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SENSORY REGRESSION TIME FROM SUBARACHNOID BLOCK WITH HYPERBARIC 0.75% BUPIVACAINE IN THE OBESE PATIENT

George Leslie Hilton

School of Allied Health Professions
Virginia Commonwealth University

This is to certify that the thesis prepared by George Leslie Hilton entitled SENSORY REGRESSION TIME FROM SUBARACHNOID BLOCK WITH HYPERBARIC 0.75% BUPIVACAINE IN THE OBESE PATIENT has been approved by his committee as satisfactory completion of the thesis requirement for the degree of Master of Science in Nurse Anesthesia.

[redacted]
Director of Thesis

[redacted]
Committee Member

[redacted]
Committee Member

[redacted]
Department chairman

[redacted]
School Dean

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Date

SENSORY REGRESSION TIME FROM SUBARACHNOID BLOCK WITH HYPERBARIC 0.75% BUPIVACAINE IN THE OBESE PATIENT

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Nurse Anesthesia at Virginia Commonwealth University

BY

George Leslie Hilton

Bachelor of Science in Business Administration
The University of Toledo, 1974

Bachelor of Science in Nursing
The University of Toledo, 1986

Director: Charles H. Moore, MS, CRNA
Assistant Professor
Department of Nurse Anesthesia
School of Allied Health Professions

Virginia Commonwealth University
Richmond, Virginia
August, 1989

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Abstract

SENSORY REGRESSION TIME FROM SUBARACHNOID BLOCK WITH HYPERBARIC 0.75% BUPIVACAINE IN THE OBESE PATIENT

George L. Hilton, BBA, BSN

School of Allied Health Professions, Virginia Commonwealth University, 1989

Major Director: Charles H. Moore, MS, CRNA

The purpose of this study was to determine if obese patients have a different sensory regression time from subarachnoid block than non-obese patients using hyperbaric 0.75% bupivacaine. A quasi-experimental design was used. Twenty patients were separated into two groups; one group was classified as obese, and the other group was classified as non-obese. The data consisting of age, height, weight, sex, and surgical procedure were recorded preoperatively. All the patients received hyperbaric 0.75% bupivacaine via subarachnoid puncture. The levels of spinal anesthesia were recorded at the highest level achieved. The injection time was also recorded. When the surgery was completed, the patient was transferred to the recovery room and levels of sensory blockade were checked by pin-prick with an 18-gauge needle every 10 minutes until complete recovery

from the spinal anesthesia had been achieved.

The hypothesis, there will be no difference in sensory regression time from SAB with hyperbaric 0.75% bupivacaine between obese and non-obese patients, failed to be rejected. No statistically significant difference, using linear regression analysis, was found in mean regression time between groups (obese versus non-obese).

Chapter One

Introduction

Subarachnoid block (SAB) was first reported by Dr. Corning in 1885. He made his discovery while experimenting with cocaine as a local anesthetic in a dog. Subsequently, Augusta Beard in 1898 refined the SAB technique. This technique was studied in many different ways; however, there was little current research published addressing obesity and the effect it had on sensory regression time.

A prolonged block can cause many problems for the patient, such as anxiety, financial expense in terms of a longer period of time spent in the recovery room, and risk of an accident due to loss of sensation and motor control. An insufficient block requires the patient to have an additional anesthetic for the same surgical procedure, and exposes the patient to additional risks and complications. By acquiring the knowledge of sensory regression time from SAB in obese patients, it is possible to plan accordingly for obese patients in terms of preoperative teaching, preparation, type and dose of local anesthetic to use, and postoperative care. Increased knowledge from research in

this area will benefit patients in terms of safety, comfort, cost, and convenience.

Statement of Purpose

The purpose of this study was to determine if obese patients have a different sensory regression time from subarachnoid block than non-obese patients using hyperbaric, 0.75% bupivacaine.

Statement of Problem

Is the sensory regression time from SAB with hyperbaric, 0.75% bupivacaine different in the obese and non-obese patient?

Hypothesis

There is no difference in sensory regression time from SAB with hyperbaric, 0.75% bupivacaine, between obese and non-obese patients.

Variables

Dependent. The dependent variable was the time for sensory regression from SAB.

Independent. The independent variable was body weight.

Significance

Increased knowledge gained from this study will help clinicians provide a safer and more cost effective SAB for obese patients. Knowledge gained from this study will also help the clinician prepare the patient for the SAB procedure and decrease patient anxiety. Other gains include decreased cost to the patient in terms of time spent in the recovery room and decreased risk of injury to the patient due to loss of sensation and motor control.

Definition of Terms

Obese. Patients whose body weight is above their weight range according to the chart of Desirable Weights for Men and Women prepared by the Metropolitan Life Insurance Company (1980) (see Appendix B) are classified as obese.

Non-obese. Patients whose body weight is within their weight range according to the chart of Desirable Weights for Men and Women prepared by the Metropolitan Life Insurance Company (1980) (see Appendix B) are classified as obese.

Sensory regression time. Following a SAB, the time required for a patient to have complete return of a "sharp" sensation from the stimulus of an 18-gauge needle. This sensation is intact from the nipple line to the tip of the 2nd and 3rd toes bilaterally.

Subarachnoid block. Anesthesia produced by injecting hyperbaric, 0.75% bupivacaine by means of a 25-gauge spinal

needle into the subarachnoid space through a lumbar vertebral space, either at L2-3, L3-4, or L4-5.

0.75% Bupivacaine. An amide type local anesthetic commonly used to produce analgesia when injected into the subarachnoid space. The concentration used in this study contained 7.5 milligrams of bupivacaine per milliliter of solution (mg/ml).

Hyperbaric. A solution heavier than that solution into which it is injected. Dextrose is a common additive used to make solutions hyperbaric. The concentration used in this study contained 84 mg/ml.

Assumptions

1. There were no defects in the spinal anatomy of the patients.

2. The spinal anesthetic "hyperbaric 0.75% bupivacaine in 8.4% dextrose" was pure and the dose per ampule was accurate as indicated on the label.

3. The dose of local anesthetic given was presumed to be adequate to produce T4 level sensory analgesia in all patients.

4. Each subject possessed a normal blood acid-base balance.

5. Scales were uniformly accurate between patients.

Limitations

1. The methodology included a lack of randomization, with unequivalent comparison groups.

2. The individual may have performed an inconsistent assessment of sensory levels due to patient differences in the perception of sharpness.

3. More than one individual performed the assessments of the levels of anesthesia, and there may have been different judgments about where the level of anesthesia was actually located.

4. The individual could be biased in his or her judgment regarding the anesthesia level because of preconceived ideas about what effect obesity had on the recovery time from SAB.

Delimitations

1. Patients could only be studied if they agreed to participate.

2. Informed consent was required.

3. All patients were accurately weighed on the same scale prior to surgery.

4. All patients were female presenting for postpartum bilateral tubal ligations.

Conceptual Framework

Introduction. Patients who are obese or pregnant experience higher levels of analgesia following SAB when

compared to non-obese or non-pregnant patients given a similar dose of bupivacaine anesthetic (McCulloch & Littlewood, 1986). Since there are several reasons to explain these higher sensory levels, it is necessary to discuss the basic anatomy of the spinal column, production and flow of cerebrospinal fluid, blood supply to the spinal cord, local anesthetic site and mechanism of action, local anesthetic spread and duration of action, and the local anesthetic bupivacaine.

Anatomy of the vertebral column. The vertebral column consists of seven cervical, twelve thoracic, five lumbar, five sacral and usually, four coccygeal vertebrae. There are four curves in the vertebral column cervical, thoracic, lumbar, and pelvic (Bridenbaugh & Kennedy, 1980). Normally, the cervical and lumbar curves are convex anteriorly and the thoracic and pelvic curves are concave anteriorly. These curves affect the spread of local anesthetic in the subarachnoid space (Smith, 1968). The upper lumbar and lower thoracic vertebral canal is inclined anteriorly in the supine patient 8 to 12 degrees. These inclined vertebrae cause a more cephalad spread of hyperbaric analgesics to the fourth or fifth thoracic segments.

There are three main ligaments that provide the spinal column stability and flexibility. The ligaments preceding from a ventral to dorsal direction are as follows. The ligamentum flavum covers the interlaminar space. The interspinous ligament connects the spinous processes and

blends dorsally with the supraspinous ligaments. The supraspinous ligament connects the apices of the spinous processes from cervical vertebrae number seven to the sacrum (Bridenbaugh & Kennedy, 1980).

Exposure of spinal nerves to local anesthetic causes sensory blockade and analgesia to the corresponding dermatomes. There are 31 pairs of spinal nerves arranged as follows: eight cervical, twelve thoracic, five lumbar, five sacral, and one coccygeal. The spinal nerves leave the vertebral canal by passing through the intervertebral foramina. The union of ventral and dorsal roots form the spinal nerves. The ventral roots contain axons of motor neurons, and the dorsal roots contain axons of sensory neurons (Bridenbaugh & Kennedy, 1980).

Cerebrospinal fluid. Cerebrospinal fluid (CSF) is formed in the choroid plexus and is an ultrafiltrate of blood (Bridenbaugh & Kennedy, 1980). It is a clear, colorless fluid found in the spinal and cranial subarachnoid spaces and in the ventricles of the brain. It has a pH of 7.4 to 7.6 and a specific gravity of 1.003 to 1.009. The electrolyte concentrations similar to that of plasma but with a sodium and chloride ion content slightly higher and a protein content slightly lower. The total volume of CSF in the average adult ranges from 120 ml to 150 ml with 25 ml to 35 ml in the spinal subarachnoid space. The pressure of the CSF ranges from 60 millimeters of water (mm H_2O) to 80 mm H_2O. According to Bridenbaugh

and Kennedy (1980), cerebrospinal fluid flows from the fourth ventricle to the two foramen of Lushka, and under the influence of the cephalad circulation in the vertebral veins circulates upward over the surface of the brain. It also passes through the median foramen of Magendie and then proceeds downward to the spinal cord. The composition of the CSF is kept constant by osmosis alterations in posture, and arterial pulsations. Cerebrospinal fluid passes back into the bloodstream by filtration and osmosis, which takes place in the supratentorial region of the brain through the arachnoid villi and granulations.

Blood supply. There are one anterior and two posterior spinal arteries that supply blood to the spinal cord (Bridenbaugh & Kennedy, 1980). The anterior spinal artery descends in front of the anterior longitudinal sulcus of the spinal cord to the filum terminale. Many vessels branch off the anterior spinal artery to encircle the spinal cord and supply its periphery. The posterior spinal arteries send penetrating branches into the posterior white and gray matter. These branches freely connect with the anterior spinal artery.

Blood is drained from the spinal cord by two ascending vertebral veins that lie posterior and lateral to the spinal cord. These veins become engorged in the obese individual, especially when the patient is in the supine position. It is the blood flowing in the cephalad

direction in the vertebral venous system that influences the cephalad movement of CSF.

Local anesthetic site and mechanism of action. According to Howarth (1949), when a local anesthetic was injected into the subarachnoid space, it was absorbed by neural elements. It was believed the spinal nerve roots are the site of action of local anesthetics; however, the exact site of action was debatable. Howarth discovered that radioactive-isotope-labeled local anesthetics gathered around the lateral and posterior aspects of the spinal cord, as well as along the spinal nerve roots. Howarth postulated that local anesthetics act by reducing the permeability of the cell membrane to sodium ions so that there was a marked depression of the rate of depolarization such that it failed to reach threshold potential. Consequently, an action potential did not occur and neural blockade resulted.

There were three theories regarding the exact mechanism that resulted in neural blockade from the administration of local anesthetics (Strichartz, 1973). First, the receptor site theory stated that local anesthetics bind to receptors in sodium channels. Strichartz reported that the sodium channel gates must be open for local anesthetic to enter and to block ionic conduction through the nerve. Strichartz postulated that the quartenary molecules of the local anesthetics bind to open sodium channels resulting in blocked neural

conduction. Lee (1979) suggested four possible sites for action of the local anesthetics on sodium channels (a) within the pore, (b) on protein surfaces exposed to the aqueous phase, (c) at the lipid-protein-water interface, and (d) within the membrane.

Second, the membrane expansion theory stated that local anesthetics expand nerve membranes that in turn block the sodium channels and neural conduction. The nerve membrane was regarded as being essentially impermeable to cations except at special regions where holes or channels exist through which cations move during an action potential. Anesthetic agents were postulated to increase the freedom of movement of the lipid molecules, especially at the aqueous-lipid interface, with the result that some part of the membrane that is critical for conduction is in an expanded state (Ritchie, 1975).

Third, the surface charge stated that the local anesthetic agent was bound to the membrane by the lipophilic, aromatic end of the molecule with its cationic head remaining in solution. As a result, the fixed negative charges on the membrane were neutralized so that the resting potential across the membrane increased considerably. If this increase in transmembrane potential was great enough, the action potential might be insufficient to reduce the membrane potential to its threshold level and a conduction block occurred (Ritchie, 1975).

According to Wood (1982), there were three properties that had an influence on the action of local anesthetics. They included lipid solubility, protein binding, and the dissociation constant (pKa). It was the lipophylic portion of the drug molecule that penetrated the cell membrane. The more lipophylic the local anesthetic; the more potent and longer acting was the drug. Increased lipid solubility of the local anesthetic caused more extensive entry into body membranes and tissues. Anesthetic action was prolonged by increased protein binding. As the local anesthetic releases from the protein; the neural blockade continues.

The pKa is the pH at which the drug is 50% ionized and 50% un-ionized. Local anesthetics, including bupivacaine, are weak bases. Weak bases are more ionized in an acidic solution, so decreasing the pH increases the ionization of the base. The closer the pKa is to physiologic pH, the more un-ionized form of the drug is available to cross the cell membrane and to have a faster onset of action (Wood, 1982).

Local anesthetic spread. Local anesthetic spread refers to the movement of the local anesthetic after it is injected into the subarachnoid space. The spread of a local anesthetic solution, after it was injected into the CSF, has an effect on sensory regression time. The addition of a glucose solution (7.5-10%) to a local anesthetic produces a hyperbaric solution that influences

the spread of local anesthetic. Gravity influences the movement of a hyperbaric solution, when injected into the subarachnoid space. If the patient remains in a sitting position, the hyperbaric solution moves in the caudad direction. Placing the patient in a Trendelenberg position facilitates a cephalad spread of the anesthetic drug. A hyperbaric solution becomes isobaric within 10 to 15 minutes after injection into the CSF. At this point, the level of anesthesia becomes "fixed" and changes in patient position do not affect the anesthetic level (Norris, 1988).

There are patient characteristics that affect the spread of local anesthetics in CSF. As the age of the patient increases, the onset of the local anesthetic is faster and it takes longer for the anesthetic to be metabolized and cleared from the body (Veering, Burm, & Spierdijk, 1988).

Patient height also influences local anesthetic spread. According to Greene (1985), a "short" person who has a local anesthetic injected at the L3-4 interspace will have a more cephalad spread of anesthesia than a "tall" person with the same amount and type of local anesthetic. Even if the anesthetic solution spreads an equal distance in both patients, an 18 centimeter (cm) spread reaches a higher spinal segmental level in a "short" patient.

There are also height-related differences in CSF volume. The volume of CSF below the L2 interspace is greater in "tall" patients because the length of the cauda

equina is greater (Norris, 1988). Since the CSF volume at the L2-L5 interspace, where the local anesthetic is injected, is greater in "tall" patients, the anesthetic solution has greater dilution and hence less cephalad spread. The depth of the subarachnoid space (from the dura to the pia matter) is greater in "tall" patients. The increased depth means there is an increase in absolute CSF volume at any level of the cord that contributes to further dilution of the local anesthetic in the CSF (Norris, 1988).

The site of injection of local anesthetic into the subarachnoid is important to note. Above L2, the spinal cord occupies a larger portion of the subarachnoid space that subsequently decreases the amount of CSF. Therefore, less dilution of the local anesthetic occurs with greater cephalad distribution (Norris, 1988).

According to Greene (1985), the direction of the bevel of the spinal needle during injection had no effect on the spread of local anesthetics in the subarachnoid space. Greene cited studies that indicated turbulence, as a result of the rate or force of injection, did not have any clinically significant effect on local anesthetic spread in the CSF. Furthermore, Greene reported that sudden increases in CSF pressure from uterine contractions, valsalva maneuver, straining or coughing did not increase the spread of local anesthetics in the subarachnoid space.

According to Norris (1988), the dose of anesthetic affects its spread in the CSF. It is generally found that

a higher dose of local anesthetic results in a more cephalad spread of the drug in the CSF. However, the influence of concentration, volume, and baricity may override the effect of a higher dosage of local anesthetic.

Assessment of local anesthetic spread. Rocco, Raymond, Murray, Dhingra, and Freiberger (1985) demonstrated loss of sensation to touch as the best method of assessing sensation after SAB. Sensation to pin-prick and cold were also studied. Most practitioners in anesthesia today use pin-prick to assess sensory levels.

Local anesthetic duration of action. The duration of analgesia is influenced by the speed of absorption from the subarachnoid space into the bloodstream. The egress of local anesthetic agents following subarachnoid injection is primarily by vascular absorption with no hydrolysis or other form of degradation taking place in the spinal fluid (Bridenbaugh & Kennedy, 1980). Local anesthetic concentration decreased rapidly in the CSF after injection because the drug is bound to tissue and absorbed into the bloodstream. Spinal anesthesia duration is controlled by the speed that the local anesthetic is absorbed from the spinal cord, the subarachnoid space, and diffusion through the dura and the epidural space. The more absorptive surface the local anesthetic is exposed to as it spreads in the subarachnoid space, the shorter the duration of anesthesia.

According to Ritchie and Greene (1985), the lipophylicity of the local anesthetic also affects its duration. Tetracaine is highly lipid soluble and lasts 2 to 3 hours as a spinal anesthetic. Lidocaine is less lipid soluble and only lasts approximately 1 hour. Bupivacaine is highly lipid soluble and has a duration of 2.5 to 4 hours.

Bupivacaine. Bupivacaine is an amide type local anesthetic. It has a molecular weight of 288 and a pKa of 8.1. Bupivacaine has a lipid solubility partition coefficient of 28 and is 95% bound to plasma proteins. Onset of action of bupivacaine is very rapid with maximum motor blockade and maximum dermatome level achieved within 15 minutes. Bupivacaine's duration of action, when injected into the subarachnoid space, is reported by the manufacturer to be approximately 4 hours. This is considered to be an intermediate length of time as compared to other local anesthetics. The half life of bupivacaine is approximately 2.7 hours. On a potency scale of one to eight, bupivacaine has a rating of eight (Savarese & Covino, 1986).

Metabolism of bupivacaine. Amide type local anesthetics are metabolized in the liver via conjugation with glucouronic acid. The major metabolite "pipecolylxylidine" is excreted by the kidney. Only 6% of

bupivacaine is excreted unchanged in the urine (Boyes, 1975).

Obesity. When given a similar dose of local anesthetic, obese patients experience higher sensory levels following SAB when compared to normal weight patients. There are several reasons that explain the higher sensory levels. The primary reason relates to the decreased amount of CSF in obese patients. Other reasons include slower speed of absorption and decreased susceptibility at the nerve roots to local anesthetics (McCulloch & Littlewood, 1986).

According to McCulloch and Littlewood (1986), obese patients, in the supine position, have a decreased CSF capacity in their subarachnoid space. Less CSF is available to mix with the anesthetic agent; therefore, less dilution of the local anesthetic occurs. A reduced dose of local anesthetic is required to anesthetize the spinal segments. Blood volume and CSF volume have an inverse relationship with one another. Inferior vena cava compression produced by a large amount of abdominal fat causes lumbar vertebral venous engorgement that decreases the subarachnoid space capacity for CSF (Barclay, Renegar & Nelson, 1968). Occluding inferior vena cava blood flow causes a subsequent increase in blood flow through the lumbar vertebral plexus, the ascending lumbar veins, and the vertebral venous system (Robinson, 1949).

One of the causes of pressure change in CSF is a pressure change in the vertebral venous system. The vertebral venous system has partially collapsed elastic vessels that allow substantial volume increases without a pressure increase. There is an inverse relationship between vertebral venous system blood volume and CSF volume in the subarachnoid space. This explains the decreased requirement for local anesthetic in the subarachnoid space of both obese and pregnant patients (Barclay et al., 1968).

Decreased CSF volume might cause a greater spread of local anesthetic when placed in the subarachnoid space. This phenomenon was demonstrated by the use of an abdominal binder to cause abdominal compression and thereby, a decrease in the size of the subarachnoid space. Barclay et al. (1968), demonstrated that increased pressure from the binder caused increased spread of local anesthetic placed in the subarachnoid space.

There are obese patients that hypoventilate due to the large amounts of abdominal fat that restrict breathing efforts. These patients demonstrate hypercapnia and hypoxia. The hypoxic patients are slightly acidotic and have a lower physiologic pH. At the same time, an increase in cardiac output and alveolar ventilation is needed to provide for the increased metabolic needs of obese patients, resulting in an increase in both oxygen consumption and carbon dioxide (CO_2) production. It is difficult for the respiratory system to meet this increased

metabolic demand because of the excess fat. The increased chest wall and abdominal adipose tissue produce a decrease in chest wall compliance that in turn reduces lung volume. There is a decrease in functional residual capacity (FRC) and the increase in closing volume of the lungs causes perfusion to non-ventilated portions of the lungs. All of these changes result in arterial hypoxemia in obese patients (Vaughn, 1982).

Since the pKa of bupivacaine is 8.1, it is considered a weak base. Weak bases are more ionized in an acidic solution, as may be the case of the blood in obese patients. Decreasing the pH increases the ionization of the base. In obese patient, there may be more of the ionized than the un-ionized form of bupivacaine in the bloodstream. These patients will have less drug available to cross the cell membrane and hence, bupivacaine will have a slower onset of action.

Bupivacaine clearance is dependent on hepatic blood flow. Obese patients have a decreased hepatic blood flow due to changes, such as cirrhosis and fatty infiltration (Widman, 1975). Bupivacaine clearance may be prolonged in the obese patient.

Drug distributional changes alter bupivacaine elimination despite a lack of change in metabolic clearance (Abernethy, Greenblatt, Divoll, Harmatz, & Shader, 1981). The extent of drug distribution into excess body fat is related to the solubility characteristics of the drug.

Abernethy et al., discovered that antipyrine, a low lipid soluble drug, was distributed into excess body fat in small amounts. However, diazepam, a highly lipophylic compound, distributes disproportionately into excess body fat. Diazepam would have a slower clearing capacity due to its distribution into excess body fat. The relationship of this concept to bupivacaine in an obese patient is worth consideration. Bupivacaine is a highly lipid soluble drug (it has a lipid solubility partition coefficient of 28). Excess extradural fat, as in the obese patient, is readily available for bupivacaine distribution after SAB. This results in prolonged absorption into the bloodstream and prolonged clearance from the body.

Summary. It can be concluded from the above discussion that bupivacaine leaves the CSF and enters the blood stream via a concentration gradient. If the elimination half-life of bupivacaine is prolonged in the obese patient due to increased distribution into fat, there will be less of a concentration gradient of bupivacaine between CSF and blood. Bupivacaine will remain in the CSF for a longer period of time. If this theory is true, the spinal nerve roots in obese patients will be exposed to bupivacaine for a prolonged time period resulting in a prolonged sensory block. Also, obese patients have a decreased CSF volume; therefore, the spinal nerve roots are exposed to an anesthetic solution with less dilution which

will have a greater cephalad spread. This will lead to a prolonged sensory regression time from a bupivacaine SAB.

Chapter Two

Review of Literature

Bupivacaine Clearance in Obese Patients

Abernethy and Greenblatt (1984) studied lipophilic drugs, such as bupivacaine. The population consisted of 56 men and women. Thirty-one of the patients were normal body weight, and 25 were obese. They were all healthy adults who were not taking medications. None of the patients had congestive heart failure or renal failure, and they all had normal liver function.

These lipophilic drugs were shown to have marked increases in volume of distribution (Vd) and minimal change in clearance resulting in a prolonged elimination half-life and a prolonged time to reach steady-state plasma drug concentrations. The prolongation of the elimination half-life in obesity was due to the marked increase in the Vd with no significant difference in rate of drug clearance.

Obese patients have decreased hepatic blood flow due to changes, such as cirrhosis or fatty infiltration of the liver. Since hepatic blood flow is decreased in obese

patients, local anesthetic clearance will also be decreased (Abernethy & Greenblatt, 1984). It is expected that the obese group will have a prolonged sensory regression time when compared to the non-obese group.

Effect of Body Mass on the Spread of Spinal Anesthesia

Pitkanen (1987) performed a study to determine if there was a relationship between weight, height, and the spread of spinal anesthesia. The population consisted of 90 orthopedic patients, ASA I or II, having surgery on their lower extremities. The first 50 patients were anesthetized by SAB using 3 ml of 0.75% isobaric bupivacaine. The remaining 40 patients were randomly selected to receive 3 ml of hyperbaric or isobaric 0.75% bupivacaine. The patients were divided into two groups. One group consisted of patients with a normal body mass index (BMI), defined as 20.2 to 24.6 for females and 21.1 to 25.9 for males. The other group consisted of patients with a BMI greater than 30. All patients were placed in the lateral position for the SAB, and a lumbar puncture was performed at the L3-4 interspace. Three milliliters of 0.5% bupivacaine were injected and the patients were placed in the supine position. Responses to pin-prick sensation and motor blockade were recorded every 10 minutes for the first hour after injection.

Body mass index and sensory level of analgesia are of interest to this study. The subarachnoid space is

significantly different in the obese versus the non-obese patient. An individual with a high body mass index should have a higher sensory block when equivalent doses and volumes of anesthetics are injected into the subarachnoid space as compared to non-obese patients. Pitkanen's (1987) conclusions are consistent and support this theory.

There is a lack of clarity in the methodology making this study impossible to repeat based solely on the information presented in the article. It is particularly unclear why the author selected 50 patients to receive a hyperbaric solution and 40 patients to receive a hyperbaric or isobaric solution. It is also unclear as to the method used to select the 40 patients receiving the hyperbaric or isobaric solution of bupivacaine and how the patients were divided into obese and non-obese groups.

The Effect of pH on Anesthetized Nerves

Ritchie (1975) demonstrated that the pH of the solution surrounding the anesthetized nerve was crucial to the effect of the neural blockade. Non-myelinated fibers of a nerve were bathed in a long-acting local anesthetic until blockade had occurred. The anesthetic solution was then removed. No recovery of conduction occurred as long as the nerve was maintained in a neutral bathing solution. However, when the pH of the bathing solution was increased, conduction was restored. When the nerve was returned to a neutral bathing solution, conduction block again occurred.

Conduction could be restored and abolished repeatedly by switching the nerve between the alkaline and neutral bathing solutions. The conclusion was that the uncharged form was relatively inactive whereas; the charged form produced local anesthesia.

The obese patient might have a lower pH CSF when compared to the non-obese patient. This would result in more local anesthetic in the charged and active form when compared to the non-obese patient. It would be predictable that for this reason the obese patient would have a longer sensory regression time than the non-obese patient.

Ritchie's (1975) study was very clear in the methodology and would be easy to repeat. It supported the theory that the pH of the environment into which the local anesthetic was injected was crucial to the sensory blockade. It also supported the theory that it was the charged form of the local anesthetic that was active in blocking nerve conduction.

Effects of Volume of Spinal Anesthetic in the Subarachnoid Space

Sundnes, Vaagnes, Skretting, Lind, and Edstrom (1982) showed that the duration of analgesia increased with larger volumes of hyperbaric bupivacaine. All of the patients were undergoing urological surgery and were randomly assigned to receive 0.5% bupivacaine 1.5 ml, 2.0 ml, or 3.0 ml in 8.0% glucose. The results of this study showed that

the maximum spread and duration of analgesia increased with volume.

The authors did not report the relationship of dose to volume of 0.5% bupivacaine. It was conceivable that the dose of bupivacaine could have been equal in each case with the diluent added to increase the volume or CSF aspirated from the subarachnoid space immediately prior to injection to increase the volume required for their study. If this is indeed the case, their results were consistent with Norris' (1988) findings that maximum spread and duration of analgesia increases with increased volume of anesthetic in the subarachnoid space.

Inferior Vena Cava Compression

Barclay et al. (1968), demonstrated that venous compression affects the amount of CSF in the subarachnoid space. The purpose of this study was to demonstrate that compression of the inferior vena cava by an abdominal binder or by a pregnant uterus resulted in vertebral venous system engorgement that then decreased the size of the subarachnoid space and thus, the amount of CSF. Therefore, less anesthetic agent would be necessary to induce spinal anesthesia. The sample consisted of three groups. Group one consisted of 20 nonpregnant control patients of childbearing age who received a spinal anesthetic prior to hysterectomy or other gynecological surgery. Group two consisted of 15 pregnant patients at term who received a

spinal anesthetic prior to cesarean section or delivery in the late stages of labor. Group three was an experimental group of 15 patients of childbearing age whose inferior vena cava pressure was artificially increased to approximately 250 mm of water, and who then received a spinal anesthetic for a gynecological operation.

The patients in group three had a femoral catheter inserted into the inferior vena cava and connected to a transducer to monitor pressures in the vessel. Prior to the injection of an anesthetic agent, the abdomen was compressed with an inflatable rubber bladder until the inferior vena cava pressure was 250 mm of water. Each patient was placed in the lateral Sims position and a lumbar puncture was performed at the L3-4 interspace with a 19-gauge spinal needle. The projecting shaft of the needle was bent parallel to the patient's back as the patient shifted into the supine position. Four milligrams of tetracaine (1.0 ml of a hyperbaric solution) was injected as a test dose. The level of analgesia was assessed by checking for the loss of sensation to pin-prick and was recorded according to a standard dermatome chart.

The results were as follows: Group one, all of the levels of anesthesia fell below the umbilicus except for two patients; Group two, all the levels were above the umbilicus, with two being higher than T7; Group three, all the levels were higher than the umbilicus, with two being at T5. The mean level in group one was T11; in group two

it was T8; and in group three it was T7. The authors concluded there was a reciprocal relationship between venous blood volume and CSF volume that caused a decrease in CSF volume in the subarachnoid space after compression of the inferior vena cava by a pregnant uterus or an abdominal binder.

A decreased amount of CSF volume indicated there is less dilution of the local anesthetic after it is injected into the subarachnoid space. This results in a larger amount of local anesthetic being available to bathe the spinal nerve roots. The decreased CSF volume also results in a more cephalad spread of the local anesthetic. This may explain why pregnant patients during the last half of gestation and obese patients require less anesthetic agent for spinal analgesia.

Influence of Obesity on Spinal Analgesia

McCulloch and Littlewood (1986) performed a study on the influence of obesity on spinal analgesia with isobaric 0.5% bupivacaine. In this study, the height of the block in relation to obesity was the main focus. They gave 50 patients, aged 51 to 89 years, 4 ml of 0.5% bupivacaine and correlated the height of blockade and the degree of obesity. The authors concluded that increased obesity resulted in higher levels of sensory blockade. McCulloch and Littlewood speculated on the reasons for this higher sensory blockade, such as, increased vertebral blood flow,

fat deposits, and compression from abdominal fat, all impinging on the subarachnoid space.

Additional useful information could have been added to this study by correlating obesity with sensory regression time. This added information would have strengthened these authors' conclusions. In addition, there have been no studies published to date that correlate obesity with sensory regression time from subarachnoid block with isobaric or hyperbaric bupivacaine.

Chapter Three

Methodololgy

Research Design

A quasi-experimental design was used to determine if obesity affects the regression time from a SAB. The patients were separated into two groups; one group was classified as obese and the other group as non-obese. The demographic data consisted of age, height, weight, and sex. All patients had the same surgical procedure, a postpartum bilateral tubal ligation. The sensory levels were recorded in the operating room using a standard dermatome chart (see Appendix C) until the highest level was achieved. This sensory level was then recorded on the data collection form. The sensory levels were also recorded on the data collection record immediately upon arrival in the recovery room and continued until sensation was restored at the tip of the 2nd and 3rd toes bilaterally.

Population, Sample, and Setting

The population consisted of inpatients at a mid-Atlantic, university, teaching hospital. The sample consisted of female patients presenting for postpartum

tubal ligations. They were all ASA I-II classifications, without major obstetrical or medical problems. A convenience sample was selected from the operating room schedule. All the procedures were performed in an operating room of an obstetrical unit.

Data Collection

The patients were interviewed immediately prior to the procedure. All the patients were weighed, had an intravenous line started, and were preloaded with 2 liters of lactated Ringer's solution. While in the sitting position a 25-gauge spinal needle was used to perform a subarachnoid puncture either at L2-3, L3-4, or L4-5 spinal interspace. An appropriate dose, based on the patient's height and weight, of 10 to 15 mg of 0.75% bupivacaine in 8.4% dextrose was then injected. Patients were immediately placed in the Trendelenberg position. The dose of bupivacaine, the time of injection, and the highest level of sensory blockade were recorded. When the surgery was completed the patients were transferred to the recovery room. Levels of sensory blockade were checked every 10 minutes until recovery from the SAB was achieved at the tip of the patient's 2nd and 3rd toes bilaterally. Total sensory regression time was calculated from the time of injection until the previously described sensory recovery was achieved.

The patient's age, weight, height, sex, and surgical procedure were recorded on the data collection form. The time and level of sensory blockade was checked using pin-prick from an 18-gauge needle bilaterally until sensory recovery was achieved. The Dermatome Chart from Cousin's (1980) textbook, Neural Blockade, was used to record the actual level at which loss and recovery of sensation was noted (see Appendix C).

Instrumentation

Reliability and validity of tool. Each spinal nerve provides innervation to a segmental field or portion of the skin called a dermatome. By pin-pricking the dermatome areas on the patient who has received a SAB, one can assess whether or not the patient has sensation in that particular area of the skin. Lack of sensation indicates the spinal nerve innervating that area was "blocked" by the injection of the local anesthetic into the subarachnoid space.

The Dermatome Chart's (see Appendix C) validity has been determined throughout the years by its daily use in the clinical setting. A Dermatome Chart can be found in any basic anatomy book. Dermatome Charts are printed as posters, papers, and on clipboards for use clinically. Therefore, it is reasonable to conclude that the Dermatome Chart is a valid, reliable, and commonly used tool for assessment of sensory levels.

Consent

Approval for this study was obtained from the Committee for the Conduct of Human Research (CCHR). Each patient received a verbal explanation about the study and was requested to sign a form of written consent (see Appendix A).

Analysis

A regression analysis was used to build an analytical model. This model explained the relationship between the dependent and independent variables. A *p* value less than .05 was required for significance.

Chapter Four

Results

The sample consisted of 20 female patients divided into two groups (see Table 1). Ten patients were obese, weighing 137 to 203 pounds, age 22 to 37 years old, and height from 59 to 67 inches. Ten patients were non-obese, weighing 125 to 179 pounds, age 19 to 31 years old, and height from 61 to 68 inches. All 20 patients received the same operative procedure, a postpartum bilateral tubal ligation (BTL). The variables recorded were: (a) time from subarachnoid injection to sensory recovery (the dependent variable), (b) age, (c) weight, (d) height, and (e) dose (the independent variables).

The mean dose of hyperbaric 0.75% bupivacaine for the combined group was 12.25 mg with the mean dose for obese group 12.30 mg, and the mean dose for the non-obese group, 12.20 mg. The mean weight for the combined groups was 154 pounds with the mean for the obese group 178 pounds, and the mean for the non-obese group 130 pounds. Patient weight in each group was measured. The maximum and minimum

Table 1

Means, Standard Deviations, Minimum Values, and Maximum Values, for Age, Weight, Height, Dose, and Regression Time

All Groups Combined

Variable	*N*	Mean	SD	Min	Max
Time	20	155.20	16.44	125	195
Weight	20	154.35	28.00	118	203
Dose	20	12.25	1.20	10	15
Height	20	63.85	2.23	59	68
Age	20	26.20	3.83	19	33

Obese Group

Variable	*n*	Mean	SD	Min	Max
Time	10	157.40	18.77	137	195
Weight	10	178.20	18.04	142	203
Dose	10	12.20	1.39	10	15
Height	10	64.00	2.35	59	67
Age	10	27.50	3.68	22	33

Non-obese Group

Variable	*n*	Mean	SD	Min	Max
Time	10	152.80	14.36	125	179
Weight	10	130.50	8.08	118	145
Dose	10	12.30	1.05	11	15
Height	10	63.70	2.21	61	68
Age	10	24.90	3.78	19	31

Note. Number of patients (*n*), Minimum Value (Min), Maximum Value (Max), Age (years), Weight (pounds), Height (inches), Time (minutes), Dose (milligrams).

weights in the obese group were 203 and 142 pounds, and in the non-obese group 179 and 125 pounds. The time for sensory regression was also measured. The maximum and minimum time in the obese group was 195 minutes and 137 minutes, and in the non-obese group 179 minutes and 125 minutes. The mean sensory regression time for the obese group was 157.4 minutes, and for the non-obese group was 152.8 minutes. There were no significant variables at a p .05 level.

All variables in this study were treated as continuous. Regression analysis was used to build a model that explained the relationship between time and the other variables. A stepwise model building procedure was used to determine which of the independent variables were important in predicting sensory regression time. Each variable in the regression model had a level of significance calculated. Dose had the most significant p-value of the variables included in the model (p = .15). Weight, however, was the variable of interest for this study. The following model was tested:

$$Y = BO + (B1)(X1) + (B2)(X2)$$

Legend: Y = Time, X1 = Weight, X2 = Dose, BO = Average of Time, B1 = change in Y for a unit change in X1, B2 = change in Y for a unit change in X2

The coefficients of each selected independent variable were calculated. The value for BO was 79.091; the value for B1 was .094; the value for B2 was 5.026. An R-square

value was calculated to determine the amount of variability in time that can be explained by the above mode. An R-square value of .1678 was found. The hypothesis that B0 = B1 = B2 = 0 was tested. The p value of .2099 was found.

The following figures show sensory regression time from SAB and weight (see Figure 1), age (see Figure 2), height (see Figure 3), and dose of bupivacaine (see Figure 4).

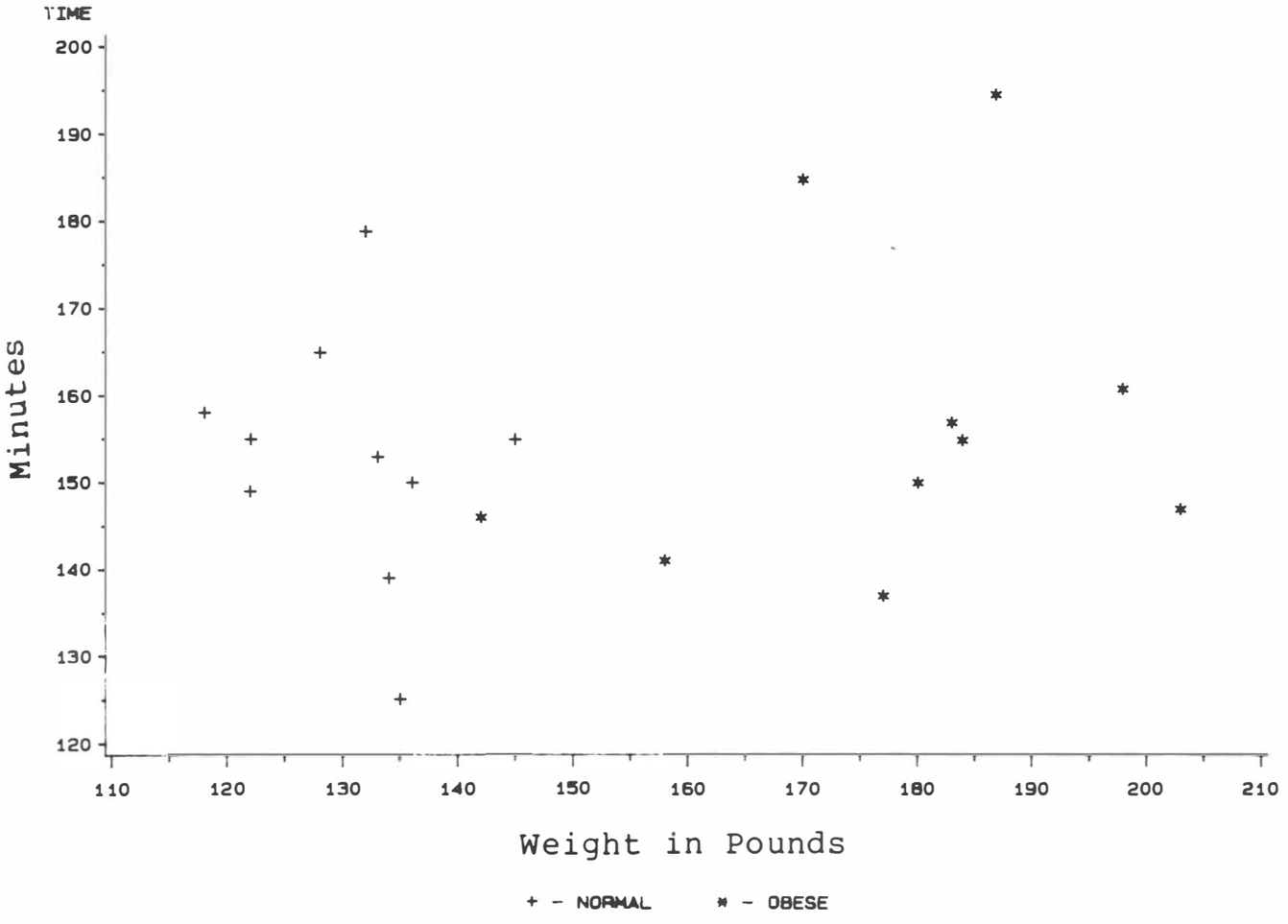

Figure 1. Time for Sensory Regression from SAB versus Weight of Patients.

Note. Time (minutes for sensory regression from SAB). Weight (pounds).

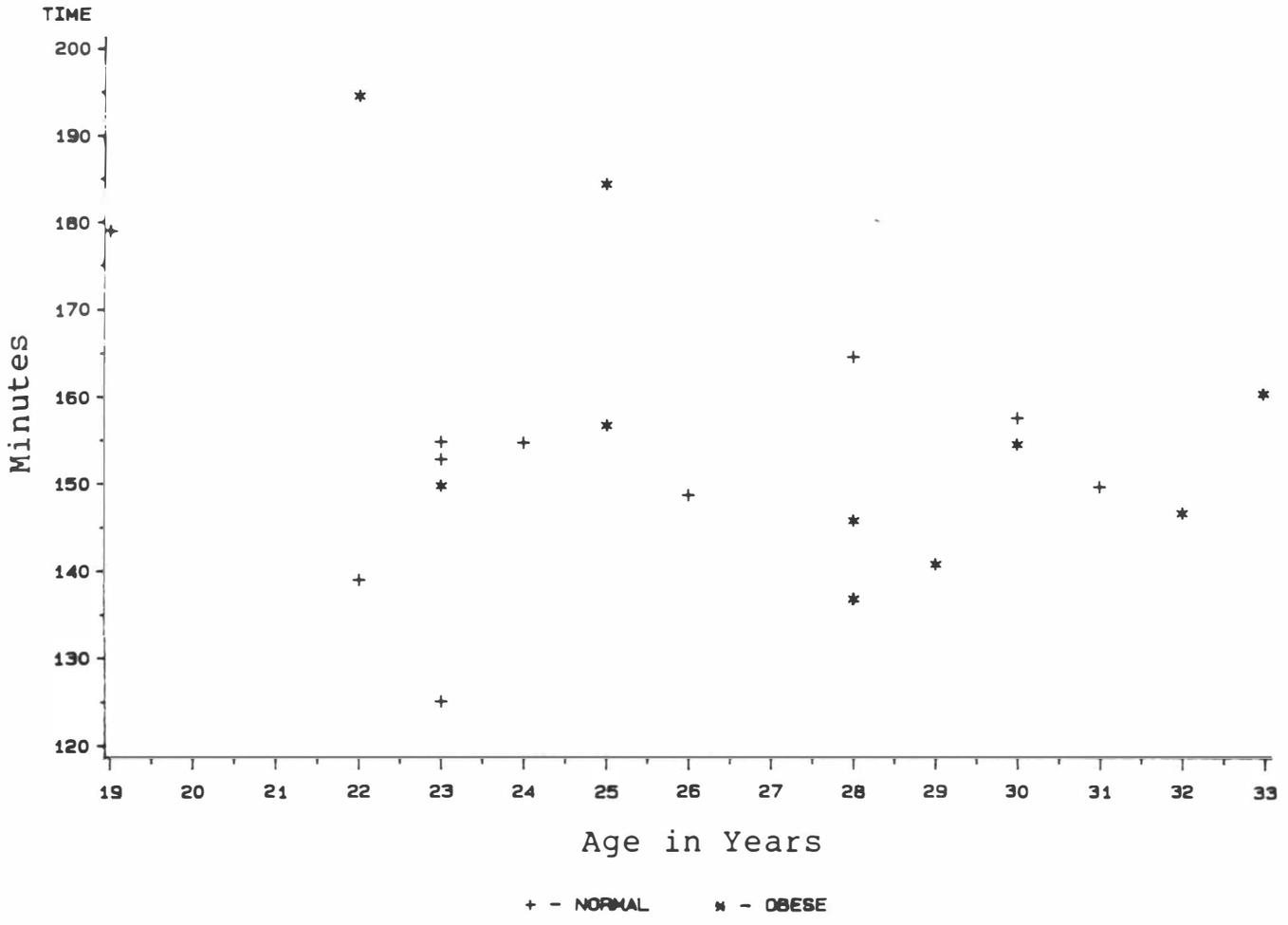

Figure 2. Time for Sensory Regression for SAB versus Age of Patients.

Note. Time (minutes for sensory regression from SAB). Age (years).

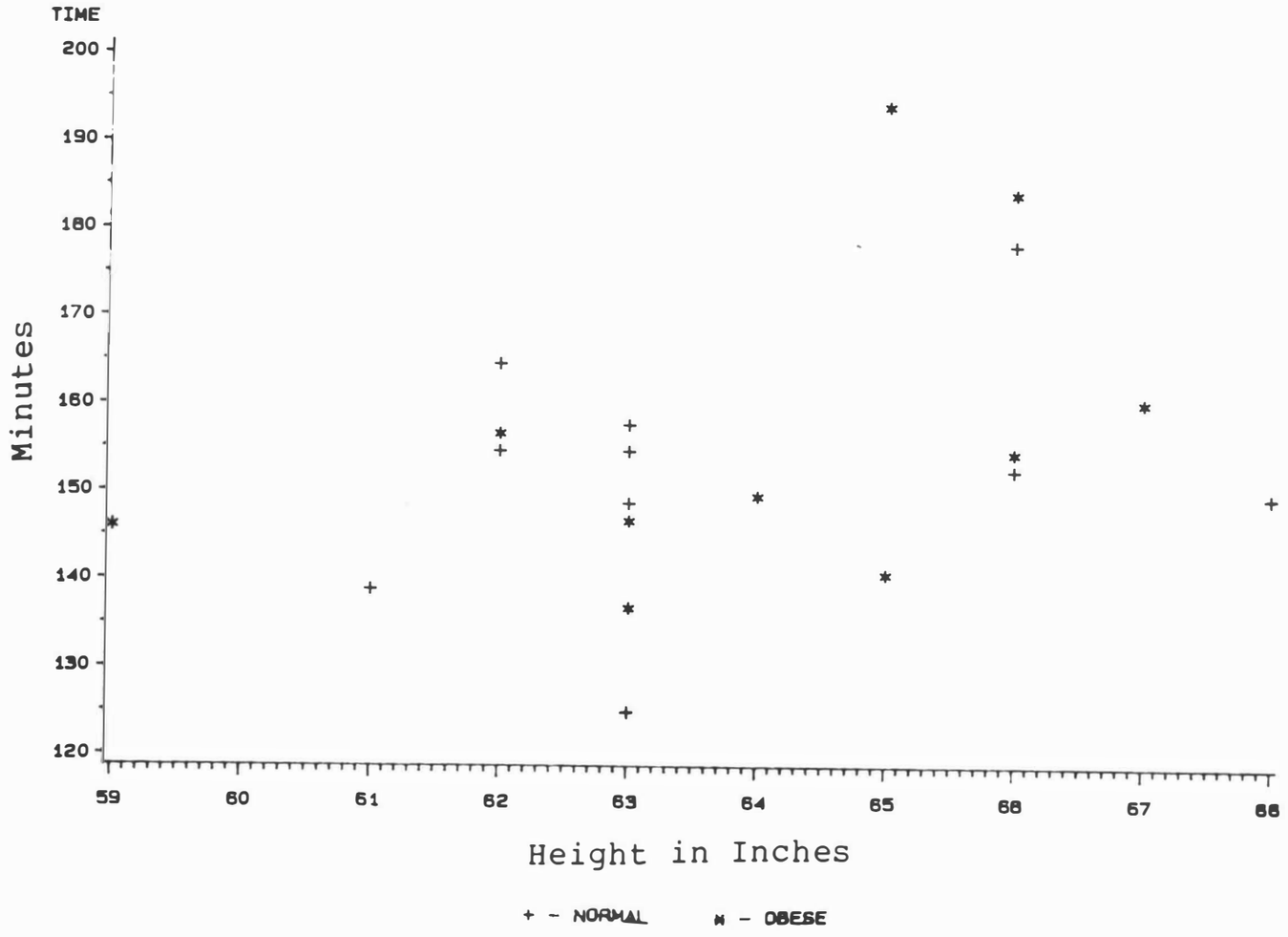

Figure 3. Time for Sensory Regression from SAB versus Height of Patients.

Note. Time (minutes for sensory regression). Height (in inches

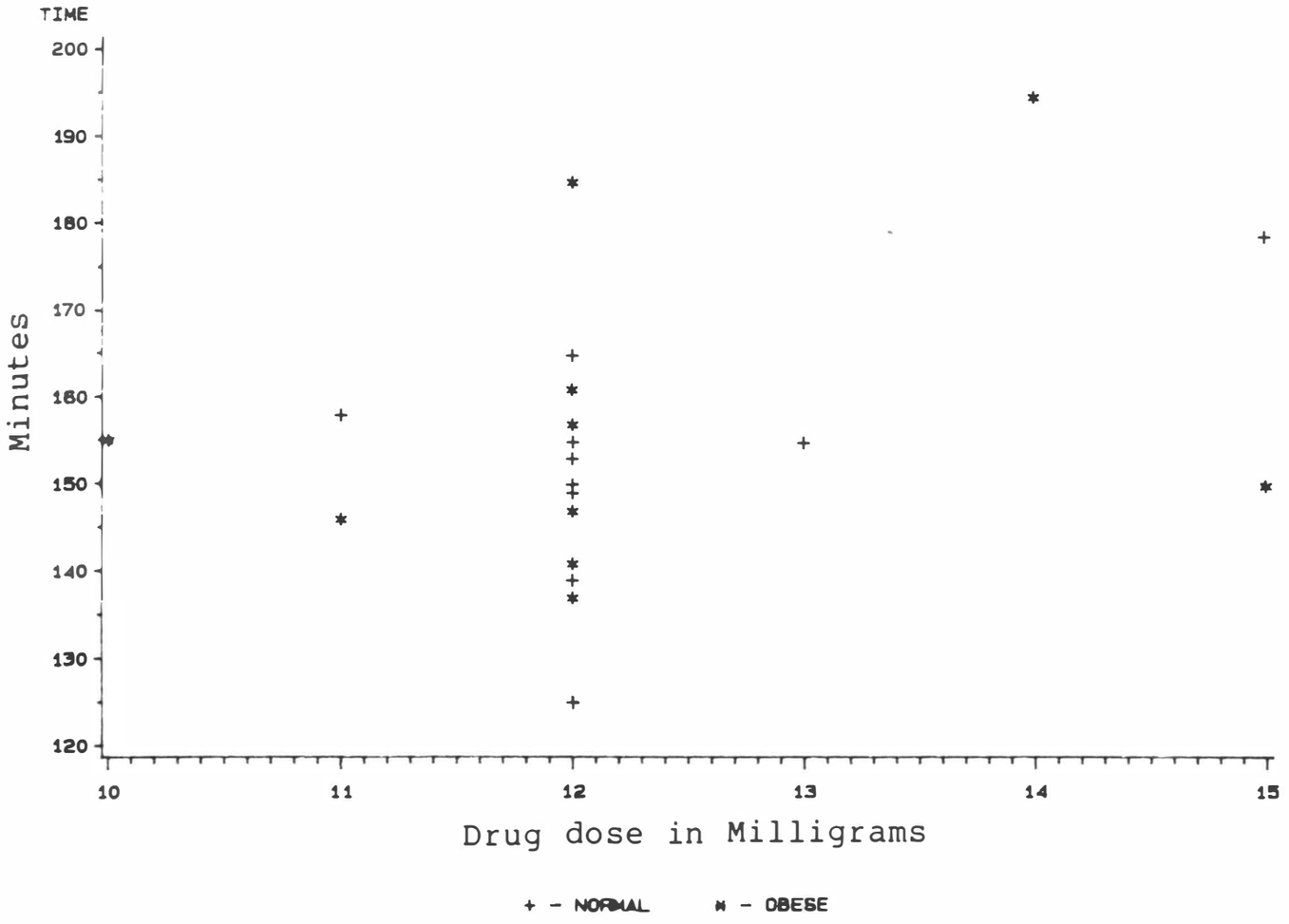

Figure 4. Time for Sensory Regression from SAB versus Dose of Bupivacaine.

Note. Time (minutes for sensory regression). Dose (milligrams of bupivacaine in 8.4% dextrose)

Chapter Five

Discussion

It is not possible to single out all of the factors that may effect sensory regression time in obese patients. It is likely that a combination of factors are involved, such as, decreased amount of CSF in the subarachnoid space, decreased pH due to some degree of hypoventilation that may result in slower speed of absorption of local anesthetic, and increased volume of blood flowing through the vertebral veins and around the subarachnoid space. In addition, the magnitude of these changes may be weight dependent.

Using a convenience sample from the operating room schedule, the patients may not have been obese enough to have a significant difference in sensory regression time when compared to the non-obese patients. Indeed, the difference in the mean weight between the obese and non-obese groups is 48 pounds.

Patients receiving larger doses of bupivacaine, such as 15 mg, had significantly longer sensory regression times. This finding was consistent with Greene's (1985) study. Statistical analysis revealed no difference in mean

regression time when height and age were studied. This was not consistent with the results of Greene's (1985) and Norris' (1988) studies. Greene reported that increasing age resulted in increased time for sensory regression from SAB. This inconsistency between Greene's results and the results in this study could be explained. Greene's study compared patients that were aged 60 or older to a younger population that resulted in a large age difference between the groups. In this study, all the patients were relatively young, less than 34 years of age, with a mean difference between the obese and non-obese groups of only 2.4 years. It could also be postulated that the failure to show a significant difference in regression time when analyzing the height of the patients was due to the small difference, 8 inches, between the tallest and shortest patient.

Difficulties with Study

It was necessary, due to occasional conflicting clinical and class obligations, for other individuals to assist in data collection. During those occasions, the recovery room nurses, after instruction, assisted by performing the sensory level physical assessments.

The mean difference in weight between the obese and non-obese groups was 48 pounds. There were several patients close to the limits of the weight range. In these

cases just a few pounds more or less would have put an obese patient in the non-obese group and vice versa.

Postpartum bilateral tubal ligations were elective procedures. On several occasions, a patient scheduled for a BTL would be delayed due to other more urgent procedures, such as a cesarean section. During the delay, the patient frequently changed her mind about proceeding with the BTL.

Conclusion

There were no variables at the $p < .05$ level. An R-square value of .17 was found, meaning there is a only small positive relationship between sensory regression time and patient weight and dose. The hypothesis, there will be no difference in sensory regression time from SAB with hyperbaric 0.75% bupivacaine, between obese and non-obese patients, failed to be rejected.

Suggestions for Future Research

The small weight difference between the obese group and the non-obese group and the small sample size may be responsible for the lack of difference in sensory regression time from SAB. Repeating this study with an increased sample size and patients selected according to a defined weight range, such as morbidly obese, may result in a more significant difference in sensory regression times. It will be easy to include, in a repeat study, a test of the pH of the CSF of all the patients to determine if a

correlation exists between pH and sensory regression time. Another factor that may have an influence on sensory regression time is gender. This study can be repeated with male patients having a different surgical procedure, such as inguinal hernia repair, to see if there is a difference in sensory regression time between obese and non-obese male patients. Several other factors that may influence regression time, such as age, type of surgical procedure, height of the patient, and height of sensory block achieved can also be studied. Although it is unlikely one factor can be considered the cause of regression time differences, the results of these studies will have clinical application.

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Appendix A

Appendix A

CONSENT FORM

George Hilton, RRNAII
Department of Nurse Anesthesia
MCV office phone 6-9808
Home phone [redacted]

1. Introduction

I am investigating the duration of the spinal anesthetic bupivacaine. You have been selected as a candidate to participate in this study because spinal anesthesia is appropriate for your surgical procedure.

2. Benefits

Bupivacaine is a long acting spinal anesthetic. It has a duration of 2.5 to 4 hours with lingering pain suppression for many more hours. Your participation in this study will help document the duration of a bupivacaine spinal anesthetic. That information may then be used to predict more precisely the duration of this anesthetic.

3. Alternative Therapy

You have been selected as a candidate for this study because spinal anesthetic has been determined to be the most appropriate form of anesthetic for your case. If you do not want spinal anesthetic, there are other forms of

anesthesia, such as general anesthesia and epidural anesthesia that I can discuss with you.

4. Risks, Inconveniences, Discomforts

The risks associated with this study are the same risks associated with spinal anesthesia. One of those risks is the risk of neurologic problems from damage to a nerve. The risk of this complication is one in 10,000. There is also a risk that you may have a headache that is caused by the spinal anesthesia procedure. There are remedies available that are 99% effective for this risk.

Assessment of the level of anesthesia is a current practice in the recovery area. For this study we will assess the level of anesthesia every 10 to 20 minutes until you have completely recovered. It is necessary to regularly assess the level of spinal anesthesia during your recovery. There will be no additional inconvenience to you as a result of participating in this study.

5. Cost of Participation

There is no cost to you above the normal fee for spinal anesthesia.

6. Pregnancy

Spinal anesthesia has a recognized use during labor and delivery. In addition, bupivacaine SAB has no known adverse effects on your baby.

7. Research Related Injury

There are no risks of injury associated with this study other than those associated with spinal anesthesia discussed above.

8. Confidentiality of Records

The information that I obtain from you during this study will remain confidential. I have taken steps to assure that your name and hospitalization number are not associated with the data collection record. Therefore, no future reference can be made to you as a result of this study.

9. Withdrawal

If you have any questions regarding the study you are encouraged to ask them now or at any time during the study. In addition, you may withdraw from the study at anytime.

"You understand that in the event of any physical and/or mental injury resulting from my participation in this research project, Virginia Commonwealth University will not offer compensation."

signed_____ Witness_____

date_____ date_____

Appendix B

Appendix B

Chart of Desirable Weight for Men and Women

DESIRABLE WEIGHTS FOR MEN
According to Height and Frame. Ages 25 - 59

HEIGHT (IN SHOES)	WEIGHT IN POUNDS (IN INDOOR CLOTHING)		
	SMALL FRAME	MEDIUM FRAME	LARGE FRAME
5' 2"	128-134	131-141	138-150
3"	130-136	133-143	140-153
4"	132-138	135-145	142-156
5"	134-140	137-148	144-160
6"	136-142	139-151	146-164
7"	138-145	142-154	149-168
8"	140-148	145-157	152-172
9"	142-151	148-160	155-176
10"	144-154	151-163	158-180
11"	146-157	154-166	161-184
6' 0"	149-160	157-170	164-188
1"	152-164	160-174	168-192
2"	155-168	164-178	172-197
3"	158-172	167-182	176-202
4"	162-176	171-187	181-207

DESIRABLE WEIGHTS FOR WOMEN
According to Height and Frame. Ages 25-59

HEIGHT (IN SHOES)	WEIGHT IN POUNDS (IN INDOOR CLOTHING)		
	SMALL FRAME	MEDIUM FRAME	LARGE FRAME
4'10"	102-111	109-121	118-131
11"	103-113	111-123	120-134
5'0"	104-115	113-126	122-137
1"	106-118	115-129	125-140
2"	108-121	118-132	128-143
3"	111-124	121-135	131-147
4"	114-127	124-138	134-151
5"	117-130	127-141	137-155
6"	120-133	130-144	140-159
7"	123-136	133-147	143-163
8"	126-139	136-150	146-167
9"	129-142	139-153	149-170
10	132-145	142-156	152-173
11	135-148	145-159	155-176
6'0"	138-151	148-162	158-179

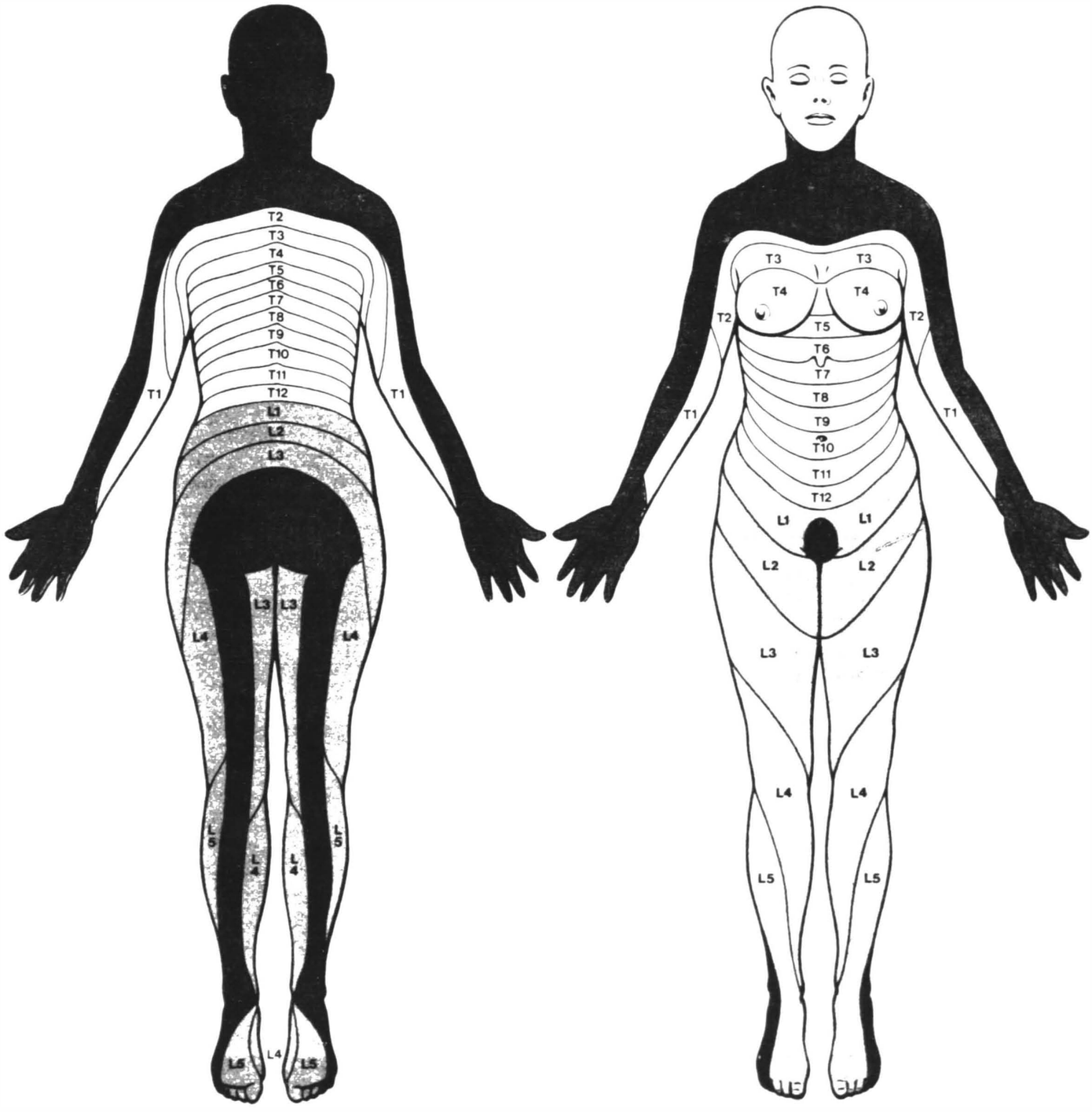
Note Prepared by the Metropolitan Life Insurance Company Derived primarily from data of the 1979 Build Study Society of aries and Association of Life Insurance Medical Directors of America. 1980

Note. From Desirable Weights for Men/Desirable Weights for Women, by Metropolitan Life Insurance Company, 1980, Society of Actuaries and Association of Life Insurance Medical Directors of America.

Appendix C

Appendix C

Dermatome Chart



Note. From Neural Blockade (p. 262) by M. J. Cousins and P. O. Bridenbaugh (Eds.), 1980, Philadelphia: J. B. Lippincott Company.

Appendix D

Appendix D

Data Sheet

Age: _____ Weight: _____ Height: _____ Sex: _____

Procedure: _____

Bupivacaine dosage: _____

Injection time of bupivacaine in Operating Room: _____

Highest sensory level achieved in operating room: _____

In recovery room sensory levels every 10 minutes:

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Time: _____ Level: _____; Time: _____ Level: _____

Total time of sensory block:

Comments: _____

_____

_____

Vita