	immediately	1 mins	2 mins	3 mins	4 mins	5 mins
N		65	65	65	65	65	65	65
Normal Parameters[a,b]	Mean	39.23	42.09	41.38	40.69	40.18	39.65	39.52
	Std. Deviation	8.503	9.000	9.081	9.010	8.847	8.997	8.904
Most Extreme Differences	Absolute	.186	.177	.176	.174	.156	.157	.165
	Positive	0186	.177	.176	.174	.156	.157	.165
	Negative	-.108	-.100	-.096	-.095	-.094	-.107	-.112
Kolmogorov-Smirnov Z		1.503	1.423	1.419	1.407	1.254	1.267	1.333
Asymp. Sig. (2-tailed)		.022	.035	.036	.038	.086	.080	.057

		6 mins	7 mins	8 mins	9 mins	10 mins	15 mins
N		65	65	65	65	65	65
Normal Parameters[a,b]	Mean	39.38	39.31	39.37	39.22	39.28.	40.18
	Std. Deviation	8.755	8.611	8.551	8.699	8.519	8.335
Most Extreme Differences	Absolute	.189	.190	.163	.154	.159	.157
	Positive	.189	.190	.163	.154	.159	.157
	Negative	-.087	-.085	-.106	-.096	-.092	-.089
Kolmogorov-Smirnov Z		1.523	1.534	1.314	1.244	1.284	1.268
Asymp. Sig. (2-tailed)		.019	.018	.063	.090	.074	.080

a. Test distribution is normal.

b. Calculated from data

Table 14: One-Sample Kolmogorov-Smirnov Test

(End-tidal CO_2 of Endotracheal Suctioning with Normal Saline Instillation)

		baseline	immediately	1 mins	2 mins	3 mins	4 mins	5 mins
N		65	65	65	65	65	65	65
Normal Parameters[a,b]	Mean	39.25	43.06	42.28	41.57	41.02	40.38	39.98
	Std. Deviation	8.422	9.078	9.116	9.145	9.101	9.065	8.763
Most Extreme Differences	Absolute	.158	.171	.128	.156	.137	.153	.145
	Positive	.158	.171	.128	.156	.137	.153	.145
	Negative	-.105	-.098	-.105	-.113	-.099	-.089	-.090
Kolmogorov-Smirnov Z		1.272	1.375	1.028	1.254	1.109	1.236	1.173
Asymp. Sig. (2-tailed)		.079	.046	.241	.086	.171	.094	.128

		6 mins	7 mins	8 mins	9 mins	10 mins	15 mins
N		65	65	65	65	65	65
Normal Parameters[a,b]	Mean	39.71	39.65	39.63	39.66	39.82	40.65
	Std. Deviation	8.939	8.945	8.674	8.592	8.507	8.512
Most Extreme Differences	Absolute	.164	.139	.130	.145	.153	.161
	Positive	.164	.139	.130	.145	.153	.161
	Negative	-.093	-.110	-.087	-.084	-.078	-.093
Kolmogorov-Smirnov Z		1.321	1.124	1.045	1.167	1.231	1.300
Asymp. Sig. (2-tailed)		.061	.160	.225	.131	.096	.068

a. Test distribution is normal.

b. Calculated from data

Table 15: One-Sample Kolmogorov-Smirnov Test

(Heart Rate of Endotracheal Suctioning without Normal Saline Instillation)

		baseline	immediately	1 mins	2 mins	3 mins	4 mins	5 mins
N		65	65	65	65	65	65	65
Normal Parameters[a,b]	Mean	114.43	139.14	136.38	133.49	130.60	127.68	121.83
	Std. Deviation	19.422	17.090	17.189	17.295	17.284	17.505	18.210
Most Extreme Differences	Absolute	.102	.151	.130	.111	.940	.101	.117
	Positive	.067	.085	.079	.072	.082	.081	.076
	Negative	-.102	-.151	-.130	-.111	-.094	-.101	-.117
Kolmogorov-Smirnov Z		.823	1.216	1.045	.899	.757	.812	.940
Asymp. Sig. (2-tailed)		.507	.104	.224	.394	.615	.524	.339

		6 mins	7 mins	8 mins	9 mins	10 mins	15 mins
N		65	65	65	65	65	65
Normal Parameters[a,b]	Mean	121.83	119.26	116.78	115.23	114.25	113.97
	Std. Deviation	18.210	18.554	18.858	19.373	19.326	19.169
Most Extreme Differences	Absolute	.117	.122	.137	.131	.125	.116
	Positive	.076	.087	.087	.075	.080	.075
	Negative	-.117	-.122	-.137	-.131	-.125	-.116
Kolmogorov-Smirnov Z		.940	.983	1.104	1.056	1.006	.935
Asymp. Sig. (2-tailed)		.339	.289	.175	.215	.264	.346

a. Test distribution is normal.

b. Calculated from data

Table 16: One-Sample Kolmogorov-Smirnov Test

(Heart Rate of Endotracheal Suctioning with Normal Saline Instillation)

		baseline	immediately	1 mins	2 mins	3 mins	4 mins	5 mins
N		65	65	65	65	65	65	65
Normal Parameters[a,b]	Mean	114.25	141.98	139.51	137.06	133.94	131.48	128.68
	Std. Deviation	19.696	16.767	16.865	17.102	17.372	17.627	17.964
Most Extreme Differences	Absolute	.118	.125	.113	.130	.116	.104	.107
	Positive	.063	.070	.081	.080	.077	.076	.082
	Negative	-.118	-.125	-.113	-.130	-.116	-.104	-.107
Kolmogorov-Smirnov Z		.952	1.009	.908	1.044	.931	.836	.864
Asymp. Sig. (2-tailed)		.325	.260	.382	.225	.351	.487	.444

		6 mins	7 mins	8 mins	9 mins	10 mins	15 mins
N		65	65	65	65	65	65
Normal Parameters[a,b]	Mean	125.97	123.20	120.60	118.18	115.91	114.86
	Std. Deviation	18.092	18.386	18.480	18.986	19.332	19.392
Most Extreme Differences	Absolute	.120	.108	.127	.133	.107	.128
	Positive	.086	.082	.069	.080	.073	.077
	Negative	-.120	-.082	-.127	-.133	-.107	-.128
Kolmogorov-Smirnov Z		.970	.873	1.023	1.068	.862	1.028
Asymp. Sig. (2-tailed)		.303	.432	.247	.204	.447	.241

a. Test distribution is normal.

b. Calculated from data

Table 17: One-Sample Kolmogorov-Smirnov Test
(Mean Blood Pressure of Endotracheal Suctioning without Normal Saline Instillation)

		baseline	immediately	5 mins	10 mins	15 mins
N		65	65	65	65	65
Normal Parameters[a,b]	Mean	69.74	83.77	74.23	67.48	67.77
	Std. Deviation	10.237	11.032	11.015	9.199	8.961
Most Extreme Differences	Absolute	.086	.124	.097	.086	.086
	Positive	.066	.072	.086	.081	.072
	Negative	-.086	-.124	-.097	-.086	-.086
Kolmogorov-Smirnov Z		.693	1.000	.783	.694	.696
Asymp. Sig. (2-tailed)		.723	.270	.573	.721	.718

a. Test distribution is normal.
b. Calculated from data

Table 18: One-Sample Kolmogorov-Smirnov Test
(Mean Blood Pressure of Endotracheal Suctioning with Normal Saline Instillation)

		baseline	immediately	5 mins	10 mins	15 mins
N		65	65	65	65	65
Normal Parameters[a,b]	Mean	70.46	86.48	77.46	70.66	68.65
	Std. Deviation	9.882	10.770	10.476	10.175	9.176
Most Extreme Differences	Absolute	.071	.123	.106	.109	.079
	Positive	.068	.064	.097	.109	.076
	Negative	-.071	-.123	-.106	-.087	-.079
Kolmogorov-Smirnov Z		.575	.995	.855	.881	.636
Asymp. Sig. (2-tailed)		.896	.275	.458	.420	.814

a. Test distribution is normal.
b. Calculated from data

BIOGRAPHY

NAME	Miss. Jiraporn Punyoo
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