POSTPARTUM HEMORRHAGE AND ITS RELATIONSHIP TO ANEMIA AND OXYTOCIN STIMULATION AMONG NAVAJO WOMEN

by

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ABSTRACT

The purpose of this study was to determine if relationships existed between iron deficiency anemia (diagnosed at labor onset) and postpartum hemorrhage (PPH) and between oxytocin stimulation during labor and PPH among a select group of Navajo women.

Data from the medical records of 563 Navajo women who had vaginal deliveries of full-term, single infants at the Shiprock Indian Health Service Hospital during fiscal year 1975-76 were analyzed. All the women who met the criteria were included in the study. The following variables were analyzed with respect to postpartum hemorrhage: anemia, oxytocin stimulation (including induction and augmentation), age, and parity.

The occurrence of PPH (8.7%) in this study was similar to other studies that showed a higher incidence of PPH among Navajo women compared to the general population. The occurrence of anemia was 10.2%, but this could not be compared to other research samples due to variations in both sample selection and definitions of anemia. The occurrence of oxytocin stimulation (both induction and augmentation) was 11.8%. The occurrence of induction in this study (5.9%) was far less than that reported in the
literature (13.5 - 42.1%). No comparison of augmentation occurrences could be made due to lack of information on augmentation in the literature.

When considered alone, no significant associations were found between age and PPH, between parity and PPH, or between age-and-parity (for example, 18-35 year old primigravidas) and PPH.

A highly significant association (p<0.0001) was shown between anemia and PPH. Significant associations were seen between anemia and PPH for all parity groups and for all age groups, except for the ≤17 age group. A most surprising finding was that the age-and-parity group of anemic women identified as being at highest risk for PPH were those traditionally considered to be in the "normal," low-risk, childbearing age and parity groups: 18-35 year olds with parity 2-6.

Before controlling for age and parity, no significant association was demonstrated between oxytocin stimulation and PPH. Significant associations were found between oxytocin stimulation and PPH controlling for age (p < 0.03) and for parity (p < 0.01). Only one age group (18-35) (p < 0.03) and one parity group (primigravida) (p < 0.01) showed significant associations between oxytocin stimulation and PPH. The age-and-parity group of oxytocin-stimulated women identified as being at highest risk for PPH were 18-35 year old primigravidas: 35.7% of the
women in this group had PPH.

No significant associations were found between type of oxytocin stimulation (induction and augmentation) and PPH, nor were those women who were both anemic and oxytocin-stimulated found to be at higher risk for PPH than all others in the study.
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CHAPTER 1

REVIEW OF THE LITERATURE

Introduction

United States maternal mortality rates (maternal deaths per 100,000 live births) have remarkably declined since the 1940s; the 1950 rate (83.3) was only 22% of the 1940 rate (376.0) (Barraba, 1979). Prior to this time, the leading cause of maternal death was infection (Phillips, Hulko, Vincent & Christy, 1965). As maternal mortality declined (11.2 in 1977) a shift in the leading causes occurred. Hemorrhage is now the leading cause of maternal mortality (Hellman & Pritchard, 1971) ranging from 33% (Williams & Kempers, 1969) to 66% (Wiener, 1979) of all maternal deaths. Carpenter and Bryans (1965) found hemorrhage to be the leading cause (40% of the total) of "direct obstetrical deaths" (deaths due to complications of pregnancy or childbirth and not to other medical conditions). Over 50% of these were due, more specifically, to postpartum hemorrhage (PPH). Pritchard and MacDonald (1976) indicated PPH as the most common cause of serious blood loss in obstetrics. PPH is commonly defined as loss of over 500 ml of blood during the first 24 hours.
following the birth of the baby (Oxorn & Foote, 1975). From 1959 to 1963, 29% of all maternal deaths in Mississippi were caused by PPH (Newton, 1966).

PPH as the leading cause of maternal death has a particular significance among the Navajo Indians where the problem is more frequently encountered. Slocumb and Kunitz (1977) reported an incidence of PPH of 9.6% among Navajos compared to 5.6% for non-Indians. These figures are in agreement with a reported incidence of PPH in the general population of between 5 and 6.5% (Pastore, 1936; Williams & Kempers, 1969), and in the Navajo population of 9.3% (Wilson, 1977). Vaughan (1969) reported the Indian mortality caused by hemorrhage in New Mexico to be 5.4 times the rate among whites there. When blood loss was measured (rather than estimated) both Wilson (1977) and Dyer (1978) reported the incidence of blood loss greater than 500 ml among the Navajos to exceed 60%. A possible explanation for high incidences of PPH among Navajo women is genetic predisposition. This theory is not supported by Nickerson's (1977) study which compared the rate of PPH among Navajos at Shiprock to that of primarily Caucasian women at Hill Air Force Base near Ogden, Utah. The expectation that the Navajo group would have greater blood loss at delivery was not supported by the data. Excessive blood loss was seen as "...clearly more than a function of racial differ-
ence. Other variables influencing the amount of blood lost at delivery must certainly be operant in both populations." (p. 32)

The gravity of the relatively high incidence of PPH among Navajo women lends credence to the importance of identifying factors which would aid the maternal health practitioner in predicting PPH. With this knowledge the care provider might prevent or anticipate an episode of PPH and thus, eradicate it or reduce its severity. The value of this seems obvious not only in cases of maternal deaths, but also in complications of the puerperium due to, or accentuated by PPH. Pritchard and MacDonald (1976) focused on anemia in the puerperium as a consequence of acute blood loss. This debilitating state may initiate a vicious cycle involving tissue hypoxia and increased cardiac output (Guyton, 1977), proneness to infection (Pitkin, 1977), and fatigue (Varney, 1980).

Simultaneously with making physiologic adjustments to the consequences of blood loss, the new mother must begin to make the physical and psychosocial adjustments to the new infant.

"...moderate amounts of blood loss during delivery may complicate the puerperium and debilitate the mother so that she is not in optimum condition for the stress of caring for her newborn baby." (Newton, 1966, p. 711)

Thus, with empirical knowledge of the predisposing factors, practitioners may prevent or anticipate (with
preparation to intervene) an episode of PPH and possibly reduce the stresses and adverse consequences associated with it.

Basically, the etiology of PPH can be traced to two conditions: excessive bleeding from the placental implantation site, and/or trauma to the genital tract and adjacent structures (Pritchard & MacDonald, 1976).

Upon placental separation, the veins and arteries at the intracotyledonary spaces are severed. In order to minimize blood loss, the state of "uterine hemostasis" must exist:

A firm uterus is indicative of effective uterine hemostasis. Uterine hemostasis is effected by contraction of the uterus. When contracted the entwining muscle fibers in the myometrium serve as ligatures to the open blood vessels at the placental site and bleeding is controlled naturally. (Varney, 1980, p. 278)

Adherent pieces of placenta or large blood clots may prevent effective contraction and retraction of the myometrium and thereby diminish hemostasis at the placental site, resulting in an atonic or hypotonic uterus and PPH (Pritchard & MacDonald, 1976).

Blood loss is physiologically controlled elsewhere in the reproductive tract via intrinsic vasospasm and local formation of blood clots. This mechanism is also operative at the placental site, but is far less important than myometrial contraction and retraction (Pritchard & MacDonald, 1976).

There is general agreement in the literature
about factors predisposing to PPH. These include:

1. Uterine overdistention
   A. multiple fetuses
   B. large baby
   C. polyhydramnios
   D. fibroids

2. Trauma to the reproductive tract
   A. episiotomy
   B. laceration of the genital tract
   C. operative delivery (forceps, surgery, version)
   D. ruptured uterus
   E. inverted uterus

3. Maternal history
   A. grand multiparity
   B. history of PPH
   C. history of uterine atony
   D. antepartal hemorrhage

4. Labor and Delivery
   A. vigorous/rapid/precipitate labor
   B. prolonged labor (first and second stages)
   C. dysfunctional labor
   D. oxytocin stimulation
   E. sedation and analgesia
   F. general or conduction anesthesia
   G. rapid delivery
5. Postpartum

A. uterine atony
B. mismanagement of third stage
C. premature separation of placenta
D. retained placental fragments
E. partial separation of placenta
F. placenta previa
G. placenta accreta

6. Hematologic

A. maternal clotting defects
B. anemia

Questionable

A. maternal age
B. uterine infection

(Thomas, 1962; Conn, Vant & Cartor, 1941; Beecham, 1939; Calkins, 1929; Fugo, 1969; Menon, 1968; Newton, 1966; Greenhill & Friedman, 1974; Pastore, 1936; Varney, 1980; Pritchard & Macdonald, 1976; Myers, 1975).

The most commonly cited factors are: uterine atony, mismanagement of the third stage, trauma, overdistention of the uterus, rapid labor and delivery, placental abnormalities or retention, grand multiparity, and analgesia/anesthesia. Controversy exists over the association between PPH and oxytocin stimulation, anemia, and to lesser degrees, maternal age and parity greater than one but less than six. Part of the controversy is the discrepancy between what secondary
sources consider "common knowledge" and what is supported by research described in primary sources. This issue will be addressed in the discussions of anemia, oxytocin, age, and parity, below.

Anemia and PPH

There are many anemias and hemoglobinopathies which may occur during pregnancy. Varney (1980) offered a succinct list:

1. iron deficiency anemia
2. megaloblastic anemia
3. anemia resulting from blood loss
4. anemia associated with infection
5. acquired hemolytic anemia
6. macrocytic anemia
7. microcytic anemia
8. aplastic or hypoplastic anemia
9. sickle cell anemia
10. sickle cell-hemoglobin C anemia
11. sickle cell-thalassemia anemia
12. thalassemia (hereditary microcytic anemia)

Most authors have identified iron deficiency anemia as constituting roughly 95% of all anemias of pregnancy (Varney, 1980; Greenhill & Friedman, 1974). For this reason, this discussion is restricted to iron deficiency anemia. "Anemia" is defined as "iron deficiency anemia" unless otherwise stated.
The incidence of iron deficiency anemia in pregnant women ranges from 10 to 75% (Greenhill & Friedman, 1974). Lund (1951) surveyed 4,015 pregnant women and found that half were anemic. Incidences vary with the definition of anemia. Fluctuations in red blood cell quantity at different altitudes compound the definition discrepancies since as altitude increases, circulating erythrocyte mass increases in order to increase oxygen transport (Hytten & Lind, 1975).

Anemia is usually defined as a reduction of hemoglobin and/or red blood cells per unit volume (hematocrit) below some arbitrary standard. (Lund, 1951, p. 959).

McFee (1973) maintained that, at sea level, "most authorities throughout the world use a hemoglobin level of either 10 grams or 11 grams percent (hematocrit of 30-33 percent) as the level below which anemia exists during pregnancy" (p. 154). At Shiprock, New Mexico, where the altitude is greater than 5,000 feet above sea level, the criteria for clinical diagnosis of anemia is a hematocrit less than or equal to 35% (University of Utah, College of Nursing, 1979). The arbitrary nature of standards for anemia may account for the discrepancies found in the literature regarding incidence of anemia among the Navajo. For example,

The Navajo seldom shows clinical evidence of gross nutritional deficiency. . .He has no problem of anemia. . .The absence of anemia speaks for a good intake of iron. . .(Darby, Salsbury, McGarrity, Johnson, Bridgforth, & Sandstead, 1956, p. 75).
However, when Reisinger (1972) studied Navajo nutrition, using altitude-modified National Nutrition Survey Standards for hematocrit values, 20% of the non-pregnant, non-lactating female sample between the ages of 17 and 44 were below the standard. This corresponds with Schaefer's (1977) research on serum iron and transferrin saturation indices among Navajos: among females above age 12, "unacceptable levels" were found for serum iron in 29% and for transferrin saturation in 38% of the women.

In pregnancy, normal physiological changes occur in the blood to support the synergistic metabolism of the mother, uterus, fetus, and placenta. There are substantial increases in both total blood volume and erythrocyte production:

The magnitude of the blood volume increase, which reflects primarily plasma volume, averages 50%; there is wide variation, however, from 20% to almost 100%. Red cell volume normally increases also, but only by 20-30%. Thus, physiological hemodilution is apt to drop the hemoglobin levels to less than those of the nonpregnant state. At times there is so much discrepancy between total blood and red cell volumes that low hematological values result. (McFee, 1973, p. 153)

In summary,

Thus, by dilution a "physiological anemia" ensues with development of decreased values for hemoglobin and hematocrit. These values begin to decrease at 3-5 months' gestation, reach their lowest point at 5-8 months' gestation, rise slightly at term, and return to normal at 6 weeks' post-partum. (Worthington, Vermeersch, & Williams, 1977, p. 94)
The gravid uterus has a greatly hypertrophied vascular system in comparison to its nongravid state, with a concomitant increase in blood volume demands. The blood volume increases can beneficially affect the mother and fetus. For example, both the mother and fetus are protected against the deleterious effects of impaired venous return when the mother is in the supine and erect positions. Further, the mother may also be protected against the adverse effects of blood loss associated with childbearing (Pritchard & Macdonald, 1976).

In support of this theory of a natural safeguard mechanism, Pritchard, Baldwin, Dickey and Williams (1962) believed that most of the blood added to the circulation can be lost during delivery and immediately postpartum and "still the postpartum blood volume will be stable and the hematocrit will not be markedly altered from its value prior to parturition." (p. 1280)

The differences between non-threatening "physiological anemia" due to the relative increase of blood volume over erythrocyte volume, and potentially-threatening iron deficiency anemia due to diminished iron stores are hazy, at best. As Messer (1974) noted, "with these physiologic changes in plasma, red cells, and total blood volume in mind, it is not easy to arrive at a precise hemoglobin level during pregnancy that can be called 'anemia'" (p. 164). A further
complication in assessing anemia is the fact that diminished iron stores are not reflected in a lowered hemoglobin level until the stores are exhausted, making it possible to have iron deficiency without anemia. Therefore, in "iron deficiency anemia," iron stores are absent (Messer, 1974).

Approximately two-thirds of a young woman's total body iron is stored in hemoglobin; varying lesser amounts are in the liver, spleen, and bone marrow; and even smaller amounts are in iron-containing enzymes and myoglobin (Messer, 1974). In a classic study of iron deficiency in healthy young college women, Scott and Pritchard (1967) showed (via bone marrow aspirations) that iron stores were scant to absent in two-thirds of the women. Holly and Grund (1959) concluded from their research that storage iron deposits are decreased in the majority of women at the onset of pregnancy and that storage iron is "available iron" and can be utilized to satisfy pregnancy demands. These iron demands are made by the fetus, placenta, and the expanding maternal hemoglobin mass. Nearly all the iron for these purposes is utilized during the latter half of pregnancy. Since most women have insufficient body stores of iron,

...the desired increase in maternal erythrocyte volume and hemoglobin mass will not develop unless exogenous iron is made available in adequate amounts...
Hemoglobin production in the fetus, however, probably will not be impaired, since the placenta obtains iron from the mother in amounts sufficient for the fetus to establish normal hemoglobin levels even when the mother has severe iron deficiency anemia. (Pritchard & MacDonald, 1976, p. 183)

Thus, the concept of the fetus as a "parasite" for maternal iron stores is upheld, as is the idea that the one most likely affected by the iron deficiency is the mother.

The link between anemia and PPH is one of the frequently asserted areas of "common knowledge" mentioned above. Indeed, the assumption of a cause and effect relationship by a Shiprock nurse-midwife, who used the rationale that anemia leads to PPH to motivate her clients to take their iron supplements (Darlington, 1980), was one of the initial stimuli for the authors' selection of this research topic.

A number of authors have clearly stated a cause and effect relationship: "Anemia predisposes to excessive blood loss" (Pastore, 1936, p. 92). Likewise, Beecham (1939) cited anemia as a predisposition to PPH: "Prevention of postpartum hemorrhage begins in the prenatal period, with physical conditioning of the patient, paying particular attention to her nutrition" (p. 260). Kamperman wrote, "We are also impressed by our experience that patients who are anemic are also most likely to lose blood" (Conn et
al., 1941, p. 785). More recently, Roessing (1974) noted:

The patient who goes into labor with anemia enters the race with a handicap. We may find a longer labor, weaker contractions, increased operative deliveries, fetal distress, or a uterine atony in the third stage (p. 588).

Slocumb and Kunitz (1977) claimed that anemia, malnutrition, and lack of prenatal care are potentiating factors that can result in severe hemorrhage and maternal mortality (when the patient is unable to adjust to blood loss) (p. 352). Later in the same article, the authors redefined the "effect" of anemia, calling it "inability to withstand hemorrhage" rather than hemorrhage itself. They also related this concept to native Americans:

Perhaps the most significant maternal complication of iron deficiency and other anemias is the inability to withstand hemorrhage. We have noted that effects of hemorrhage among American Indian patients are somewhat more severe than among other populations, and anemia may be a significant predisposing factor (p. 355).

Other researchers have supported this more carefully-worded "effect." "The most common serious maternal consequence of anemia throughout the world is the inability of the anemic to withstand hemorrhage" (McFee, 1973, p. 155). The decreased red blood cell volume of anemia results in reduced oxygenating capacity per volume of blood. Thus, significant blood losses are apt to reduce peripheral oxygenation and produce shock more
readily in the anemic than in the non-anemic parturient, since the anemic woman has less of a reserve to tolerate appreciable blood loss than the non-anemic woman (Myles, 1975). Pritchard and MacDonald (1976) and Pitkin (1977) agreed with this theory of anemia leading to decreased tolerance to PPH.

Only two studies demonstrating decreased blood losses among anemic patients as compared to patients without anemia were found. Calkins (1929) showed that his cases of "severe anemia" had slightly less than average blood losses, with a mean loss of 170 ml. Using Wilson's raw data (1977), the authors found that the incidence of anemia among women who hemorrhaged was 17%, but the incidence of anemia among those who did not hemorrhage was nearly twice as high at 33%. Using the chi square test (alpha of 0.05), the relationships were not statistically significant, however, presumably due to a small sample size.

In summary, most authors believe that there is an association between anemia and PPH. The controversy lies in whether anemia actually predisposes childbearing women to PPH, or whether anemia lessens their ability to physiologically cope with PPH once it has occurred.

**EBL vs MBL**

But what really constitutes postpartum hemorrhage? How is blood loss calculated? Estimation of blood loss
has been an "imperfect science" for decades. In 1929, Calkins' study of 853 vaginal deliveries found an average estimated blood loss (EBL) of 222 ml. His review of contemporary literature showed EBL ranges from 250 to 700 ml. "This element of variability is so great that we have not had, in the past, a satisfactory definition of what should be regarded as normal loss and what should be looked upon as pathologic" (Calkins, 1929, p. 528). Although others of this era suggested a limit of 500 ml, Calkins recommended a limit of 600 ml, stating that this was in agreement with what Williams proposed in 1919 (Calkins, 1929, p. 578). Pritchard and MacDonald (1976) still considered normal blood loss to be approximately 600 ml, stating that "...a blood loss somewhat in excess of 500 ml by accurate measurement is not necessarily an abnormal event for vaginal delivery" (p. 744). They rationalized using the now traditional \( \geq 500 \text{ ml} \) definition of PPH by noting that this cut-off level, though low, "may call attention to mothers who are bleeding excessively and warn the physician that dangerous hemorrhage is imminent" (p. 744). Other authors have maintained that the 500 ml level is an average blood loss and therefore is an appropriate cut-off level (Greenhill & Friedman, 1974; Pritchard et al., 1962).

In research where EBL was compared to measured blood loss (MBL) great discrepancies between the two
occurred. "In most cases the tendency is to grossly underestimate the amount of bleeding so that actual blood loss is many times greater than the recorded amount" (Fugo, 1969, p. 374). Newton, Mosey, Egli, Gifford and Hull (1961) documented a mean error of 56% when EBL was compared to MBL. In an investigation of Navajo women, Wilson (1977) showed a 17% incidence of $EBL > 500$ ml but a 66% incidence of $MBL > 500$ ml among the same patients.

In comparing the means of the measured and estimated blood loss, it is apparent that there is a tendency to underestimate blood loss by approximately one-half the actual amount measured (Wilson, 1977, p. 31).

As a result of the confusion surrounding a definition of PPH, researchers must now set their own criteria for PPH evaluation and recognize the subjective, tentative nature of their data.

**Oxytocin Stimulation and PPH**

For centuries accoucheurs have attempted to initiate and stimulate the uterine contractions of labor. As early as the mid-1500's Paré used artificial rupture of membranes to induce labor (Fields, 1968). The use of "ergot of rye" to produce uterine contractions appears in Camerarius' 1668 "Actes des Curieux de la Nature" (Huston, 1848). Bergersen (1976) suggested that it may have been used even earlier: ergot was known as an obstetric herb to midwives long before it was recognized by the medical
profession (p. 587). Huston (1848) pointed out the

pitfall of using ergot as an inducing agent:

The incessant action of the uterus under the
influence of ergot, is very unlike the inter-
mittent contractions which occur in natural
labor. This state of permanent contraction
of the organ, it appears to me, either detaches
the placenta, or so compresses it as to destroy
its function before the child is in a situation
to respire. (p. 245)

In 1920, Watson and Reed were the first to discuss
the use of pituitary extract for induction of labor
(Fields, 1968). Many of the controversies and problems
associated with the use of oxytoxic agents for induction
have their roots in the early use of this naturally-
produced hormone, which was usually contaminated with
vasopressin from the anterior pituitary. Vasopressin,
also known as antidiuretic hormone (ADH), intensified the
pituitary extract’s side effects of water intoxication
and hypertension (Bergersen, 1976).

The common route for administration of pituitary
extract at that time was intramuscular, which did not allow
for controlled infusion rates or immediate withdrawal
of the drug. Since the development of pure synthetic
oxytocin in the early 1950’s by DiVigneaud (Oxorn & Foote,
1975) and its use in intravenous-drip administration, new
standards for use and safety have been established. As
with any medical procedure the desirable state is that
the benefits of oxytocin outweigh its risks. Thus, the
controversy of “elective” versus “medically indicated”
induction or augmentation of labor has been a central issue in the development of standards concerning oxytocin use.

"Induction" means the initiation of uterine contraction via artificial means (Oxorn & Foote, 1975).

"Augmentation" means to improve, hasten or strengthen the contractions of labor once labor has spontaneously begun (Donald, 1979).

"Elective" induction means labor initiated for the convenience of the care provider, patient and/or institution without regard to medical indications for the mother and/or fetus (Donald, 1979).

"Medically-indicated" induction or augmentation means oxytocin stimulation used in those cases where the life or health of the mother and/or fetus is in greater danger if the treatment is withheld (Greenhill & Friedman, 1974).

Vorherr' (1974) listed the "medical" indications for induction of labor with oxytocin or amniotomy, including:

1. Fetal death
2. Placental insufficiency
3. Premature rupture of membranes (after 35-36th week)
4. Rhesus incompatibility
5. Preeclampsia, unresponsive or only temporarily responsive to therapy
6. Renal insufficiency or hypertension
7. Prolonged pregnancy, more than 10-14 days postterm.
8. Diabetes mellitus
9. Polyhydramnios
10. Hemorrhage caused by low-lying placenta or premature separation of the placenta (p. 310)

To these, Oxorn and Foote (1975) would add:

1. Cancer
2. History of rapid labor
3. Patient resides far from hospital
4. History of recurrent intrauterine death
5. Excessive size of fetus (p. 494)

Vorherr (1974) listed the reasons for augmentation:

1. Dysfunctional labor: prolonged latent phase caused by cervical dystocia, excessive analgesia or anesthesia, myometrial hypotonicity or incoordinate motility (desultory labor).
2. Secondary arrest of labor (cervix 6-7 cm dilated and no further progress) caused by myometrial insufficiency (maternal exhaustion; rule out fetopelvic disproportion).
3. Prolonged second stage or arrest of second stage.
4. In support of forceps, vacuum extraction or breech delivery.
5. In support of second twin.
6. Expediting placental delivery
7. Prophylaxis and therapy of atonic PPH (p. 307).

As of November, 1978, based on recommendations from
its Fertility and Maternal Health Drugs Advisory Committee, the FDA has required new labeling restrictions for injectable oxytocin. The label now states that oxytocin should be used only for induction in medically-indicated cases, not electively. It further restricts the route of administration for induction and augmentation of labor. "Intravenous drip is the only acceptable method, because it provides the most accurate control of dosage" (Kennedy, 1978, p. 30). The FDA cited the "known side effects":

1. Uterine hypertonicity
2. Uterine rupture
3. Fetal and neonatal bradycardia
4. Inadvertant premature delivery (Kennedy, 1978, p. 30). To this list, Oxorn and Foote (1975) would add:
   1. Fetal death, unexplained
   2. Prolonged labor
   3. Genital and fetal infection following a long period of ruptured membranes
   4. Failure of induction (p.495)

Greenhill and Friedman (1974) would include:
   1. Abruptio placentae
   2. Cervical laceration
   3. Anoxemia (p. 305)

Oxytocin has its hormonal effect on two specific areas of the body: The myometrium of the uterus and the smooth muscle of the mammary gland. Released from the
hypothalamus, oxytocin is stored in the posterior pituitary gland and released again in response to three specific stimuli: the unknown initiator of labor; the sensation related to suckling; and the act of coitus (Bergersen, 1976).

Oxytocin exerts itself on the myometrial cell of the uterus. By increasing the cell wall's permeability to the potassium ion, the cell's excitability threshold is reduced. Thus, large numbers of uterine cells react simultaneously to a spontaneous stimulus that originates near the insertion of the fallopian tubes (Oxorn & Foote, 1975; Zelenik, 1965). The force exerted is greater in the fundal area (Bergersen, 1976); thus, the major vector of the force is directed inferiorly, facilitating expulsion of the contents of the uterus. Also, the strength and frequency of contractions is increased, thus improving the "efficiency" of the contracting uterus (Oxorn & Foote, 1975).

The incidence of oxytocin-stimulated labors varies from institution to institution. Fields (1968) reported an incidence of induction of 13.5% of which only 15.5% were "indicated." Eleven years later, the same author reported an incidence of 18.4% induced labors, 78% of which were "elective" (Fields, Greene & Franklin, 1979). At St. Mary's Hospital in England, Brinsden and Clark (1978) reported induction rates ranging from 36.5 to 42.1% for the years 1967 to 1975.
Several sources were found, including classic obstetrical textbooks, that reported a "common knowledge" causal relationship between oxytocin stimulation and PPH (Cornell, 1941; Thomas, 1962; Fugo, 1969; Greenhill & Friedman, 1974; Pritchard & MacDonald, 1976; Varney, 1980).

Rare was the researcher who claimed that oxytocin has little or no association with PPH. Pastore (1936) claimed "no direct effect" between induction and PPH in 45 patients who had received "medical induction" though he did not define what he meant by "medical induction." Bishop (1958) reviewed 325 electively-induced patients' labors and deliveries and concluded, "We were unable to demonstrate any significant increase in the number of complications of pregnancy or delivery after elective induction of labor" (p. 1954).

Many authors made a distinction between elective inductions and medically-indicated inductions in assessing the relationships between oxytocin stimulation and PPH. The rationale was that the very factors which are indications for induction may predispose women to PPH. For example, Fields (1968) reported a PPH rate among elective inductions of 1.3% as opposed to 4.8% in medically-indicated inductions. In a later study, Fields et al. (1979) reported a PPH rate of 1.5% among elective inductions as opposed to 5.9% among indicated inductions.

Brinsden and Clark (1978) expressed the belief that
any use of oxytocin, whether elective or indicated, puts childbearing women at higher risk for PPH:

The increased incidence of PPH after induced labor is not due entirely to the fact that induced cases are more likely to be complicated. Normal oxytocic regimens make the uterus work much harder than usual for a shorter time so that when the fetus is expelled the uterus may become atonic and "exhausted" and the cervix may be damaged. (p.856)

One way to clarify this issue would be to compare incidences of PPH among electively-induced women (assumed to be low-risk) to incidences of PPH among normal spontaneous vaginal deliveries. D'Esopo, Moore, & Lenzi (1964) used this method by comparing 1,000 women whose labors were electively induced to a matched control group of 1,000 women who had spontaneous labors and deliveries. They stated their expectation and rationale:

Since cases for elective induction by definition are selected from a normal obstetrical population they should invariably show better results than in the remainder of contemporary noninduced cases unless the induction carried with it a significant risk, which it obviously does not. (p.562)

Contrary to their prediction, the incidence of PPH in the induced group (26/1,000) was more than twice that of the noninduced group (12/1,000). Other results of interest included a higher incidence of precipitate labors in the induced group as opposed to the non-induced (257/1,000 versus 152/1,000), more than twice the incidence of cervical tears in the induced group (14/1,000 versus 6/1,000) and nearly twice the incidence of "depressed" infants (Apgar scores less than six) in the induced
The increased incidence of postpartum hemorrhage in the induced group may be a corollary of short labors. It is a matter of common experience that a very short labor with an actively contracting uterus may be followed by a period of atony. Atony of the uterus was the cause of hemorrhage in all cases with the exception of three in which a laceration of the cervix was the cause. (p.564)

This was the only study of its kind found in the literature; its results were significant, warranting further research following a similar design.

**Theoretical Framework**

A. Oxytocin stimulation may predispose childbearing women to PPH by forcing the uterus to work harder for a shorter period of time than during spontaneous labor. This leads to an "exhausted myometrium," failure of myometrial contraction and retraction, and bleeding from the placental site.

B. Iron-deficiency anemia may predispose childbearing women to PPH, and probably is associated with decreased tolerance to PPH itself. Thus, the resultant hypotension, shock and postpartum morbidity are more likely to occur in the anemic than in the non-anemic parturient. These sequelae to anemia may be due to diminished tissue oxygenation secondary
to decreases in hemoglobin mass and blood volume.

C. Comprehension of these linkages between anemia and PPH and between oxytocin stimulation and PPH, is essential for careproviders and may provide rationale for them to operate in a "prevention and preparedness" mode rather than a "crisis intervention" mode.

D. Early recognition, prevention and preparedness are modes more in tune with nursing and nurse-midwifery philosophies and practices than is crisis intervention, which has traditionally been the operant model of obstetrics.

E. Employing the concepts of early recognition, prevention and preparedness places less stress on childbearing families than does employing the concept of crisis intervention. The result is the provision of more satisfying experiences for childbearing families, a primary goal of nurse-midwifery practice.

Rationale and Purpose

Due to the sudden, often unexpected onset of PPH, and its potentially grave consequences, any tool or knowledge that would enable the birth attendant to predict, prevent or minimize an episode of PPH would prove invaluable.
This (third stage) is indeed the unforgiving stage of labour, and in it there lurks more unheralded treachery than in both the other stages of labour combined. The normal case can, within a minute, become abnormal, and successful delivery can turn swiftly to disaster. (Donald, 1979, p. 748)

This study sought to offer a scientific basis of association between anemia and PPH, and between oxytocin stimulation and PPH, as an alternative to the "common knowledge" that assumes these associations exist.

By supplying careproviders with scientific rationale for the occurrence of PPH, the choice of intervention modes may be influenced. For example, if it is understood that oxytocin stimulation is associated with an increased risk of PPH, the careprovider may re-evaluate the risks and benefits of that intervention. If, having done so, stimulation is the chosen intervention, then the careprovider can be better and sooner prepared for PPH; for example, by typing and crossmatching the patient for a blood transfusion and having Methergine at hand for possible use following delivery of the placenta. Similarly, if it is understood that iron-deficiency anemia is associated with an increased risk of PPH, early detection, nutrition teaching, and iron supplementation may be stressed antepartally. If the childbearing woman is found to be anemic when she presents for delivery, an intravenous line may be started early in labor, along with the other preparations mentioned for oxytocin stimulation.
Specifically, the purpose of this study was to support or refute these null hypotheses:

There is no significant association between PPH and anemia diagnosed at the time of hospital admission for delivery among Navajo women.

There is no significant association between PPH and oxytocin stimulation given during labor among Navajo women.
CHAPTER 2

METHODOLOGY

Design

An ex post facto, descriptive and associational design was used to collect and analyze data from the Indian Health Service (IHS) Hospital of the Shiprock Service Unit. Limitations of the ex post facto design include: lack of randomization in sample selection, lack of control over data collection due to use of extant data, and absence of a control group.

Subjects

About 150,000 Navajos occupy a semi-arid canyon and plateau land area (25,000 square miles) in northeastern Arizona, northwestern New Mexico and southern Utah (DeStefano, Coulehan & Wiant, 1979). The Navajo population is a relatively young group with a median age of 16.2 years, compared to 19.4 years for other American Indians and 29.0 years for the overall U.S. population (May & Broudy, 1980). The Navajo male/female ratio, 48.2/51.8, differs little from that of the overall U.S. ratio of 48/52 (May & Broudy, 1980). The Navajo crude birth rate (total live births/year/1,000 people) was more than twice the U.S. rate (31.0
versus 14.9) for the 1975-77 averages (May & Broudy, 1980).

The Navajo are traditionally sheepherders and small-scale farmers and are widely scattered over the Reservation and small border towns. Each "camp" consists of an extended family which is matriarchal in nature. "Anglo" schools, hospitals, and government offices have formed the nucleus of small towns on the Reservation (DeStefano et al, 1979). Most Navajos live in one-room hogans and one or two-room frame houses, only 59% of which have "modern provisions for sanitation" (May & Broudy, 1980, p. 35). According to the 1970 census, the annual per capita income of the Navajo was $758 (May & Broudy, 1980). A study of Navajo attending the Shiprock Maternal-Child Health Clinic showed the average family to be 4.5 members and the average annual family income to be $5,736 (Sevcovic & Freeland, 1976). An earlier study of Maternal-Child Health clients at Shiprock showed that (of those answering questions on income) 68% had annual family incomes of less than $6,000, 27% had $6,000 - $15,000, and 5% had $15,000 or more. Of those responding to questions on educational level, 16% had received a kindergarten to sixth grade education, 12% seventh to ninth grade, 56% tenth to twelfth, and 16% had some college or vocational training (Freeland, 1975).
The health care system for the Navajo consists of:
Navajo Area Indian Health Service (NAIHS) facilities
(15 full-time health centers, 7 school health centers,
20 part-time health stations, and 215 health locations
open several times a month); a far smaller number of
private health care facilities (3 hospitals and 1
clinic); and the Shiprock Maternal Child Health Project,
which is collaboratively operated by the Navajo Health
Authority, IHS and the University of Utah (Sevcovic,
1980). The NAIHS facilities are free to all Navajo.
The principle focus of IHS has been on acute, emergency
and surgical treatment. The staff/patient ratio in
IHS hospitals is approximately 1.4/1, whereas "mini­
mally acceptable nursing care requires a 3.2/1 ratio"
(May & Broudy, p. 37).

The sample for the study was obtained from the
intrapartum medical records of Navajo women who utilize
the 75-bed Shiprock Indian Hospital, an institution
which serves approximately 25,000 people. Sevcovic
(1980) has stated that the Shiprock Navajo clientele is
representative of all reservation Navajos on the vari­
ables of age, family income, and educational level,
but is not representative of all reservation Navajos
on the variable of parity (the Shiprock clientele's
average parity is lower). Thus, generalization to
the entire Navajo reservation population may be some­
what limited.
Operational Definitions

1. "PPH" - "P.P. Hemorrhage" as recorded on the IHS - MCH Project Intrapartum Care Form (see Appendix A) under "Problems Consulted for:" "yes" or "no" (PPH was present if marked at all).

2. "Oxytocin Stimulation"
   A. "Induced" -- recording of "induced" under "Labor Onset" on the Intrapartum Care Form.


4. "Parity" -- number of full-term and premature infants a woman has previously delivered and the current pregnancy as recorded under "Parity" on the Intrapartum Care Form: divided into three groups: "prima" (primigravida), "2 to 6" and "greater than 6".

5. "Age" -- age as recorded on the Intrapartum Care Form divided into three groups: "less than or equal to 17", "18 to 35", and "greater than 35".
Procedure

A letter of approval for permission to perform this study was requested by the authors per "Criteria for Collecting Thesis Data for Shiprock MCH Project." Approval was granted by the Project Professional Education Coordinator (See Appendix B).

The proposal was accepted by both the Supervisory Committee of the researchers and the Human Subjects Committee of the University of Utah.

Data Analysis

Data collected via the optical scanner data collection sheets (Intrapartum Care Forms) at the MCH Project, Shiprock Service Unit, for the fiscal year, 1975-76, was analyzed. This time frame was selected for a number of reasons: great care was taken to obtain and code accurate data by both the research assistant and care provider during the intrapartum and early postpartum periods, statistical clerks reviewed the sheets for completeness, consistency and accuracy, and compared them to medical records; no revisions of the intrapartum data collection sheet was made during this time; careproviders (1 physician and 7 nurse-midwives) were consistent; and the validity of the data used from this period has been documented (Taylor, 1980). The only records used were those of Navajo women who had, after 36 weeks gestation, vaginal
deliveries of single infants. Variables to further describe the sample included age, parity, and marital status. Socioeconomic variables were not available from the data sheets.

Analysis was directed at determining a description of the sample by the use of contingency tables of all variables of interest. These contingency tables were analyzed by the raw Chi Square test and/or the Cochran-Mantel-Haenszel Q-statistic which tested the degree of association of variables of interest.

Frequencies and statistical test results were found for the following tables:

1. Anemia vs. PPH
2. Oxytocin stimulation vs. PPH
3. Type of oxytocin stimulation (induction/augmentation) vs. PPH.
4. Anemia and oxytocin stimulation vs. PPH.
5. Above tables arranged according to age groupings (less than or equal to 17, 18 to 35, greater than 35).
6. Above tables arranged according to parity groupings (primigravida, parity 2 to 6, parity greater than 6).
7. Above tables arranged according to age and parity groupings.
The alpha level of significance for this study was set at 0.05.

Limitations

In addition to the limitations already mentioned regarding ex post facto research designs, the following were acknowledged:

1. The degree of anemia was unknown.
2. The duration of anemia was unknown.
3. The amount of oxytocin used was unknown.
4. The length and pattern of oxytocin stimulation was unknown.
5. Blood was estimated, not measured.
6. Nominal, dichotomous nature of the variables restricted data analysis to chi square tests and only degree of association could be shown.

In an informed survey of the practicing nurse-midwives at the Shiprock MCH Project by Darlington (1980) there was a general consensus that EBLs were underestimated. By way of compensation, they would often record EBL as though it were a dichotomous, rather than continuous variable; i.e. "PPH" would be recorded as some number greater than 500 ml and "not PPH" as less than 500 ml. It is the authors' assumption that whenever "PPH" was coded on the Intrapartum Care Form's "Problems Consulted for" list, it truly was a PPH.
CHAPTER 3

RESULTS

Description of the Sample

Five-hundred-sixty-three Navajo women were included in this study, the purpose of which was to assess the relationships between iron-deficiency anemia and post-partum hemorrhage (PPH) and between oxytocin use during labor and PPH.

The sample included all Navajo women who, after at least 36 weeks' gestation, had vaginal deliveries of single infants at the Shiprock IHS Hospital during fiscal year 1975-76. Since all women who met these selection criteria were included (rather than a sampling taken from those who met the criteria), the "sample" is equivalent to the universal population. This interpretation eliminates the chances of committing both a Type I error (rejecting a true null hypothesis) and a Type II error (accepting a false null hypothesis), but also restricts generalizations to those women who met the criteria.

Of the total sample, 434 (77.1%) were married; 2 (0.4%) were widowed; 123 (21.8%) were single; and 4 (0.7%) were divorced. Data regarding education and income were not available. These variables are discussed
in Chapter 2.

The distribution of age and parity are shown in Table 1. As expected, a significant association \( p < 0.0001 \) was demonstrated between age and parity.

**Description of Variables**

**Postpartum Hemorrhage**

The occurrence of PPH among the women in this study appears in Table 2.

**Anemia**

The occurrence of anemia in the study population is shown in Table 3.

**Oxytocin Stimulation**

The occurrence of oxytocin stimulation among the women in this study appears in Table 4. Table 4 also illustrates a further categorization of the "stimulated" group into "augmentation" and "induction" groups.

**Results of Hypotheses Testing**

The Chi-square test was computed to determine whether or not significant associations existed between PPH and: age, parity, anemia, oxytocin stimulation, and type of oxytocin stimulation (induction and augmentation). The Cochrane-Mantel-Haenszel (CMH) test was computed when possible to determine whether or not age and parity operated as confounding factors affecting the outcome of
Table 1
Distribution of Sample
by Age and Parity

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>Parity</th>
<th>Frequency</th>
<th>% of Age Groups</th>
<th>% of Total Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 17</td>
<td>Primigravidas</td>
<td>40</td>
<td>78.4%</td>
<td>7.1%</td>
</tr>
<tr>
<td></td>
<td>2 to 6</td>
<td>10</td>
<td>19.6</td>
<td>1.8</td>
</tr>
<tr>
<td>&gt; 6</td>
<td></td>
<td>1</td>
<td>2.0</td>
<td>0.2</td>
</tr>
<tr>
<td>&lt; 17 Group Totals</td>
<td></td>
<td>51</td>
<td>100.0%</td>
<td>9.1%</td>
</tr>
<tr>
<td>18-35</td>
<td>Primigravidas</td>
<td>133</td>
<td>27.8%</td>
<td>23.5%</td>
</tr>
<tr>
<td></td>
<td>2 to 6</td>
<td>315</td>
<td>65.7</td>
<td>56.0</td>
</tr>
<tr>
<td>&gt; 6</td>
<td></td>
<td>31</td>
<td>6.5</td>
<td>5.5</td>
</tr>
<tr>
<td>18-35 Group Totals</td>
<td></td>
<td>479</td>
<td>100.0%</td>
<td>85.0%</td>
</tr>
<tr>
<td>&gt;35</td>
<td>Primigravidas</td>
<td>0</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
<tr>
<td></td>
<td>2 to 6</td>
<td>8</td>
<td>24.2</td>
<td>1.4</td>
</tr>
<tr>
<td>&gt; 6</td>
<td></td>
<td>25</td>
<td>75.8</td>
<td>4.4</td>
</tr>
<tr>
<td>&gt;35 Group Totals</td>
<td></td>
<td>33</td>
<td>100.0%</td>
<td>5.9%</td>
</tr>
<tr>
<td>SAMPLE TOTALS</td>
<td></td>
<td>563</td>
<td>100.0%</td>
<td></td>
</tr>
</tbody>
</table>
Table 2
Occurrence of PPH

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPH</td>
<td>49</td>
<td>8.7%</td>
</tr>
<tr>
<td>No PPH</td>
<td>514</td>
<td>91.3%</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>563</strong></td>
<td><strong>100.0%</strong></td>
</tr>
</tbody>
</table>

Table 3
Occurrence of Anemia

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>57</td>
<td>10.1%</td>
</tr>
<tr>
<td>No Anemia</td>
<td>506</td>
<td>89.9%</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>563</strong></td>
<td><strong>100.0%</strong></td>
</tr>
</tbody>
</table>

Table 4
Occurrence of Oxytocin Stimulation

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulation (Induction)</td>
<td>33</td>
<td>5.9%</td>
</tr>
<tr>
<td>Stimulation (Augmentation)</td>
<td>33</td>
<td>5.9</td>
</tr>
<tr>
<td>No Stimulation</td>
<td>497</td>
<td>88.2%</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>563</strong></td>
<td><strong>100.0%</strong></td>
</tr>
</tbody>
</table>
associations between PPH and: anemia, oxytocin stimulation, and type of oxytocin stimulation. Using the CMH test enables a determination of whether or not an association between two variables (such as anemia and PPH) exists when the influence of a third "confounding" variable (for example, age) is removed. This allows an assessment of the confounding variable's effect on the association as well as of the strength of the association in the absence of the variable's influence.

The first null hypothesis was:

There is no significant association between PPH and anemia diagnosed at the time of hospital admission for delivery among Navajo women.

A highly significant association (p < 0.0001) was found between anemia and PPH (See Table 5). The null hypothesis was therefore rejected as false.

The second null hypothesis was:

There is no significant association between PPH and oxytocin stimulation given during labor among Navajo women.

No significant association (p < 0.131) was found between oxytocin stimulation and PPH (See Table 5), and the null hypothesis was therefore accepted.
Table 5

Hypotheses Test Results

<table>
<thead>
<tr>
<th></th>
<th>$x^2$</th>
<th>df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia vs. PPH</td>
<td>41.77</td>
<td>1</td>
<td>0.0001</td>
</tr>
<tr>
<td>Oxytocin Stimulation vs. PPH</td>
<td>2.29</td>
<td>1</td>
<td>0.131</td>
</tr>
</tbody>
</table>

Other Findings

Influences of Age and Parity on PPH

No significant association between age and PPH was found; a nearly significant association ($p < 0.08$) was noted between parity and PPH. When compared with the other two parity groups, the incidence of PPH was highest among primigravidas (12.7% of primigravidas hemorrhaged, compared to 6.9% for the 2-6 parity group, and 7.0% for the greater than 6 parity group). When parity groups were rearranged to comprise two groups, primigravidas and all multigravidas (parities 2-6 and > 6 combined), a significant association ($p < 0.05$) between these two parity groups and PPH was seen. The occurrence of PPH among primigravidas in the 18-35 age group (15.0%) was three times that of primigravidas in the presumed higher risk
(≤ 17) age group (5.0%).

When the effect of both age and parity on PPH was considered (age vs. PPH controlling for the original three parity groups), no significant associations resulted. However, among primigravidas, a nearly significant association (p < 0.10) was seen between two age groups (≤ 17 and 18-35) and PPH (no women older than 35 were in the primigravida group). Among primigravidas, 5.0% of the ≤ 17 age group had PPH, compared with 15.0% in the 18-35 age group.

**Influence of Anemia on PPH**

Table 6 summarizes the significant and nearly significant associations found between anemia and PPH. Anemia and PPH were significantly associated (p < 0.0001). When controlling for age, significant associations between anemia and PPH were found in the 18-35 age group (p < 0.0001) and in the > 35 age group (p < 0.04). The association between anemia and PPH remained when the effect of age was removed by the CMH test. Age had an additive effect on the anemia-PPH association, as can be seen in the numerical difference between the Chi-square value of 41.77 for anemia vs. PPH and the CMH summary Chi-square value of 30.21 for anemia vs. PPH controlling for age.

When controlling for parity, a significant association between anemia and PPH was found in all parity groups:
Table 6
Anemia and PPH*

<table>
<thead>
<tr>
<th>Control Variables</th>
<th>$X^2$</th>
<th>df</th>
<th>p-value</th>
<th>% of women in group who had PPH</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANEMIA VS. PPH</td>
<td>41.77</td>
<td>1</td>
<td>&lt;0.0001</td>
<td>31.6% (all ages, all parities)</td>
</tr>
<tr>
<td>by Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-35</td>
<td>30.21**</td>
<td>1</td>
<td>&lt;0.0001</td>
<td>31.6 (all ages)</td>
</tr>
<tr>
<td>&gt; 35</td>
<td>4.31</td>
<td>1</td>
<td>&lt;0.04</td>
<td>33.3</td>
</tr>
<tr>
<td>by Parity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primigravida</td>
<td>38.22**</td>
<td>1</td>
<td>&lt;0.0001</td>
<td>31.6% (all parities)</td>
</tr>
<tr>
<td>2 to 6</td>
<td>11.64</td>
<td>1</td>
<td>&lt;0.0006</td>
<td>34.8</td>
</tr>
<tr>
<td>&gt; 6</td>
<td>21.13</td>
<td>1</td>
<td>&lt;0.0001</td>
<td>27.6</td>
</tr>
</tbody>
</table>
| Note: *Only significant (p < 0.05) and nearly significant (0.05 < p < 0.10) associations are shown
** CMH $X^2$ used, all others are Chi-square
primigravidas ($p < 0.001$), parity 2-6 ($p < 0.0001$), and parity $>6$ ($p < 0.003$). Parity also had an additive effect on the anemia-PPH association, though its contribution was not as strong as that of age. Parity had its strongest effect in the 2-6 parity group (Chi-square $= 21.13$ vs. $11.64$ for primigravidas and $9.14$ for the $>6$ parity group).

By controlling for both age and parity, nine possible age-and-parity groups were created, but due to small sample sizes, some comparisons could not be made. Three of the nine age-and-parity groups demonstrated significant associations between anemia and PPH: 18-35 year old primigravidas ($p < 0.0008$); 18-35 year olds with parity 2-6 ($p < 0.0001$); and 18-35 year olds with parity $>6$ ($p < 0.01$). A nearly significant association between anemia and PPH ($p < 0.085$) was seen among the women older than 35 with parity $>6$.

Thus, among anemic women, those at highest risk for PPH were those in the normal childbearing years and the most common parity group: 18-35 year olds of parity 2-6. This was confirmed by observing that this age-and-parity group had the highest Chi-square value ($20.87$) of all age-and-parity groups in which a significant association between anemia and PPH was found.

Influence of Oxytocin Stimulation on PPH

Table 7 summarizes the significant associations found...
Table 7
Oxytocin Stimulation and PPH*

<table>
<thead>
<tr>
<th>Control Variables</th>
<th>$X^2$</th>
<th>df</th>
<th>p-value</th>
<th>% of stimulated women in group who had PPH</th>
</tr>
</thead>
<tbody>
<tr>
<td>STIMULATION VS. PPH</td>
<td>2.29</td>
<td>(NOT SIGNIFICANT)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>by Age</td>
<td>3.31**</td>
<td>1</td>
<td>$&lt; 0.03$</td>
<td>13.6% (all ages)</td>
</tr>
<tr>
<td>18-35</td>
<td>3.84</td>
<td>1</td>
<td>$&lt; 0.05$</td>
<td>16.4</td>
</tr>
<tr>
<td>by Parity</td>
<td>7.64**</td>
<td>1</td>
<td>$&lt; 0.01$</td>
<td>13.6% (all parities)</td>
</tr>
<tr>
<td>Primigravidas</td>
<td>4.73</td>
<td>1</td>
<td>$&lt; 0.03$</td>
<td>29.4</td>
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<tr>
<td>by Age-and-Parity (CANNOT COMPUTE CMH)</td>
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<tr>
<td>18-35 &amp; primigravida</td>
<td>5.24</td>
<td>1</td>
<td>$&lt; 0.02$</td>
<td>35.7</td>
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Note. *Only significant ($p < 0.05$) associations are shown. Cannot compute age and parity.

**CMH $X^2$ used, all others are chil-square
between oxytocin stimulation and PPH. No significant association was found between oxytocin stimulation and PPH in the overall sample. However, when controlling for age, the group aged 18-35 had a significant association between oxytocin stimulation and PPH ($p < 0.05$). When controlling for parity, a significant association between oxytocin stimulation and PPH ($p < 0.03$) was found in the primigravida group. The identification of 18-35 year old primigravidas as the group of oxytocin-stimulated women at highest risk for PPH was confirmed by the significant association ($p < 0.02$) between oxytocin stimulation and PPH in this age-and-parity group.

Oxytocin stimulation was subdivided into two groups, induction and augmentation. No significant association was demonstrated between the type of oxytocin stimulation and PPH. Nor, when controlling for age, parity, or age-and-parity, was a significant association shown between type of oxytocin stimulation and PPH.

Influence of Oxytocin Stimulation and Anemia on PPH

The combined effects of anemia and oxytocin stimulation on PPH were assessed by comparing women who had both anemia and oxytocin stimulation to all other women in the sample. No significant association was found between anemia-and-oxytocin and PPH, nor was significance demonstrated when controlling for age, parity, or age-and-parity in this group.
CHAPTER 4

DISCUSSION

The relationships between anemia and postpartum hemorrhage (PPH), and between oxytocin stimulation and PPH were investigated among a select group of Navajo women. The goal was to question the "common knowledge" assumptions of associations between anemia, oxytocin, and PPH and to offer alternatives based on scientific inquiry.

Several studies were cited in the review of the literature that showed the incidence of PPH among Navajo women to be significantly higher than that of the general population. Assuming that genetic make-up is not a predisposing factor (Nickerson, 1979), the researchers reviewed the literature for other predisposers to PPH. Anemia, oxytocin stimulation, age, and parity were identified and chosen as variables.

Five hundred and sixty-three Navajo women were included in the sample, all of whom had, after at least 36 weeks' gestation, a vaginal delivery of a single infant at the Shiprock IHS Hospital during the fiscal year 1975-76. A random sample was not taken; rather all women who met the selection
criteria for that year were included.

The occurrence of PPH found (8.7%) is comparable to that which Slocumb and Kunitz (1977) found in their Navajo sample (9.6%). These figures show that PPH is nearly 50% more common among Navajos than in the general population, the PPH incidence of which has been reported at between 5 and 6.5% (Pastore, 1936; Williams & Kempers, 1969).

Influences of Age and Parity on PPH

Age and parity are two variables assumed to contribute to PPH. Women at both ends of the childbearing age range are considered to be at higher risk for both poor nutrition (for example, anemia) and poor pregnancy outcome (such as PPH) (Worthington et al., 1977). Women of high parity are thought to be at increased risk of PPH from uterine atony (Pritchard & MacDonald, 1976). However, in this sample a significant association between age and PPH was not found, and only a nearly significant ($p < 0.08$) association between parity and PPH was found. When other variables (such as anemia and oxytocin stimulation) were not considered, age was not associated with PPH, and primigravidas were at higher risk than multigravidas for PPH. Both findings are contrary to accepted tenets of obstetrics. Possible explanations are that the age and parity risk factors applicable to the general population are not operant among Navajo
women; or that other risk factors (such as anemia and oxytocin stimulation) must be present for age and/or parity to impact the risk of PPH in either or both populations.

**Influence of Anemia on PPH**

It is difficult to compare incidences of anemia in different populations due to the varying definitions of anemia. Anemia as defined in this study (hematocrit \( < 35\% \) upon hospital admission for labor and delivery) does not take into account women whose anemia was resolved prior to admission, nor does it include anemic women who did not meet the sample criteria. Greenhill and Friedman (1974) stated that from 10 to 75\% of pregnant women are anemic. The occurrence of anemia in this research was 10.1\%. Thus, the only comparison that can safely be made is that the pregnant Navajo women investigated probably fall into the same broad range of incidence of anemia as does the general population. Of all anemic women, 31.6\% had PPH, a remarkable finding in itself. What appeared even more remarkable was the fact that PPH occurred significantly more often in anemic women of the age group considered by common obstetrical knowledge to be most "normal" (18-35 years). And, though all parity groups showed significant associations between anemia and PPH, it was the group considered most "normal" (parity 2-6) that had the greatest strength
of association. Together, these two groups were singled out when age-and-parity were combined, showing that 18-35 year olds with parity 2-6 had the most significant association between anemia and PPH.

Considering that anemia and PPH were significantly associated in all age and parity groups (there were no < 17 year olds with anemia), the careprovider should remain alert to PPH whenever anemia is present. Moreover, the fact that the greatest strength of association was found in the essentially "normal" age and parity groups should stress the importance of maintaining this vigil even when the patient is of traditionally-defined "low-risk" age and parity.

One speculative explanation for the association between anemia and PPH is uterine muscle hypoxia. Hemoglobin is the oxygen-carrying protein in blood. Anemia is the state of a reduction in hemoglobin levels, and therefore, in the blood's oxygen-carrying capacity. After hours of maximal exertion, it is easy to imagine the musculature of an anemic women's uterus "giving up" due to insufficient oxygen, relaxing and hemorrhaging.

**Influence of Oxytocin on PPH**

The occurrence of oxytocin stimulation in this sample was 11.3% (n = 66). Half of those receiving oxytocin were induced and half were augmented. The occurrence of induction (5.9%) found in this sample of Navajo women
differs greatly from the incidences for the general population reported by Fields (1968) (13.5%), Fields et al. (1978) (18.4%) and Brinsden and Clark (1978) (36.5 to 42.1% for an eight year period). Comparison of these figures is restricted in that no differentiation between elective (assumed to be lower risk) and medically-indicated (higher risk) inductions were made in this and other studies' samples. The exclusion of women who did not meet this study's selection criteria who may have received oxytocin stimulation must also be kept in mind. Incidences of augmentation were unavailable in the literature reviewed, making comparisons between this and other samples impossible.

Although no significant association was found between oxytocin stimulation and PPH in the overall population, a significant association between these variables was shown among 18-35 year old primigravidas. A remarkably high percentage (35.7%) of oxytocin-stimulated women in this group had PPH. Once again, these findings are contrary to traditional obstetrical doctrine, whose focus is on women of high parity and on those who are outside the 18-35 age group.

Influence of Oxytocin and Anemia on PPH

Considering the associations found between anemia and PPH and between oxytocin stimulation and PPH, it is reasonable to suspect that those women who were both
anemic and oxytocin-stimulated would have shown a high occurrence of PPH. Similarly, if the speculations on why anemia might cause PPH (reduced myometrial oxygenation and exhaustion) and why oxytocin stimulation might cause PPH (easily weakened myometrium) are well-founded, then the risk of PPH among anemic/oxytocin-stimulated women would be even greater than that of anemic women and of oxytocin-stimulated women. However, this increased risk for PPH was not shown, due at least partially to the small number of anemic/oxytocin-stimulated women in the sample. Only four women had both anemia and oxytocin stimulation, and only one of them had PPH -- an 18-35 year old primigravida.

**Nurse-Midwifery Implications**

Nurse-midwifery, though not dedicated to any one theoretical framework, has used the concept of "preventive maintenance" as a working basis for well-woman and normal maternity health care. Nurse-midwives have stressed the need to maintain and improve upon an individual's present health status in order to reduce the need for sudden and often drastic "crisis intervention." The relationship between anemia and PPH found in this study is a prime example of when this "preventive maintenance" modality is indicated.

Obstetrical "common knowledge" has assumed that associations existed between anemia and PPH, and between
oxytocin stimulation and PPH. These assumptions were based on little, or even contradicting, "scientific rationale." This study sought to replace "common knowledge" (correct or incorrect) with "scientific rationale." One common knowledge assumption was supported (anemia and PPH), while one was refuted (oxytocin stimulation and PPH). The scientific testing of common knowledge assumptions prior to their application in clinical practice is considered an important premise in nurse-midwifery research.

The assumption of an association between anemia and PPH was made by a Shiprock nurse-midwife. Hearing her advise her clients that the anemia, if unresolved, could lead to PPH (Darlington, 1980) lent impetus to the selection of anemia and PPH as variables worthy of study. The highly significant association found between the two variables showed that the nurse-midwife's assumption was probably appropriate. Thus, the need for effective means to prevent and/or cure anemia prenatally is evident.

The group of anemic Navajo women identified as being at highest risk for PPH were those in the 18-35 year old, 2-6 parity group. This group also comprises the bulk of the "essentially normal" childbearing women commonly cited as appropriate for nurse-midwifery care (Varney, 1980). These findings are in striking contrast with conventional obstetrical wisdom and suggest that, at least
among Navajo childbearing women, greater focus in research and patient management should be placed on the "normal" rather than on the "extreme" age and parity groups.

The resolution of anemia prior to delivery should be stressed as a major goal of prenatal care. Iron-deficiency anemia (which comprises 95% of all anemias) is easily diagnosed, and theoretically, easily resolved with diet modification and/or iron supplementation. Thus, nurse-midwives must assume the responsibility of resolving anemia prenatally, and of anticipating PPH when anemia remains unresolved during the intrapartum period.

A common assumption is that Navajo women are disdainful of "white man's medicine" and often do not take prescribed vitamin and iron supplements (Darlington, 1980). The question of how to motivate anemic Navajo women to take their supplements arises, and is an area requiring further research. For example, do threats of hemorrhage work? Is there an effective method of educating Navajo women about anemia, its prevention and its cure? Is the assumption that Navajo women underutilize iron and vitamin supplements valid? What approaches to diet counseling and modification might work?

The group of oxytocin-stimulated Navajo women identified as being at highest risk for PPH was 18-35 year-old primigravidas, a group also considered appropriate
for nurse-midwifery management. Although the association between oxytocin stimulation and PPH was somewhat tenuous, nurse-midwives could lessen the chances for PPH, especially for clients in this risk group, by reserving the use of oxytocin until other nurse-midwifery tactics (such as ambulation) have been employed. When oxytocin must be used, the nurse-midwife should be prepared to handle PPH quickly, for example, by typing and crossmatching the woman's blood for possible transfusion, and by having Methergine readily available at the time of delivery.

By practicing modes of preventive maintenance of women's health (for example, resolution of anemia) and anticipation and preparedness for life-threatening emergencies (such as PPH), nurse-midwives may reduce the physical and emotional stress placed on childbearing families. The net result, and ultimate goal, is the provision of more safe and satisfying childbirth experiences.

**Summary and Recommended Research**

In summary, the group of anemic women found to be at highest risk for PPH was 18-35 year old women of parity 2-6. The group of oxytocin-stimulated women at highest risk for PPH was 18-35 year old primigravidas. The identification of both of these as risk groups for PPH was unexpected, and has serious implications for
nurse-midwives who care for women in these traditionally-defined "essentially normal" age and parity groups.

An interesting and rather surprising finding was that there was no significant association between anemia-and-oxytocin stimulation and PPH, i.e. women who were both anemic and oxytocin-stimulated were not at higher risk for PPH than all others in the sample. Further study of anemic, oxytocin-stimulated women is warranted to clarify this issue.

Other areas for recommended research include:
- Occurrence of PPH controlling for dosage and length of oxytocin stimulation.
- Occurrence of PPH controlling for elective induction, medically-indicated induction, and spontaneous labor.
- Complications of the puerperium secondary to PPH.
- Complications of the puerperium secondary to anemia.
- PPH among women who are both anemic and oxytocin-stimulated.
- Incidence and duration of iron-deficiency anemia among Navajos in general and among Navajo women in particular.
- Anemia as an index of overall nutritional status.
- Ascertaining the need for, and discovering the means of effecting compliance with dietary counseling and iron supplementation during the prenatal period among Navajo women.
- Iron-rich foods and herbs in the Navajo diet.
- Seasonal anemia among the Navajo as a reflection of livestock and produce harvest.
- Replications of this study with other Navajo samples, and with non-Indian samples.
## APPENDIX A

### INTRAPARTUM CARE FORM

<table>
<thead>
<tr>
<th>Management of Patient by:</th>
<th>Disposition of Patient</th>
<th>Reason for Transfer</th>
<th>Diagnostic Services</th>
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<tr>
<td>NIC</td>
<td>CNM</td>
<td>SNM</td>
<td>MD</td>
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<tr>
<td>Placenta</td>
<td>Ectopic</td>
<td>Location</td>
<td>Diagnosis</td>
</tr>
<tr>
<td>Delivery</td>
<td>Normal</td>
<td>Assisted</td>
<td>Cesarean</td>
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<tr>
<td>Time spent in labor by day:</td>
<td>Primary care provider by day:</td>
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<tr>
<td>First stage</td>
<td>Second stage</td>
<td>Third stage</td>
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APPENDIX B

LETTERS OF PERMISSION

MATERNAL AND CHILD HEALTH PROJECT
Shiprock Service Unit
P. O. DRAWERM • TELEPHONE: 505/368-4941
SHIPROCK, NEW MEXICO 87420

November 20, 1980

Ann Darlington, SNM
Tom Lloyd, SNM
University of Utah
College of Nursing
25 South Medical Drive
Salt Lake City, Utah 84112

Dear Ms. Darlington:

I approve your request to use data from the Shiprock Maternal and Child Health Project Statistics sheets for your Thesis entitled "Postpartum Hemorrhage and Its Relationship to Anemia and Oxytocin Stimulation among the Navajo".

You will also need the approval of Dr. Taylor McKenzie as Shiprock Service Unit Director. This request has been made, I will notify you of his response as soon as it is received.

Sincerely yours,

[Signature]

Ruth Shiers, CNM
Project Director
Maternal and Child Health Project

cc: Ann Darlington
    Laraine Guyette
    Pile
    Chrono
November 21, 1980

Laraine Guyette, CNM
MCH Project
Shiprock Service Unit
P. O. Drawer M
Shiprock, New Mexico 87420

Dear Ms. Guyette:

Your request for approval of the research proposal titled "Postpartum hemorrhage and its relationship to Anemia and Oxytocin Stimulation among the Navajo" is hereby granted.

Sincerely,

Taylor McKenzie, M. D.
Service Unit Director
Shiprock Service Unit


Darby, W.J., Salsbury, C.G., McGanity, W.J., Johnson, H.F., Bridgforth, E.B., & Sandstead, H.R. A
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