

***Effect of the length of second stage of labor  
in primigravidae on Maternal & neonatal  
outcomes***

**Thesis**

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# List of Contents

<i>Title</i>	<i>Page No.</i>
<b><i>Introduction .....</i></b>	<b><i>1</i></b>
<b><i>Aim of the Work .....</i></b>	<b><i>4</i></b>
<b><i>Review of Literature</i></b>	
<i>Onset of labor .....</i>	<i>5</i>
<i>Mechanics of normal labor .....</i>	<i>20</i>
<i>Stages&amp; duration of normal labor .....</i>	<i>30</i>
<i>Active management of labor .....</i>	<i>42</i>
<i>Second stage of labor</i>	
<i>Duration</i>	<i>58</i>
<i>Physiological concept</i>	<i>60</i>
<i>Position</i>	<i>63</i>
<i>Fundal pressure</i>	<i>66</i>
<i>Instrumental delivery</i>	<i>67</i>
<i>Prolonged second stage&amp;its outcome</i>	<i>72</i>
<b><i>Patients And Methods .....</i></b>	<b><i>76</i></b>
<b><i>Results .....</i></b>	<b><i>85</i></b>
<b><i>Discussion .....</i></b>	<b><i>95</i></b>
<b><i>Summary.....</i></b>	<b><i>99</i></b>
<b><i>Conclusion .....</i></b>	<b><i>101</i></b>
<b><i>Recommendations .....</i></b>	<b><i>102</i></b>
<b><i>References .....</i></b>	<b><i>103</i></b>
<b><i>Arabic summary</i></b>	

## List of Figures

<b><i>Fig. No.</i></b>	<b><i>Title</i></b>	<b><i>Page No.</i></b>
<b>Fig. (1):</b>	regulation of uterine activity during pregnancy and labor .....	8
<b>Fig. (2):</b>	metabolic regulation of myometrial smooth muscle contraction and relaxation .....	12
<b>Fig. (3):</b>	uterine electrical activity recorded from two distinct sites S1 and S2. ....	22
<b>Fig. (4):</b>	foetal presentations and positions in labor .....	27
<b>Fig. (5):</b>	cervical dilation curve for nulliparous labor.....	32
<b>Fig. (6):</b>	modified WHO curve.....	33
<b>Fig. (7):</b>	the cardinal movements of labor. ....	38
<b>Fig. (8):</b>	length of the second stage in both group .....	86
<b>Fig. (9):</b>	assisted vaginal delivery and cs in both groups.....	87
<b>Fig. (10):</b>	perineal tears in both groups .....	88
<b>Fig. (11):</b>	post partum haemorrhage in both groups .....	89
<b>Fig. (12):</b>	puerperal infection in both groups .....	90
<b>Fig. (13):</b>	apgar score in both groups.....	91
<b>Fig. (14):</b>	umbilical ph in both groups.....	92
<b>Fig. (15):</b>	admission in both groups to nicu.....	93
<b>Fig. (16):</b>	bmi in both group.....	94

## List of Tables

<b><i>Table No.</i></b>	<b><i>Title</i></b>	<b><i>Page No.</i></b>
<b>Table (1):</b>	Progression of spontaneous labor at term. ....	34
<b>Table (2):</b>	Active management of labor scheme.....	44
<b>Table (3):</b>	Organizational components.....	45
<b>Table (4):</b>	Medical components. ....	46
<b>Table (5):</b>	dorsal position possible advantage.....	63
<b>Table (6):</b>	as regard general data.....	85
<b>Table (7):</b>	comparison between the studied group as regard length of second stage .....	86
<b>Table (8):</b>	comparison between the studied group as regard assisted delivery.....	87
<b>Table (9):</b>	comparison between the studied group as regard perineal tears.....	88
<b>Table (10):</b>	comparison between the studied group as regard post partum haemorrhage .....	89
<b>Table (11):</b>	comparison between the studie-puerperal infection.. ....	90
<b>Table (12):</b>	comparison between the studied group as regard apgar score at 5 minutes.....	91
<b>Table (13):</b>	comparison between the studied group as regard umbilical ph.....	92
<b>Table(14):</b>	comparison between the studied group as regard NICU admission. ....	93
<b>Table (15):</b>	comparison between studied group as regard body mass index .....	94

## List of Abbreviations

ACTH	Adrenocorticotropin Releasing Hormone
ADP	Adenosine Di Phosphate
AMP	Adenosine Mono Phosphate
ATP	Adenosine Tri Phosphate
C.T	Computed Tomography
C.I	Confidence Interval
CPD	CephaloPelvic Disproportion
CRH	Corticotropin Releasing Hormone
CS	Caesarean Section
Ca <sup>++</sup>	Calcium ions
DHEAS	Dehydroepiandrosterone
GIT	GastroIntestinal Tract
L	Liter
M	Muscarinic
ML	Milliliter
Mm.Hg	Millimeter mercury
MU	Mille unit
MW	Molecular Weight
PG	Prostaglandin
RNA	RiboNucleic Acid
R.R	Relative Risk
SD	Standard Deviation
SPSS	Statistical Package of Social Science
T1/2	Half life
UK	United Kingdom
USA	United States of America
WHO	World Health Organization

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# ***ABSTRACT***

## ***Background***

Valsalva pushing efforts sustained for longer than 5 to 6 seconds lead to alterations in maternal and fetal hemodynamics, such as a lowering of maternal blood pressure and blood flow to the placenta, decreased fetal pH and PO<sub>2</sub>, elevated PCO<sub>2</sub>, fetal acidemia, more nonreassuring fetal heart rate deceleration patterns with delayed recovery to baseline, and lower Apgar scores. Compounded by the use of a supine maternal position, which also has deleterious hemodynamic consequences these effects may become even more hazardous .

## ***Conclusion***

The use of Valsalva during second stage of labor has been associated with a significant reduction in the duration of the second stage of labor in primigravidae but there is no significant association between using Valsalva and adverse maternal outcome ,also there was significant association between coached Valsalva and Apgar score less than 7 and fetal acidosis (pH below 7.2)

## ***Key word***

Primigravidae - second stage of labor - Valsalva pushing - maternal&neonatal outcomes

## Introduction

The second stage begins with complete cervical dilatation and ends with the delivery of the fetus. The American College of Obstetricians and Gynecologists (ACOG) has suggested that a prolonged second stage of labor should be considered when the second stage of labor exceeds 3 hours if regional anesthesia is administered or 2 hours in the absence of regional anesthesia for nulliparas. In multiparous women, such a diagnosis can be made if the second stage of labor exceeds 2 hours with regional anesthesia or 1 hour without it (*ACOG.*, 2003)

Studies performed to examine perinatal outcomes associated with a prolonged second stage of labor revealed increased risks of operative deliveries and maternal morbidities but no differences in neonatal outcomes (*cheng et al.*, 2004 / *janni et al.*, 2002)

Maternal risk factors associated with a prolonged second stage include nulliparity, increasing maternal weight and/or weight gain, use of regional anesthesia, induction of labor, fetal occiput in a posterior or transverse position, and increased birth weight. (*O'Connell et al.*, 2003 / *Senécal et al.*, 2005)

A number of randomized controlled trials have shown that, in nulliparous women, delayed pushing, or passive descend, is not associated with adverse perinatal outcomes or an increased risk for operative deliveries despite an often prolonged second stage of labor. (*Fraser et al.*, 2000 / *Fitzpatrick et al.*, 2002)

Furthermore, investigators who recently compared obstetric outcomes associated with coached versus uncoached pushing during the second stage reported



a slightly shortened second stage (13 min) in the coached group, with no differences in the immediate maternal or neonatal outcomes. (*Bloom et al., 2006*)

When a prolonged second stage of labor is encountered, clinical assessment of the parturient, the fetus, and the expulsive forces is warranted. A randomized controlled trial performed by Api et al determined that application of fundal pressure on the uterus does not shorten the second stage of labor. (*Api et al., 2009* )

Although the 2003 ACOG practice guidelines state that the duration of the second stage alone does not mandate intervention by operative vaginal delivery or cesarean delivery if progress is being made, the clinician has several management options (continuing observation/expectant management, operative vaginal delivery by forceps or vacuum-assisted vaginal delivery, or cesarean delivery) when second-stage arrest is diagnosed.

The association between a prolonged second stage of labor and adverse maternal or neonatal outcome has been examined. While a prolonged second stage is not associated with adverse neonatal outcomes in nulliparas, possibly because of close fetal surveillance during labor, but it is associated with increased maternal morbidity, including higher likelihood of operative vaginal delivery and cesarean delivery, postpartum hemorrhage, third- or fourth-degree perineal lacerations, and peripartum infection. (*Menticoglo et al., 1995*) Therefore, it is crucial to weigh the risks of operative delivery against the potential benefits of continuing labor in hopes to achieve vaginal delivery. The question of when to intervene should involve a thorough evaluation of the ongoing risks of further expectant management versus the risks of intervention with vaginal or cesarean delivery, as well as the patients' preferences .

The fetal response to Valsalva bearing-down efforts has also been studied scientifically. (*Aldrich et al., 1995* ) The fetus experiences a decrease in oxygen saturation with Valsalva bearing down that is not observed when the mother is supported to use spontaneous bearing down. (*Simpson and James ., 2005*) More specifically,

Valsalva pushing efforts sustained for longer than 5 to 6 seconds lead to alterations in maternal and fetal hemodynamics, such as a lowering of maternal blood pressure and blood flow to the placenta, decreased fetal pH and PO<sub>2</sub>, elevated PCO<sub>2</sub>, fetal acidemia, more nonreassuring fetal heart rate deceleration patterns with delayed recovery to baseline, and lower Apgar scores. (*Caldeyro-Barcia et al., 1981*) Compounded by the use of a supine maternal position, which also has deleterious hemodynamic consequences (*Sleep et al., 2000*) these effects may become even more hazardous (*Gupta Et al., 2004*)

## Aim of the work

The primary purpose of this study is to compare the effects of two different methods of care during the second stage of labor for nulliparous women (coached versus uncoached pushing ) on the length of the second stage and how this may affect maternal and neonatal outcome .

## Onset of labor

Labor is a clinical diagnosis characterized by regular, painful uterine contractions that increase in frequency and intensity are associated with progressive cervical effacement or dilatation. More specifically, it is associated with a change in the myometrial contractility pattern from irregular “contractures” (long-lasting, low-frequency activity) to regular “contractions” (high-intensity, high frequency activity) (*Liao et al., 2005*).

Uterine contractility increases during the latter stages of pregnancy to reach a crescendo at labor. The mechanism whereby the uterus is transformed from a relaxed, inactive state to an efficient contractile unit is both fascinating and complex (*Caldero-Barcia, 1959*).

The progression of pregnancy to term relies on profound physiologic and anatomic changes within the uterus and on a state of prolonged myometrial quiescence. Quiescence is a function of decreased myometrial excitability, with resistance to activation and with associated incomplete cell-cell coupling, resulting in low basal smooth muscle tone and absence of contractions. At present, no single factor has been shown to be responsible for these physiologic changes in pregnancy. Pregnancy may rather represent a balance between "propregnancy" factors, such as progesterone, that maintain myometrial quiescence and allow uterine expansion and "prelabor" factors, such as prostaglandins, that result in the onset of uterine contractions, cervical remodeling and ultimately delivery of the foetus (*Dennes et al., 1999*).

Animal studies indicate that either the foetus or the intrauterine environment may affect the timing of parturition (*Zhang et al., 2008*).

The mechanisms involved in the onset of labor at term remains ill defined. Major questions continue to surround the biologic processes that influence human parturition. More characterization of maternal and foetal signals would be useful in understanding the physiology of both normal and abnormal labor (*Dennes et al., 1999*).

Most parturition researchers have been struggling with two general theories, these are: (1) the retreat from pregnancy maintenance hypothesis and (2) the uterotonic induction of parturition theory. Some researchers speculate that the mature human foetus is the source of the initial signal for the commencement of the parturitional process; in this manner, the foetus could be in charge of its own destiny with respect to a timely birth. Foetal induced retreat from continued pregnancy maintenance is a satisfactory theory but as yet has little direct experimental support in human parturition (*Cunningham et al., 2005*).

Alternatively, because the myometrium is the site of uterine contractility, and because greater quantities of inflammatory cytokines are found in the uterus than in other tissues, it seems possible that the mother is in fact the primary producer of labor inducing signals. On the other hand the proximity of the amnion and chorion to the decidua and that of the decidua to the myometrium may increase the relative importance of factors produced by these tissues (*Hirsch et al., 1999*).

However, there appears to be no single path to the onset of labor. Cervical, decidual and foetal membrane cells may act in concert to promote parturition. Amniochorionic and decidual cells of the placenta, located as they are between the myometrium and foetus, are strategically poised to respond to physiologic, hormonal paracrine and autocrine regulation of this process (*Lockwood, 1999*).

For the purposes of considering how uterine activity is regulated during the latter part of pregnancy and labor, four distinct physiologic phases are described (*Norwitz et al., 2001*). During pregnancy, the uterus is maintained in a state of functional quiescence (Phase 0) through the integrated action of one or more of a series of inhibitors, including progesterone, prostacyclin, relaxin, nitric oxide, parathyroid hormone-related peptide, calcitonin gene-related peptide, adrenomedullin, and vasoactive intestinal peptide. Before term, the uterus undergoes a process of activation (Phase 1) and stimulation (Phase 2). Activation is brought about in response to one or more uterotropins (such as estrogen) with increased expression of a series of contraction-associated proteins (including myometrial receptors for prostaglandins and oxytocin), functional activation of select ion channels, and an increase in connexin-43 (a key component of gap junctions). After activation, the “primed” uterus can be acted upon by uterotonins, such as oxytocin and the stimulatory prostaglandins (E2 and F2a), and stimulated to contract. Phase 3 events (uterine involution) occur after delivery and are mediated primarily by oxytocin and possibly thrombin (*Liao et al., 2005*).

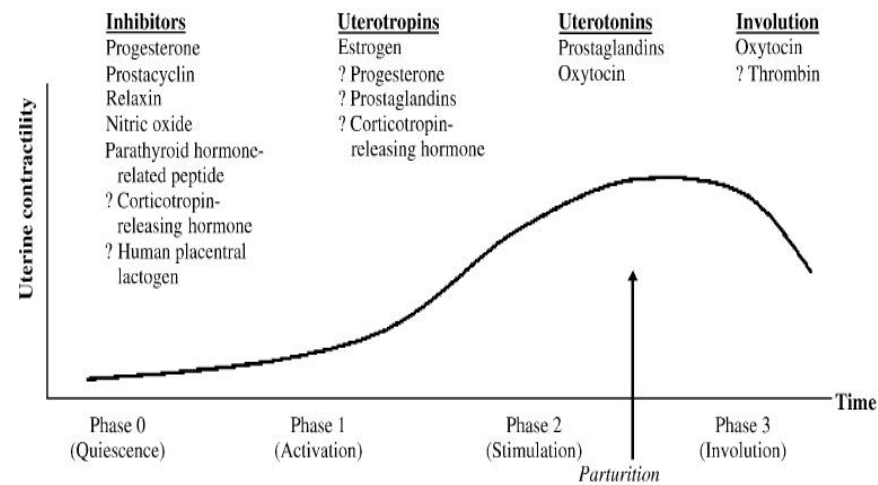


Fig 1. Regulation of uterine activity during pregnancy and labor. (*Challis et al., 1994*).