COMPARISON OF SAFETY AND EFFICACY OF INTRAVENOUS MAGNESIUM SULPHATE AND ORAL NIFEDIPINE IN TREATMENT OF PRETERM LABOUR

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OBSTETRICS AND GYNAECOLOGY

K.A.P. Viswanatham Government Medical College
Tiruchirappalli

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CERTIFICATE

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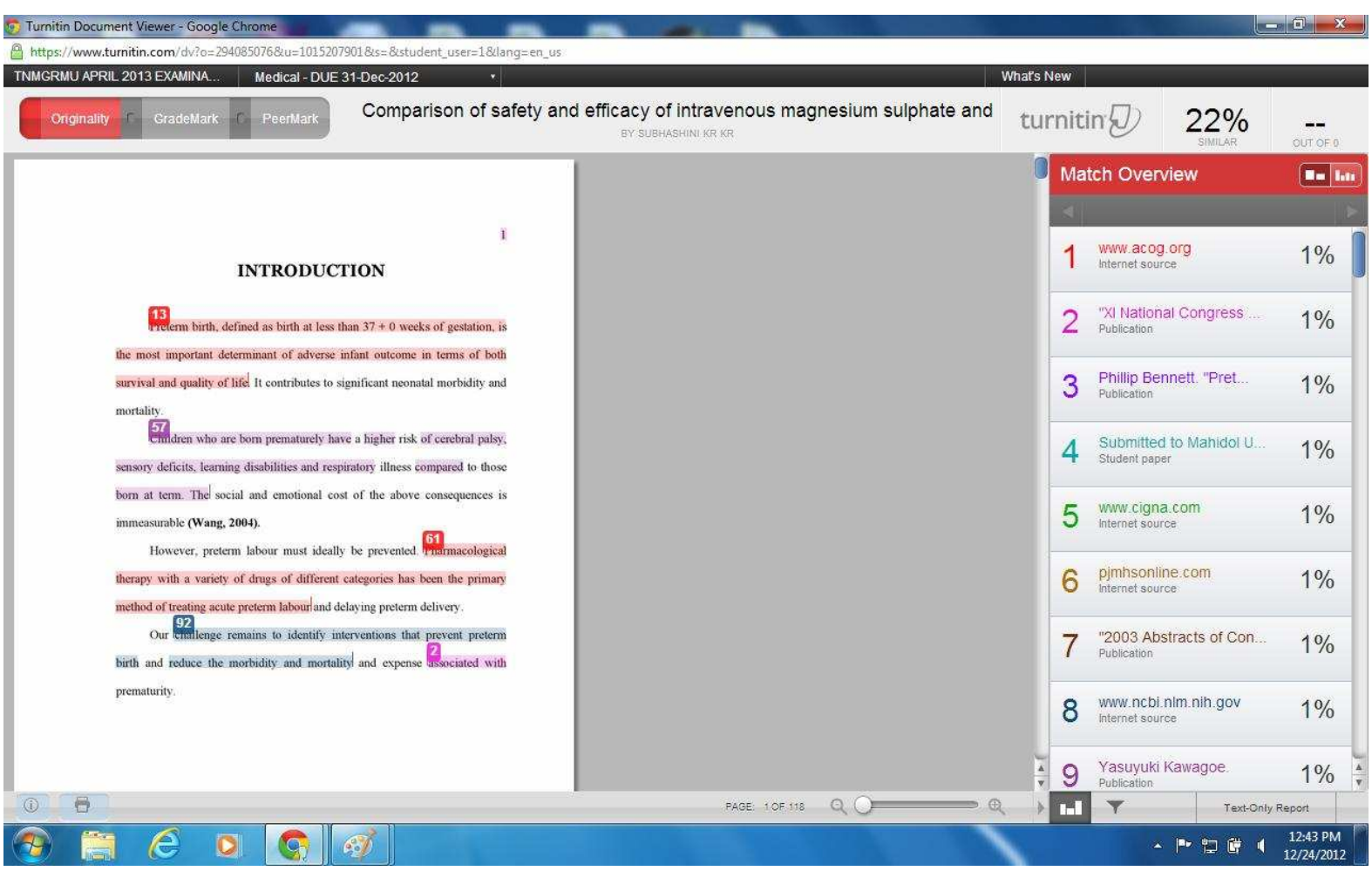
PLAGIARISM ORIGINALITY CERTIFICATE



CONTENTS

S. No	Title	Page No.
1	Introduction	1
2	Aim of the Study	2
3	Review of Literature and Drug Pharmacokinetics	3
4	Materials and Methods	48
5	Analysis of Results	56
6	Discussion	76
7	Summary	79
8	Conclusion	83
9	Bibliography	85
10	Proforma	92
11	Master Chart	

INTRODUCTION

INTRODUCTION

Preterm birth, defined as birth at less than 37 + 0 weeks of gestation, is the most important determinant of adverse infant outcome in terms of both survival and quality of life. It contributes to significant neonatal morbidity and mortality.

Children who are born prematurely have a higher risk of cerebral palsy, sensory deficits, learning disabilities and respiratory illness compared to those born at term. The social and emotional cost of the above consequences is immeasurable **(Wang, 2004).**

However, preterm labour must ideally be prevented. Pharmacological therapy with a variety of drugs of different categories has been the primary method of treating acute preterm labour and delaying preterm delivery.

Our challenge remains to identify interventions that prevent preterm birth and reduce the morbidity and mortality and expense associated with prematurity.

AIM OF THE STUDY

AIM OF THE STUDY

- To administer corticosteroid therapy to improve fetal lung maturity.
- To compare the safety and efficacy of intravenous $MgSO_4$ and oral nifedipine in acute tocolysis.
- To record the effects of the above two drugs on the mother and the fetus.

Review of Literature and Drug Pharmacokinetics

REVIEW OF LITERATURE AND DRUG PHARMACOKINETICS

DEFINITION

Preterm labour is defined as the occurrence of regular, painful, frequent uterine contractions associated with progressive cervical effacement and dilatation before 37 completed weeks of gestation from the first day of last menstrual period **(WHO, 1992).**

The period of viability varies in different countries from 20 to 28 weeks depending on the facilities available for newborn care and the likelihood of survival. In India, for legal purposes viability is defined as any gestation carried beyond 28 weeks (196 days). On a practical note, every centre has to recognise its lower limit for salvaging babies in the event of such a delivery.

American Academy of Pediatrics and American College of Obstetricians and Gynaecologists considered preterm labour to be established if regular uterine contractions can be documented atleast 4 in 20 minutes or 8 in 60 minutes, with cervical dilatation greater than 1 cm and cervical effacement of 80% or greater **(Cunningham et al., 2010)**.

INCIDENCE

Spontaneous preterm birth before 37 weeks gestation occurs in 7 - 11% of pregnancies and before 34 weeks gestation in 3 - 7% of pregnancies **(Maternal and Child Health Consortium. 6th Annual Report, 1999).**

Preterm delivery, particularly that before 34 weeks gestation, accounts for three-quarters of neonatal mortality and one-half of long term neurological impairment in children, including developmental delay **(Paneth NS, 1995; Stewart AL , Amess PN et al., 1999).** The incidence in India being 10-14% **(FOGSI).** In Mahatma Gandhi Memorial Government Hospital, Trichy the incidence is 13%.

IMPACT OF PRETERM BIRTH

A preterm infant is defined as one who is born at less than 259 days (37 completed weeks) of pregnancy **(WHO, 1977).**

Maternal mortality and morbidity as a consequence of preterm birth are rare. The most common maternal complication is postpartum endometritis which responds rapidly to administration of antibiotics **(Fernando Arias, 3rd Edn.)**

The birth and subsequent hospitalisation of a very premature infant evokes considerable psychological distress in mothers.

Risks of preterm infant

Neonatal survival for preterm infants is directly related to their gestational ages and birth weights **(Fernando Arias, 3rd Edn.).** Problems of preterm birth are related to difficulty in extrauterine adaptation due to immaturity of organ systems.

Short term problems (Manual of Neonatal Care, 2011)

(1) Respiratory

- Perinatal depression
- Respiratory distress syndrome
- Apnoea due to immaturity

(2) Neurologic

- Intraventricular haemorrhage (germinal matrix haemorrhage)
- Periventricular leukomalacia

(3) Cardiovascular

- Hypotension
- Patent ductus arteriosus.

(4) Haematologic

- Anemia
- Hyperbilirubinemia

(5) Nutritional Problems

- Growth failure

(6) Gastrointestinal

- Necrotising enterocolitis
- Feeding intolerance

(7) Metabolic

- Hypoglycemia
- Hypocalcemia

(8) Renal

- Water and electrolyte imbalance
- Acid - base disturbances

(8) Temperature Regulation

- Hypothermia

(9) Immunologic

- Sepsis

(10) Ophthalmologic

- Retinopathy of prematurity

Long term problems (Manual of Neonatal Care, 2011)

(1) Chronic Lung Disease

- Bronchopulmonary dysplasia
- Wilson Mikity Disease
- Chronic pulmonary insufficiency of prematurity.

(2) Nutritional

- Failure to thrive

(3) Cardiovascular

- Pulmonary hypertension
- Hypertension in adulthood

(4) Immunologic

- Recurrent respiratory infections

(5) Central Nervous System

- Cerebral palsy
- Developmental delay
- Sensorineural deafness
- Language disorder
- Learning disabilities

(6) Ophthalmologic

- Retinal detachment
- Myopia
- Strabismus

(7) Metabolic

- Impaired glucose regulation
- Insulin resistance

Preterm birth is a significant cost factor in healthcare, not considering the expenses of long-term care for individuals with disabilities (who require special needs, education, mobility aids and additional healthcare) due to preterm birth. It has been calculated that prematurity is responsible for 35% of all healthcare spending **(Lewit E, Baker L et al., 1995)**. The costs increase exponentially with decreasing gestational age and weight. There are also major implications in terms of psychological and social impact of disability on the individuals and their careers.

ETIOLOGY

A wide variety of etiological factors have been implicated in the causation of preterm labour, although in large majority of patients no definite cause can be found.

Nearly 50-60% of preterm births occur following spontaneous labour, 30% due to preterm premature rupture of membranes and the rest are iatrogenic terminations for maternal or fetal benefit **(Goldenberg RL, 2002; Leitich H, 2005).**

(1) Infections

Goldenberg and Colleagues (2008) have reviewed the role of infection in preterm birth.

(a) Uterine

Infection is the most clearly recognised and more widely studied cause of preterm birth. Infection is responsible for about 50% of all cases of spontaneous preterm birth **(Klein LL, Gibbs RS, 2005).** The most rigorous criteria for diagnosis of infection are positive cultures or demonstration of bacterial “fingerprints” by Polymerase Chain Reaction (PCR) in the amniotic fluid.

The most accepted mechanism of infection causing preterm birth is ascending infection. Bacteria may also gain access to the amniotic cavity through haematogenous spread or by introduction at the time of invasive procedures.

Studies demonstrate association between colonisation of genital tract with specific microorganisms and preterm labour. These include niesseria gonorrhoea, group B streptococci, chlamydia trachomatis **(Alger et al., 1988),** mycoplasma hominis and ureaplasma urealyticum **(Lamont et al., 1987),** gardnerella vaginalis, bacteroides species and haemophilus species **(McDonald et al., 1991, Kurki et al., 1992).** Asymptomatic bacterial vaginosis and trichomonas vaginalis confers a modest risk of spontaneous preterm birth.

The relative risk of preterm labour is doubled if the mother has bacterial vaginosis **(Goldenberg RL, Iams JD, Mercer BM, et al.)** Women with bacterial vaginosis and susceptible TNF-α genotype had a 9 fold increased incidence of preterm labour. Bacterial vaginosis infection in early pregnancy may be at a greater risk factor than in late second trimester and early third trimester of pregnancy **(Hay PE, Lamont RF et al.).** Bacterial vaginosis **(Gravett et al.)** has association with low birth weight.

(b) Extrauterine

Approximately 5-10% of patients in preterm labour have infection outside the uterus, most commonly in the urinary tract **(Romeo et al., 1988).**

Systemic infections like pyelonephritis, pneumonia, acute appendicitis often lead to increased uterine activity and preterm labour **(Am J Obstet Gynecol., 2006)**. Periodontal disease is associated with preterm labour **(Xiong X., 2006).**

(2) Placental

- Abnormal placentation
- Anatomical abnormalities
- Placenta previa
- Abruptio placentae

(3) Uterine

- Congenital abnormalities (1-3%)
- Anatomic or physiologic abnormalities of uterine cervix **(Nohr et al., 2007).**
- Uterine overdistension.

(4) Genetic

The recurrent, familial and racial nature of preterm birth has led to the suggestion that genetics may play a causal role **(Anum, 2009; Lie, 2006 and all their co-workers).**

Single gene polymorphisms of cytokines in both mother and fetus may be responsible. Polymorphism involving tumour necrosis factor -α 308 (TNF-α 308), interleukin-1 beta (1L-1β) and interleukin-6 have been most consistently associated with spontaneous preterm labour and preterm birth **(Varner MW, Esplin MS, 2005).**

(5) Vaginal bleeding in early pregnancy is associated with increased adverse outcomes later **(Weiss and Associates, 2004).** Threatened first or early second trimester miscarriage doubles the risk of subsequent preterm labour.

(6) Fetal

Dolan and colleagues (2007) found that birth defects were associated with preterm labour.

Pregnancies complicated by fetal malformations, in particular multiple anomalies, renal anomalies and anterior abdominal wall defects, deliver preterm more often than expected.

(7) Preterm labour of unknown origin (20-30%)

(8) Iatrogenic

Developments in the field of antenatal diagnosis, maternal fetal medicine and neonatology have lead to increased early interventions by obstetricians. Conditions which threaten maternal and fetal well being (which account for one third of cases) are at risk of being delivered before term.

This number is on the rise with the availability of advanced fetal surveillance and imaging technology such as cardiotocograph and high end ultrasound machines. The practice of **'defensive medicine'** as a result of increase in litigation has only increased the burden **(Arulkumaran, 3rd Edn.).**

Increase in the number of multiple pregnancies, particularly higher order pregnancies, resulting from the use of fertility drugs and assisted reproduction is also one of the major reasons for increase in incidence of preterm birth.

PATHOPHYSIOLOGY

The molecular basis of initiation of labour is unclear but a number of theories have been proposed.

Maternal stress is a well recognised cause of activation of the mechanism of normal parturition, regulated by hypothalamic secretion of corticotrophin - releasing hormone or CRH **(Hobel et al., 1999).**

The fetal pituitary adrenal axis needs to be intact **(Gonik B et al.)**. As parturition nears, the fetal adrenal axis becomes more sensitive to ACTH and there is an increased production of cortisol. This stimulates 17- hydroxylase in the trophoblast resulting is decreased progesterone secretion. The reversal of the estrogen-progesterone ratio leads to increased prostaglandin formation and initiation of labour.

Cytokines are released when there is inflammatory response to infection and intrauterine bleeding **(Cox & Colleagues, 1993)**. These inturn stimulate arachidonic acid and prostaglandin production.

Labour as an Inflammatory Process

Cytokines	Action	Effect
IL-6, IL-8, IL-1, TNF-α	Degradation of collagen fibres	Cervical ripening
IL-1 & TNF-α	Induce matrix metalloproteinases	Membrane rupture
IL-1, IL-2, IL-6, TNF-α	Increase PGE2, $PGF_{2\alpha}$	Uterine contractions

Differential production of PGE_2 and $PGF_{2\alpha}$ by the three enzymes – phospholipases, PGH_2 synthase, 15-hydroxy prostaglandin dehydrogenase may be a key in the balance between uterine quiescence and activity.

Whatever the mechanism to initiate labour, three physiological processes have to occur, namely, softening and dilatation of cervix, uterine myometrial contractions and weakening and rupture of the membranes.

EPIDEMIOLOGY

(1) Racial and Ethnic Disparity

Black women have twice the risk of preterm delivery compared to whites **(Varner MW, Esplin MS, 2005).** This may be explained by socioeconomic status, medical disorders and genetic predisposition.

The magnitude of risk is greatest for extremely preterm deliveries **(Schoendorf et al., 1992).**

(2) Age

There is an increased risk of preterm delivery in women under 20 years of age **(Lumley, 1993)** and in women over 35 years of age.

(3) Parity

Primiparity is associated with a higher rate of preterm delivery independent of age **(Bakketeig and Hoffman).**

(4) Socioeconomic Status

The universal effect of low socioeconomic status on health appears to directly affect the incidence of preterm labour **(Moutquin, 2003).**

(5) Weight

Poor nutrition, low maternal prepregnancy weight (BMI < 20 kg / m^2) and poor weight gain (less than 0.24 kg / wk) are associated with an increased risk of preterm labour **(Wen et al., Barros et al., 1992).** Prenatal weight gain is specifically associated with preterm birth **(Hickey & Colleagues, 1995).**

(6) Stature

Short statured mothers have more tendency to produce preterm babies.

(7) Addiction

Smoking was found to be a significant independent factor associated with increased risk of preterm labour at 27-32 weeks **(Burguet A, Kaminshi M, 2004).**

Cocaine use increases the rate of preterm delivery, in part a consequence of a higher risk of abruptio placentae **(Volpe, 1992).**

An increased risk as a consequence of alcohol consumption remains unproven.

More recent data confirm an excess of preterm births in opiate users **(Boer et al., 1993, Walkinshaw et al., 1993).**

(8) Work During Pregnancy

Occupational factors such as prolonged walking or standing, strenuous working conditions and long weekly work hours (> 42 hrs / week or > 6 hrs / day) had a greater risk of preterm labour **(Casanueva, 2005, Giel Chinsky, 2002).**

PREDISPOSING FACTORS

(1) Psychosocial Factors

Recent studies have shown association between preterm birth and anxiety, stress and depression, negative life events, perception of racial discrimination and domestic violence **(Goffinet F, 2005; Hogne CJ; Bremner JD, 2005).**

(2) Coitus

Coitus during early pregnancy was not found to be associated, but increasing number of sexual partners increased the risk of recurrent preterm delivery **(Yost NP, Owen J, 2006).**

(3) Past Obstetric History

(a) Previous preterm birth

Previous preterm birth is the single best predictor of preterm delivery **(Wen et al., 1990)**. The recurrence risk ranges from 17-40% depending on the number of previous preterm deliveries **(Carr-Hill et al., 1985)**. Risk increases with the number of preterm births and decreases with the number of term deliveries.

(b) Previous spontaneous miscarriage

There is no association with history of one or two first trimester abortions, but three or more abortions increase the risk of preterm labour.

Previous second trimester spontaneous miscarriage carries an increased risk of subsequent preterm labour **(Keirse et al., 1978).**

(c) Uterine abnormalities

The most clinically significant of these conditions are the septate and bicornuate uterus with incidence of preterm birth varying between 16-20 weeks.

(d) Previous pregnancy bleeding

Women with a past history of antepartum bleeding especially abruptio placentae, are at increasing risk of recurrence **(Crenshaw et al., 1973)** and therefore are at increased risk of repeated preterm delivery.

(e) Cervical insufficiency (or incompetence)

Cervical insufficiency is a known risk factor for preterm labour and contributes to 10-25% of second trimester losses **(Vidaeff AC et al., 2006).**

(4) Current Pregnancy Complications

Many pregnancy complications are associated with preterm birth.

- Multiple pregnancy
- Preexisting medical illness
- Asymptomatic bacteriuria
- Preeclampsia

- Antepartum haemorrhage
- Polyhydramnios
- Fetal malformation
- Assisted conception pregnancy

(5) Interval Between Pregnancies

It was shown that intervals shorter than 18 months and longer than 59 months were associated with preterm birth **(Conde-Agudelo, 2006).**

(6) Fetal Gender

The main fetal factor influencing the rate of preterm delivery is fetal sex, with the preponderance of males delivering preterm.

RISK FACTOR SCORING

A risk scoring system devised by **Papiernik** and modified by **Creasy and Govit (1986)** has been tested in several regions. Women with a modified risk assessment score of 10 or higher are considered to be at high risk for spontaneous preterm labour.

Scoring system is based on epidemiological risk factors. Past reproductive history plays a major role, with a history of previous preterm births, second trimester miscarriages and cone biopsy all scoring highly **(Creasy et al., 1980; Holbrook et al., 1989).** Overall performance of risk scoring, though easily applied, has been poor. Pooled results give a sensitivity of only 40%, positive predictive value of 20-30% and false positive rate of almost 80% **(McLean et al., 1993)**. Such results fail to identify most women at risk and falsely label many normal pregnancies as at risk, exposing them to potential interventions and is of limited clinical use.

PAPIERNIK RISK SCORING SYSTEM

Screening for risk of preterm labour, other than historical risk factors, is not beneficial in the general obstetrical population **(ACOG, 2008).**

Points	Socioeconomic Factors	Previous Medical History	Daily Habits	Aspects of Current Pregnancy
1	Two children at home, low socio-economic status	One abortion, less than 1 year since last birth	Works outside	Unusual fatigue
2	Maternal age < 20 years or > 40 years, single parent	Two abortions	Smokes more than 10 cigarettes per day, more than 3 flights of stairs without elevator	Gain of < 5 kg by 32 weeks
3	Very low socio-economic status, height < 150 cm, weight < 45 kg	Three abortions	Heavy or stressful work that is long and tiring, extensive travelling, long daily commuting	Breech at 32 weeks, weight loss, head engaged at 32 weeks, febrile illness
4	Maternal age < 18 years	Pyelonephritis		Bleeding after 12 weeks, short cervix, opened internal os, uterine irritability
5		Uterine anomaly, second trimester abortion, DES exposure, cone biopsy		Placenta previa, hydramnios
10		Preterm delivery, repeated second trimester abortion		Twins, abdominal surgical procedure

Table adapted from **Creasy et al., 1980.**

SCREENING METHODS FOR PREVENTION OF PRETERM LABOUR

Prediction of preterm birth is an important strategy in reducing neonatal morbidity and mortality. Various means have been used to identify the women at risk. A good primary marker should be applicable to both asymptomatic as well as symptomatic women. The two most promising markers currently available are fetal fibronectin levels and ultrasound assessment of cervical length.

(1) Cervical Changes

(a) Cervical Dilatation

It has been widely assumed that premature effacement or dilatation of cervix is related to an increased risk of preterm delivery. Approximately 25% of women whose cervices are dilated 2 or 3 cm deliver prior to 34 weeks. Positive predictive value of cervical changes range from 4 to 30% **(Leveno et al., 1986; Mortensen et al., 1987).**

(b) Cervical Length – The short cervix

Changes in the anatomy of cervix - endocervical shortening , dilatation and herniation of membranes (funneling) are considered as changes along a continuum in the early stages of preterm labour. This cervical remodeling can be viewed on ultrasound imaging. Transvaginal ultrasound is currently recommended as the most reliable method which is reproducible as opposed to transabdominal and translabial methods **(Berghella et al., 2005)**. Studies have shown that the risk of preterm labour is inversely proportional to the cervical

length at 24 to 28 weeks. Serial measurement appears to be superior to a single measurement in assessing the risk of preterm delivery.

Transvaginal cervical measurement identifies about 1% of the population who are at high risk for preterm delivery. A cervical length greater than 25 mm would be considered normal. A shortened cervix in association with ‘funneling’ has been shown to be an indicator of greater risk of preterm delivery **(Rust et al., 2005).**

Berghella et al showed that funneling or dilatation of the internal cervical os > 5 mm is associated with a positive predictive value of 33 – 40 % for preterm labour versus 11.3 % for a short cervix.

(2) Cervicovaginal Fetal Fibronectin

Fetal fibronectin is a basement membrane protein produced by trophoblasts. It acts as a ‘glue’ holding the placental membrane and the decidua together.

Several metaanalysis have confirmed the value of fetal fibronectin in the prediction of preterm birth in symptomatic and asymptomatic women **(Faron et al., 1998; Honest et al., 2002).**

Swabs can be taken from ectocervix or posterior vaginal fornix and an ELISA with an FDC-6 monoclonal antibody is used to detect fetal fibronectin. Levels > 50 ng / ml is usually considered positive. A negative fetal fibronectin indicates that delivery will not occur in the next 2 or 3 weeks, while a positive

result will increase markedly the possibilities of preterm labour and delivery before 34 weeks.

It has a high false positive rate as a result of contamination of sample with maternal blood or semen, speculum or digital pelvic examination, endovaginal ultrasound examination and in women with cerclage, rupture of membranes. Serial sampling improves positive predictive accuracy but with a lower specificity.

The high negative predictive value of fetal fibronectin can be used to influence management.

The usefulness of fetal fibronectin testing along with transvaginal ultrasound evaluation of the cervical length has been assessed **(Krupa et al., 2006)** and found effective in predicting admission to delivery interval. Most important is their clinical usefulness in providing good negative predictive values **(ACOG, 2005) .**

No current data support use of home uterine activity monitoring or bacterial vaginosis screening **(ACOG 2001, 2008).**

(3) Biochemical Markers (Odibo AO, Ural SH, Macones G, 2002)

More research is necessary to validate this approach and determine the predictive values and the cost effectiveness of this approach. These include:

- Salivary estriol **(McGregor JA , Jackson GM et al., 1995)**
- Progesterone
- Serum collagenase

- Alpha fetoprotein
- Tissue inhibitor of metalloproteinase (TIMP) / Matrix metalloproteinase
- Relaxin
- Corticotrophin releasing hormone (CRH) **(McLean M , Smith R et al., 1999)**
- Adrenocorticotrophic hormone (ACTH)
- Prolactin
- Thyroid stimulating hormone (TSH)
- Alkaline phosphatase (ALP)
- Serum ferritin, ferritin / Iron ratio
- Human chorionic gonadotrophin (HCG)

Mediators of inflammation and infection

- C-Reactive Protein (CRP)
- Leucocyte esterase
- Granulocyte - colony stimulating factor
- Cytokines (TNF-α , IL-1, IL-6)
- Prostaglandins
- Amniotic fluid glucose concentration
- Zinc
- Lipocortin – 1
- Positive cultures

The US **National Institute of Child Health and Human Development Network of Maternal-Fetal Medicine Units (NICHD MFM Network)** study

showed that alpha fetoprotein , alkaline phosphatase and granulocyte-colony stimulating factor were the most promising biochemical markers **(Goldenberg RL, Iams JD et al., 2001)**

DIAGNOSIS OF PRETERM LABOUR

The signs and symptoms of labour may appear only within 24 hours of preterm labour **(Iams et al., 1994).**

(1) Symptoms

- Pelvic pressure
- Menstrual like cramps
- Watery vaginal discharge
- Low back pain
- Painful or painless uterine contractions.

Cervical changes in the presence of uterine contractions is the strongest indicator of preterm labour.

(2) Clinical Examination

Abdominal Examination	▪ Uterine contractions at regular intervals
Speculum examination (Sterile procedure to minimise risk of infection)	▪ To determine the length of cervix and extent of dilatation of cervical os ▪ To determine the presence of amniotic fluid
Digital examination	▪ To be avoided if membranes have ruptured, unless sufficient information was not obtained during speculum examination ▪ Cervical effacement and dilatation are looked for.

(3) Role of ultrasound in preterm labour

▪ Fetal viability ▪ Number of fetuses ▪ Placental localisation and morphology ▪ Fetal presentation and lie
Standard fetal biometry ▪ Gestational age ▪ Estimated fetal weight ▪ Determine growth pattern (looking for FGR)
Amniotic fluid index
Fetal activity
Indicators of preterm labour ▪ Cervical length ▪ Length of funnel ▪ Width of the internal os ▪ Cervical index
Doppler Studies ▪ Umbilical artery ▪ Middle cerebral artery
Rule out gross structural anomalies ▪ Non viable fetal conditions ▪ Markers for chromosomal abnormalities

(4) Tococardiography

Tococardiograph records uterine activity and fetal heart rate on a graph. The amplitude, duration, frequency of contractions and basal tone are monitored. The uterine activity is expressed in Montevideo units.

Changes in FHR reactivity in preterm labour is closely linked with the immaturity of autonomic nervous system of fetus.

PREVENTION OF PRETERM LABOUR

Prevention of preterm birth has been an elusive goal. This requires timely and accurate screening of pregnant women for risk of preterm birth. Recent reports, however, suggest that prevention in selected population may be achievable.

(1) Basic Care

- Interventions like increased prenatal visits, patient education, home visits and nutritional counselling may play a role in reduction of preterm birth.
- Rest and hydration.
- Social and psychological support **(Fiscella K et al., 1995).**
- Avoidance of heavy manual labour **(Papiernik E , Collin D et al., 1985)** and mental stress.
- Behavioral & lifestyle modifications

  (a) Adequate nutrition

(b) Cessation of smoking **(Abraham - Lerat L et al., 2004)**

(c) Avoidance of illicit drugs

(2) Cervical Cerclage

With the emerging concept of a functional cervical insufficiency, the role of cervical cerclage for the prevention of preterm delivery is disputed. In 2001, the **Cervical Incompetence Prevention Randomised Cerclage Trial (CIPRACT trial)** showed that patients with cervical insufficiency and cerclage placement (as compared to bed rest only) had a lower incidence of preterm delivery prior to 34 weeks in addition to lowered neonatal morbidity **(Althuisius et al., 2001)**. No benefit has been noted in twins **(Dor et al., 1982)** or in triplets **(Roman et al., 2005).**

(3) Treatment of Cervicovaginal Infection

It has been postulated that intrauterine infection may trigger labour by activating the innate immune system **(Goldenberg et al., 2008).** A meta-analysis by **Morency and Bujold (2007)** seemed to indicate that antibiotics given in the second trimester to women with a history of preterm labour would be effective in preventing recurrence of preterm labour.

Bacterial vaginosis has been consistently associated with a 1.5 to 3 times increased risk of spontaneous preterm birth **(Goldenberg R and Rouse D et al., 1998)**, but the efficacy of treatment in reduction of preterm births is conflicting. A recent systematic review however concluded that screening and

treatment of asymptomatic bacteriuria and bacterial vaginosis in low risk population may reduce the rate of preterm deliveries **(Varma R, Gupta JK, 2006).**

(4) Progesterone

Weekly intramuscular injections of 250 mg of 17α-hydroxy-progesterone caproate from 16-20 weeks gestation to 37 weeks for prevention of recurrent preterm birth has resulted in significant reduction in early preterm births and infant complications **(Meis et al., 2003).** So far there has been no reported increase in congenital anomalies **(Resseguie LJ, Hick JF, 1985; Spong CY, 2003)**. Four year follow up found no adverse health outcomes of surviving children.

Use of micronised progesterone capsules (200 mg vaginally daily) for asymptomatic women with a very short cervix (less than 15 mm) between 24 and 34 weeks of pregnancy appear to be effective **(da Fonseca et al., 2007).**

MANAGEMENT OF PRETERM LABOUR

(a) **Bed Rest and Oral or Parenteral Hydration** have traditionally been used in the management of preterm birth.

Hydration may reduce uterine contractility by increasing uterine blood flow and by decreasing pituitary release of anti-diuretic hormone and oxytocin **(Stan C, Boulvain M et al., 2002).** A systematic review of the **Cochrane**

Database (Sosa et al., 2004) found no evidence to support or refute bed rest in preventing preterm birth.

(b) Corticosteroid Therapy

The **National Institute of Health Consensus Development Conference Statement in 1995,** stated that 'antenatal corticosteroid' therapy is indicated for women at risk of preterm delivery. This will result in substantial decrease in neonatal morbidity and mortality, as well as substantial savings in health care costs. A systematic review of the **Cochrane Database (Crowley, 2003)** confirmed that antenatal corticosteroids significantly reduced the incidence and severity of neonatal respiratory distress syndrome.

All women between 24 weeks and 34 weeks of gestation who are at risk of preterm delivery within 7 days are potential candidates for corticosteroid therapy **(Ballard et al., 1995).** Its effects on fetal intestine appear to be responsible for reduction in the risk of necrotising enterocolitis (NEC).

In addition, these neonates also have a reduced risk of intraventricular haemorrhage (IVH) **(RCOG 200**2**)**.

Although the benefit on neonatal outcome is maximum between 24 hours and seven days after initiation of therapy, steroids confer significant survival advantages even when delivery occurs within 24 hours.

Betamethasone or Dexamethasone?

Band et al (1999) found betamethasone to be superior to dexamethasone in preventing RDS and periventricular leukomalacia.

Dosage

Betamethasone ▪ 2 doses of 12 mg, given im 24 hours apart.
Dexamethasone ▪ 4 doses of 6 mg, given im 12 hours apart **(RCOG, 2004).**

"Rescue" Therapy

Though the **American College of Obstetricians and Gynaecologists (2008)** recommends that a rescue dose be restricted to trials**, McEvoy et al., (2010)** showed that a rescue dose > / = 14 days after the last dose increased respiratory compliance in the treated infants.

Single or Multiple Courses

A single course of antenatal corticosteroids is still considered standard of care **(ACOG, 2008).** Further studies are required before multiple doses can be recommended.

(c) Antibiotics

Administration of antibiotics to the mother does not delay delivery **(ORACLE Trial, 2001).** A **Cochrane meta-analysis by King and Flenady (2000)** found no decrease in rates of newborn respiratory distress syndrome or of sepsis in antimicrobial treated groups.

(d) Tocolysis

Tocolytic drugs have been used in an attempt to inhibit preterm labour. Tocolytic drugs may prolong gestation for 24 to 48 hours, which can provide time for

- the administration of antenatal steroids
- inutero transfer of mother to a tertiary unit
- to prepare for neonatal care **(Merkatz et al., 1980; Guinn et al., 1997).**

As a general rule if tocolytics are given, they should be given concomitantly with corticosteroids.

There appears to be a limited role for the use of tocolysis beyond 34 weeks for the inhibition of preterm birth **(Goldenberg, 2002).** There is no first line tocolytic drug. The choice should be individualised and is usually based on maternal condition, potential drug side effects and gestational age **(ACOG, 2003).**

(1) Beta Adrenergic Receptor Agonists

The commonly used beta sympathomimetic drugs are ritodrine, terbutaline and salbutamol. In terms of clinical effectiveness the inhibition of contractions by β-adrenergic agonists is often short lived.

Ritodrine infusion is started at a dose of 50 mcg / min and increased every 20 minutes until uterus is quiescent or side effects limit escalation of dose. The maximum recommended infusion rate is 350 mcg / min. The RCOG

no longer recommends ritodrine as the first drug of choice **(RCOG Clinical Guidelines, 2002).**

Terbutaline has not been used as much as ritodrine, but is effective in temporarily arresting contractions when given parenterally.

Route	**Regimen**
Oral	2.5 - 5 mg every 4 - 6 hours
Subcutaneous	250 mcg s.c. every 20-30 minutes × 4-6 doses
Intravenous	5-10 mcg / min increased every 10-15 minutes to a maximum of 80 mcg / min

Side effects

- Palpitations, tremor, nausea, headache
- Myocardial ischemia
- Cardiac arrhythmias
- Pulmonary edema
- Metabolic derangements – hyperglycemia, hypokalemia
- Fetal tachycardia and neonatal hypoglycemia.

Contraindications

- Symptomatic cardiac disease, especially ventricular outflow obstruction.
- Symptomatic cardiac rhythm or conduction disturbances.
- Hyperthyroidism

- Sickle cell disease
- Uncontrolled insulin - dependent diabetes mellitus
- Chorioamnionitis
- Eclampsia or severe preeclampsia
- Severe anaemia
- Multifetal gestation
- Psychiatric patients on MAO inhibitors
- Asthmatic patients already on β-adrenergic agents.

(2) Nonsteroidal Anti-inflammatory Agents

Drugs like indomethacin, aspirin, ibuprofen and sulindac belong to this group, of which indomethacin has been used the most. Comparison with beta-agonists show similar efficacy, but a better side effect profile **(RCOG Clinical Guidelines, 2002).**

Prostaglandin synthetase inhibitors are highly effective in inhibiting myometrial activity, a single dose being effective in many women at term **(Reiss et al., 1976).**

Side Effects

Maternal

- Headache
- Dizziness
- Gastritis, Vomiting

- Asthma recurrence
- Diarrhoea
- Thrombocytopenia

Fetal

- Constriction of ductus arteriosus
- Persistent fetal circulation
- Hydrops
- Oligohydramnios
- Bleeding disorders
- Necrotising enterocolitis / ileal perforation
- Intraventricular haemorrhage (grade III, IV)

All these fetal side effects have restricted their use.

(3) Oxytocin Receptor Antagonist (Atosiban)

This nonapeptide oxytocin analog is a competitive antagonist of oxytocin induced contractions. Duration of treatment should not exceed 48 hours and the total dose given should not exceed 330 mg of atosiban.

Compared to beta-agonists, atosiban was found to have comparable efficacy & fewer side effects. Side effects include nausea, chest pain, headache, vomiting, dyspnoea and injection site reactions. Last generation of oxytocin receptor antagonists such as barusiban would be more efficient and have less affinity for the vasopressin receptors.

The **Food and Drug Administration** has denied its approval because of concerns regarding efficacy and fetal-newborn safety. Its high cost is also a factor limiting its use in developing countries.

(4) Nitric Oxide Donors (Glyceryl Trinitrate)

Nitric oxide is a potent endogenous hormone causing smooth muscle relaxation. Nitroglycerine, a nitric oxide donor has been used for the treatment of preterm labour. **Cochrane review (2002)** showed that nitroglycerine did not delay delivery nor improve neonatal outcome **(Duckitt et al).**

Nitric oxide is generated from the oxidation of L-arginine by NO synthase. Nitric oxide increases cyclic guanosine monophosphate levels, leading to inactivation of the myosin light chain kinases, causing smooth muscle relaxation.

10 mg of glyceryl trinitrate patch is applied over the fundal region of maternal abdomen. If tocolysis is not achieved in one hour, another 10 mg patch can be applied to a maximum dose of 20 mg in 24 hours.

Side effects were fewer when compared to other tocolytics, but the incidence of headache and hypotension were high **(Bisits et al., 2004).** Currently there is not enough evidence to recommend nitroglycerine for routine clinical practice.

(5) Potassium Channel Openers

Diazoxide is a medication structurally related to the thiazide diuretics that is used in the treatment of hypertensive crisis. The drug inhibits

genitourinary smooth muscle and is responsible for its effectiveness as a tocolytic agent.

The dosage of diazoxide is 5 mg / kg given intravenously slowly in 15-30 minutes. It can also be given as boluses of 50-100 mg every 5 minutes.

The most common maternal side effects are hypotension, tachycardia, hyperglycemia and decreased uteroplacental blood flow. Fetal side effects are hyperglycemia and fetal distress.

Potassium channel openers under study are Pinacidil and Leveromalkim. Further studies of this group of tocolytic drugs is needed.

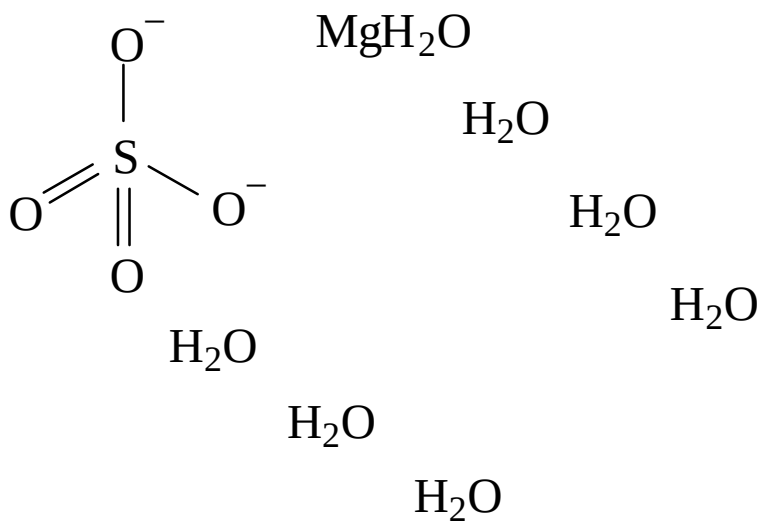
(6) Magnesium Sulphate

Prior to 1980s magnesium sulphate was widely used in the United States in the intrapartum management of preeclampsia and eclampsia and the clinical impression that magnesium sulphate made induction of labour more difficult lead to its evaluation as a tocolytic agent and is in common use now.

Magnesium sulphate has been shown to cause myorelaxation. Its role is presumably that of a calcium antagonist.

Pharmacology

- Magnesium sulphate USP is $MgSO_4.7H_2O$



Structure of Magnesium Sulphate

- It has a molecular weight of 24.3.
- 1 gram of magnesium sulphate has 98 mg of elemental magnesium.

Mechanism of action

The exact mechanism by which magnesium sulphate acts is unknown. It is proposed that it inhibits myometrial contractions by antagonizing calcium at the cellular level and in the extracellular space, reducing intracellular levels of calcium and preventing activation of actin-myosin complexes in smooth muscle. Magnesium may also act directly on calcium channels by competing for binding sites.

Proposed mechanisms

- Blockage of calcium entry via voltage gated channels **(Arango and Mejia – Mantilla, 2006).**
- Improved mitochondrial calcium buffering.
- Potentiation of adenosine action.
- Blockage of glutaminergic N-methyl-D-aspartate (NMDA) receptors.

Other actions

- Vasodilatation in vascular beds.
- Decrease in systemic vascular resistance and mean arterial pressure.
- Increased uterine blood flow.
- Increased renal blood flow.

Pharmacological effects

- Decrease in the frequency and intensity of uterine contractions and slow cervical dilatation.
- Anti-convulsant action.
- Transient hypotensive effect

Administration

Zuspan's Regime

Loading dose 4g of 20% $MgSO_4$ given intravenously over 5-10 minutes, at a rate not exceeding 1g / min **(Cunningham et al., 2010).**
Maintenance dose This is followed by an infusion of 1-2 g / hr in 50-100 ml of iv maintenance solution (NS) by controlled infusion pump **(RCOG, 2006).**

Once the uterus is relaxed, the infusion rate is maintained at its lowest effective rate for 12-24 hours and then weaned off.

Steer and Petrie (1977) concluded that intravenously administered magnesium sulphate, a 4 g loading dose followed by a continuous infusion of 1-2 g/hr usually arrest labour.

Monitoring of $MgSO_4$ therapy

After the initial loading dose, repeat doses should be given only if

- Patellar reflex is present
- Respiratory rate > 16 / min
- Urine output > 100 ml in the preceding 4 hours.

Auscultation of lung bases and pulse oxymetry also play an important role

Duley et al (1995) in his study showed that there is no need to check serum magnesium levels.

Side Effects

Maternal

- Subjective – flushing, nausea, vomiting, headache, blurred vision, generalised weakness
- Muscular weakness
- Pulmonary congestion
- Respiratory depression
- Neurotoxicity

Elliot's study (1983) of maternal side effects found that side effects occurred in 7% of patients and necessitated stopping treatment in 2% with pulmonary edema occuring in 1% of patients.

The side effect profile is relatively better compared to ritodrine.

Fetal

Maternal levels rapidly equilibrate with fetal plasma and the concentration in both compartments are similar.

It causes transient decrease in FHR variability during labour.

Neonatal

- Respiratory depression
- Hyporeflexia
- Hypotonia
- Low apgar scores

Doyle and co-workers (2009) reported that magnesium exposure is neuroprotective, if received for 24 hours **(RCOG Greentop Guidelines, 2011)** and decreased the risks of cerebral palsy in very low birth weight infants.

Toxicity

Toxicity is however rare in clinical practice, provided the drug is administered with proper monitoring. The first sign of impending toxicity occurs with loss of patellar reflex. The drug must be discontinued until patellar reflex is present.

Signs & Symptoms of Magnesium Toxicity

Blood levels	Signs & Symptoms
4-8 mg / dl	Therapeutic
9-12 mg / dl	Nausea, Warmth, flushing, somnolence, double vision, slurred speech, weakness and loss of patellar reflex
15-17 mg / dl	Muscular paralysis, respiratory arrest
30-35 mg / dl	Cardiac arrest

Pharmacological doses that inhibit myometrial contractions are achieved at levels of 5-8 mg / dl.

Toxicity is also associated with decreased myometrial activity and increased blood loss at delivery.

Management of toxicity

An excellent marker of magnesium toxicity is pulse oxymetry (≤ 96%).

Mild to moderate respiratory depression or neuromuscular toxicity.

- Usually reversible.
- Infusion is discontinued immediately.
- Oxygen should be administered (8 to 10 litres / min).
- Monitor vital signs.
- IV calcium gluconate 10 ml of a 10% solution given over 3 minutes or 1 g iv infused slowly over a period of 3 minutes.

Ca++ antagonises the effect of magnesium by increasing the amount of acetylcholine liberated by the action potentials at the neuromuscular junction.

- Check serum magnesium and creatinine levels.

For severe respiratory depression and arrest prompt tracheal intubation and mechanical ventilation are life saving.

Contraindications

- Myasthenia gravis
- Heart block
- Renal disease
- Recent MI

Distribution

About 40% of plasma magnesium is protein bound. The unbound magnesium ion diffuses into the extravascular space, into bone, skeletal muscle, across the placenta and fetal membranes and into the fetus and amniotic fluid. In pregnant women, apparent volumes of distribution usually reach constant values between the third and fourth hour after administration.

Volume of distribution of magnesium	0.25-0.44 L / Kg
Half life	5 to 6 Hours
Clearance	4.28 L / Hr

Excretion

Magnesium is excreted almost solely by kidneys. 50% of infused dose is excreted after 4 hours in urine. 90% of the bolus intravenous dose is excreted within 24 hours.

Since magnesium is eliminated by kidneys monitoring of urine output is extremely important. Magnesium intoxication is unusual when GFR is maintained.

The initial 4 g loading dose of $MgSO_4$ can be safely administered. Only the maintenance infusion rate should be altered with diminished GFR. If serum creatinine > 1 mg / dl serum magnesium levels are used to adjust the infusion rate.

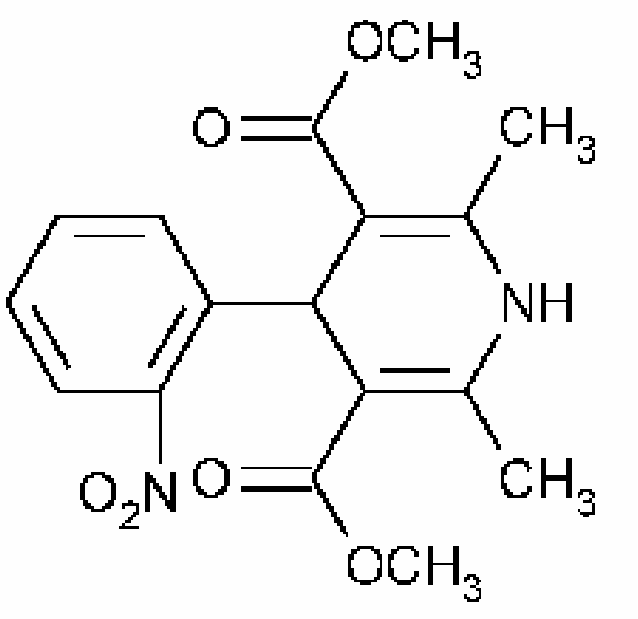
Drug Interactions with Magnesium Sulphate

Agent	Effect	Recommendation
Depolarising / nondepolarising neuromuscular blockers	Increased activity of these agents	May need dosage reduction
CNS depressants	Additive CNS depression	May need dosage reduction
Nifedipine	Hypotension	Administer with caution and adjust nifedipine dosage if necessary

(6) Calcium Channel Blockers

Myometrial activity is directly related to cytoplasmic free calcium and a reduction in its concentration inhibits contraction. Calcium channel blockers act to inhibit, by a variety of mechanisms, the entry of calcium through channels in the cell membrane.

The most common calcium channel blocker used is nifedipine. Nifedipine is a 1,4-dihydropyridine.



Structure of Nifedipine

Mechanism of action

Nifedipine inhibits the influx of extracellular calcium across the cell membrane during inward calcium current of action potential by blocking slow voltage dependent L-type channels regulating calcium influx. It also inhibits the release of intracellular calcium from the sarcoplasmic reticulum. Since less intracellular calcium is available for binding to myosin light chain kinase and

formation of calcium - calmodulin complex, phosphorylation of actin- myosin apparatus is inhibited, thereby reducing the contractility of smooth muscles.

Side Effects

- Dizziness
- Palpitations, tachycardia
- Transient hypotension
- Headache
- Flushing
- Nausea
- Peripheral edema
- Fetal hypoxia

All the adverse effects are a consequence of vasodilatation. Severe side effects are extremely rare **(Hauretty et al., 1989).**

Contraindications

- Hypotension
- Congestive cardiac failure
- Aortic stenosis
- Used with caution in diabetes or multiple pregnancy **(British National Formulatory)**

Nifedipine has been compared to beta agonists in several randomised studies which suggest that calcium antagonists are more effective, better

tolerated with fewer side effects and have lower neonatal morbidity **(King et al., 2003).** Its main advantages include fewer metabolic and cardiovascular complications. No significant change in uteroplacental flow has been reported with nifedipine.

Administration

There is no consensus on dosage or frequency for tocolysis with nifedipine. Commonly recommended protocol consists of

- 20 mg orally stat
- Followed by 20 mg orally after 30 minutes if contractions persist
- Maintenance dose of 10-20 mg orally every 6-8 hours
- Maximum dose is 160 mg / day

Tocolytic regimen given in **Obstetrics and Gynaecology Clinics of North America** is a loading dose of 30 mg orally followed by maintenance dose of 10-20 mg orally every 4-6 hours. BP should be monitored carefully. Sublingual route is not recommended because of risk of sudden hypotension.

Pharmacokinetics

Absorption

Nifedipine is absorbed rapidly from the oral mucosa and from gastrointestinal tract, with detectable levels within minutes of administration.

Bioavailability	30-60%
Onset of action	< 20 minutes
Peak plasma concentration	1 hour (15-90 minutes)
Elimination t½	2-5 hours **(Ferguson et al., 1989)**
Duration of action of a single dose	6 hours

Distribution

Volume of distribution	0.8 L / Kg
Clearance	0.42 L / Hr

Metabolism

More than 90% of the drug is metabolised in liver.

Elimination

Nifedipine is eliminated mainly through kidneys (70%) and bowels (30%) **(Amy et al.).**

The combination of nifedipine with magnesium for tocolysis is potentially dangerous. **Ben Ami and Co-workers (1994) and Kurtzman and associates (1993)** reported that nifedipine enhances neuromuscular blocking effects of magnesium that can interfere with pulmonary and cardiac function. Co-administration of calcium channel blockers with magnesium sulphate can cause hypotension **(John Studd, Vol 17; ACOG, 2003)**.

Lyell and Colleagues (2007) found no substantial differences in efficacy or adverse effects between nifedipine and magnesium sulphate.

Materials and Methods

MATERIALS AND METHODS

STUDY DESIGN

This is a prospective randomised controlled trial, conducted in the labour ward of Mahatma Gandhi Memorial Government Hospital, Trichy between July 2011 and June 2012.

The study population comprised of 100 patients, randomly assigned to intravenous magnesium sulphate and another 100 patients assigned to oral nifedipine. Both the groups had bed rest and received intramuscular corticosteroids. All the patients were counselled and informed consent was obtained.

INCLUSION CRITERIA

1. Gestational age between 28 and 34 weeks as determined by last menstrual period, clinical examination and USG.
2. Uterine contractions - 4 contractions in 20 minute period, each contraction lasting for 40 seconds.
3. Progressive cervical effacement ≤ 75%
4. Cervical dilatation ≤ 3cm
5. Intact membranes

EXCLUSION CRITERIA

Maternal Factors

1. Ruptured membranes
2. Infection / chorioamnionitis
3. Cervical dilatation > 3cm
4. Antepartum haemorrhage
5. Hypertensive disorders of pregnancy
6. Disorders of amniotic fluid volume
7. Previous caesarean section
8. Cardiac disease
9. Renal disease
10. Pulmonary disorders – Asthma, ARDS
11. Uncontrolled diabetes mellitus
12. Hyperthyroidism
13. Any contraindication for use of nifedipine or magnesium
14. Presence of cerclage
15. Tocolytic use within the last 12 hours
16. Hypotension (defined as average BP of < 90/60 mm Hg unresponsive to 1000cc of fluid bolus)

Fetal Factors

1. Fetal death
2. Nonreassuring fetal heart tracing

3. Lethal congenital or chromosomal abnormalities
4. Fetal growth restriction
5. Erythroblastosis fetalis
6. Multiple pregnancy

INVESTIGATIONS

1. Urine Analysis
2. Complete blood count
3. Blood Sugar
4. Renal function tests

   (Blood urea, Serum creatinine)
5. Vaginal swab
6. ECG
7. CTG
8. USG

DRUG PROTOCOL

In all the patients detailed obstetric and medical history were elicited. They were asked to lie in left lateral position. Vital parameters (pulse rate, blood pressure, respiratory rate and temperature) were recorded. Cardiovascular and respiratory system were examined. Detailed obstetric examination was done.

Side effects of both the drugs were documented. Maternal outcome till discharge was noted.

Group A

Intravenous Magnesium Sulphate

Loading Dose

4g of 20% Mg So_4 given intravenously over 5-10 minutes, at a rate not exceeding 1g/minute.

Maintenance Dose

This is followed by an infusion of 1g/hour in 50ml of NS by controlled infusion pump.

Once the uterus is relaxed, the infusion is maintained for 12 hours and then weaned off. The incidence of pulmonary edema is less if the total volume of intravenous infusion is limited to < 1000ml / day

Monitoring

After the initial loading dose, treatment is discontinued if

- Patellar reflex is absent
- Respiratory rate < 16/min
- Urine output < 100ml in the preceding 4 hours
- Oxygen saturation $\leq 96\%$

All these parameters were monitored every 2 hours. Uterine contractions were monitored every 30 minutes for the first 2 hours, then every 4 hours for 48 hours.

Magnesium sulphate treatment was defined as **effective** if the uterine contractions were reduced more than 30% in frequency and intensity compared to those occurring during the last two hours before magnesium sulphate infusion. Treatment was defined as **ineffective** if uterine contractions were not reduced by 30% and labour progressed with apparent changes in cervical findings.

Group B

Oral Nifedipine

Tablet Nifedipine 20mg orally stat was given. This was followed by 20mg orally after 30 minutes if contractions persist. Maintenance dose of 10mg orally 8th hrly was continued for 48hours.

Monitoring

Maternal	**Fetal**
Pulse Rate	Hearth Rate
Blood Pressure	Rhythm
Uterine Contractions	Tone

All were monitored every 30 minutes for the first 2 hours, then every 4 hours for 48 hours.

Treatment is discontinued if

- Systolic BP < 100 mmHg
- Pulse rate > 100 b/min
- Fetal heart rate > 180 b/min

Steroids

Given for both group A & Group B

Betamethasone

2 doses of 12mg, given im 24 hours apart.

Prophylactic antibiotics were also given for both the groups

RESPONSE TO TOCOLYTIC THERAPY

Definition of Success

Various authors have considered several factors for assessing the success of tocolysis.

Successful tocolysis was defined when uterine contractions subsided and delivery was delayed for more than 48 hours.

Definition of failure

Failure of tocolysis was defined as persistent uterine contractions, despite maximum dose and delivery occurring in the first 48 hours of initiation of therapy.

This study confines itself to idiopathic spontaneous preterm labour, comparing the safety and efficacy of intravenous magnesium sulphate with that of oral nifedipine in acute tocolysis for corticosteroid therapy and to study the maternal and fetal effects.

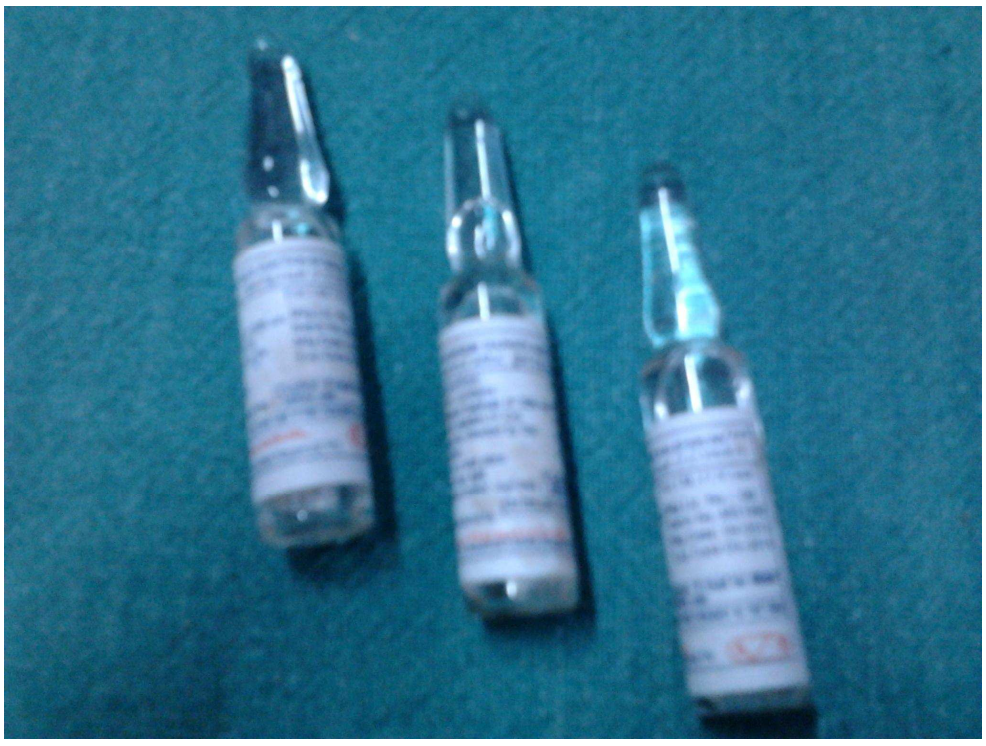
All the data were entered prospectively in a predefined data information sheet and analysed. The Statistical Package for the Social Sciences (SPSS) was used for analysis of all data.

Difference is categorical and continuous data were assessed using the chi square test and students ‘t’ test respectively.

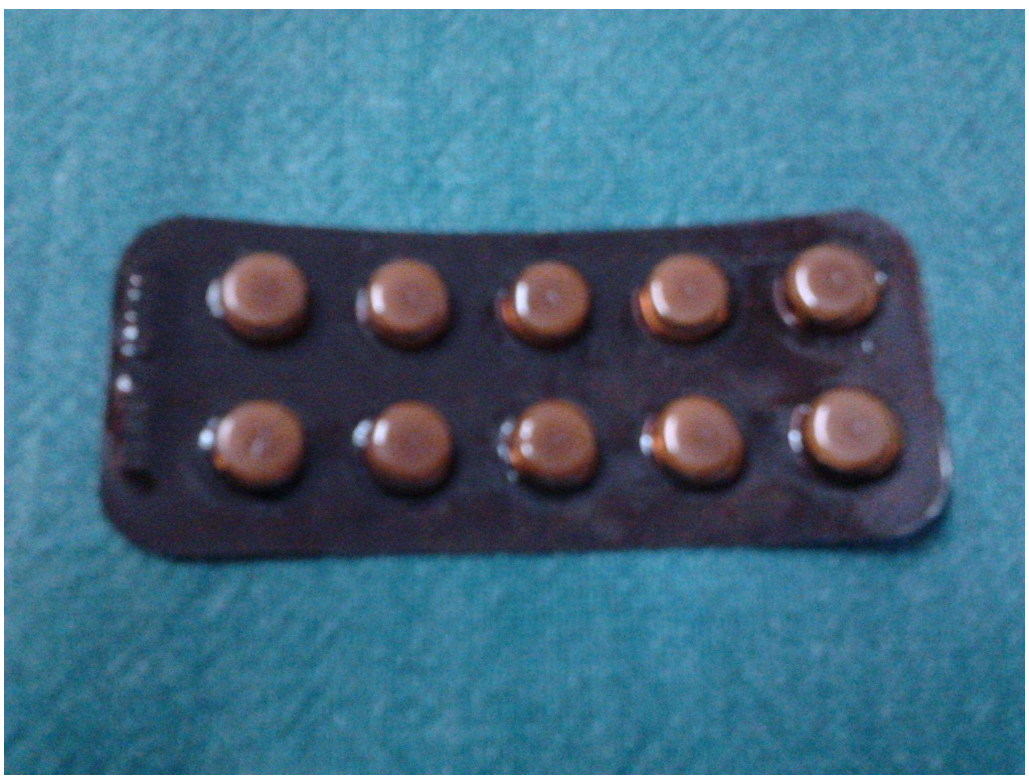
p – values

When we compare the calculated probability (p) with the level of significance, if it is less than 0.05 ($p < 0.05$) or 0.01 ($p < 0.01$), there is significant difference between the two sample populations. If the calculated p is equal to or greater than 0.05 ($p \geq 0.05$) or 0.01 ($p \geq 0.01$), there is no significant difference between the two. A statistical test is said to be significant if the p-value is less than the significance level (0.05 or 0.01).

GROUP A



GROUP B



Analysis of Results

ANALYSIS OF RESULTS

TABLE – 1

AGE DISTRIBUTION

Age (Yrs)	Group A		Group B	
	Number	%	Number	%
< 20	7	7%	7	7%
20 – 24	45	45%	43	43%
25 – 29	34	34%	35	35%
≥ 30	14	14%	15	15%
Total	100	100%	100	100%
Statistical Value	χ^2 =27.27		χ^2 =59.89	
	t = 2.77 p = 0.487			

There was no significant difference in the age distribution of both the groups of patients.

Majority of patients (44%) fell into the age group of 20-24 years.

7% of patients fell into the age group of < 20 years and 14.5% into the age group of ≥ 30years on an average.

The study subjects had a mean age of 23.2 years.

AGE DISTRIBUTION

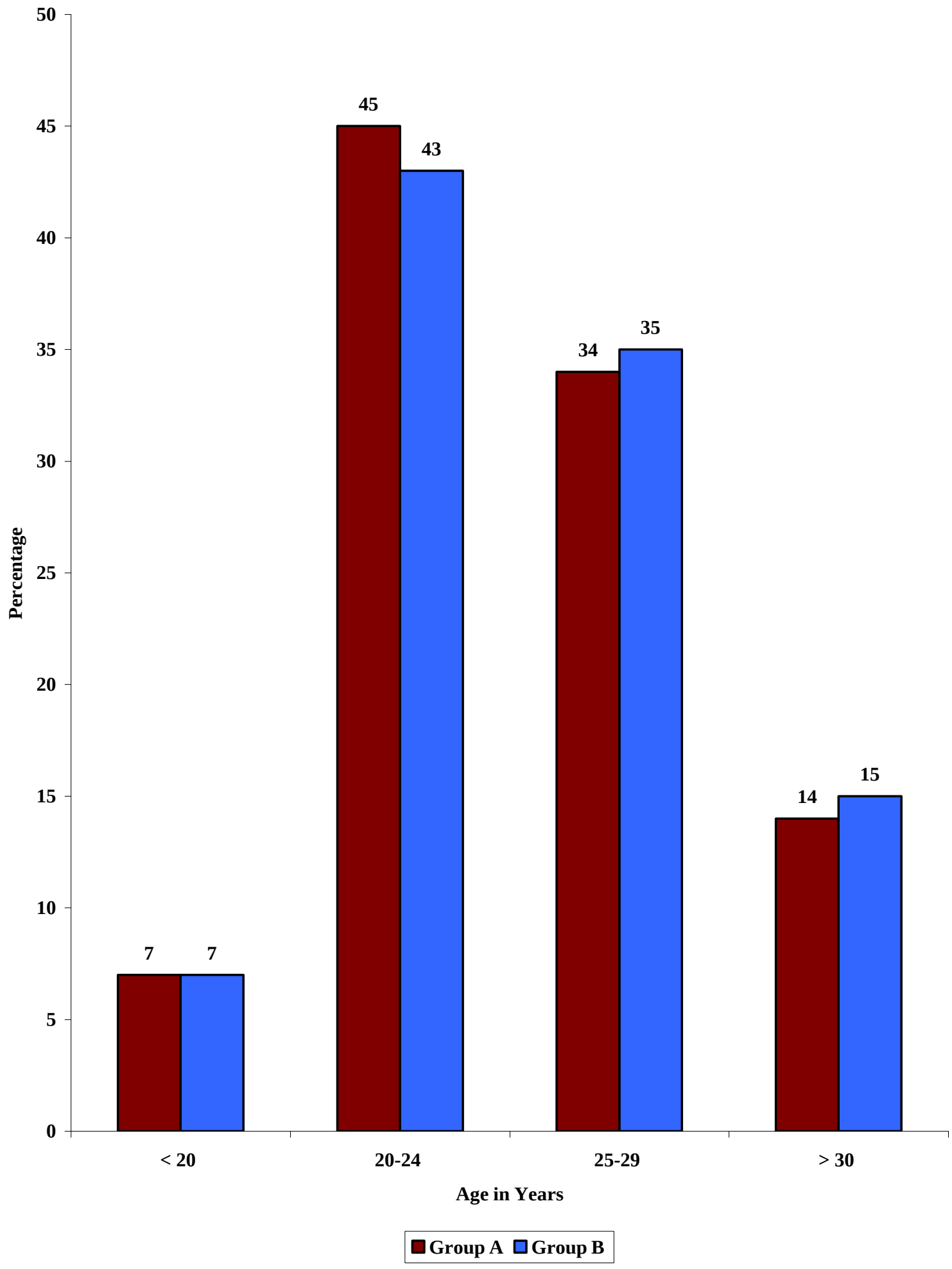


TABLE - 2

SOCIOECONOMIC STATUS

Socioeconomic Status	Group A		Group B	
	Number	%	Number	%
V	59	59%	62	62%
IV	24	24%	25	25%
III	17	17%	13	13%
Total	100	100%	100	100%
Statistical Value	χ^2 =18.43		χ^2 =23.11	
	t = 2.54 p = 0.126			

None of the patients in this study belonged to class I and II socioeconomic status.

On an average, majority (60.5%) of the patients belonged to Class V socioeconomic status. The rest of them belonged to class IV and class III socio-economic status. There was no significant difference between the groups.

Low socioeconomic status is one of the important demographic factors associated with high risk of preterm labour.

SOCIOECONOMIC STATUS

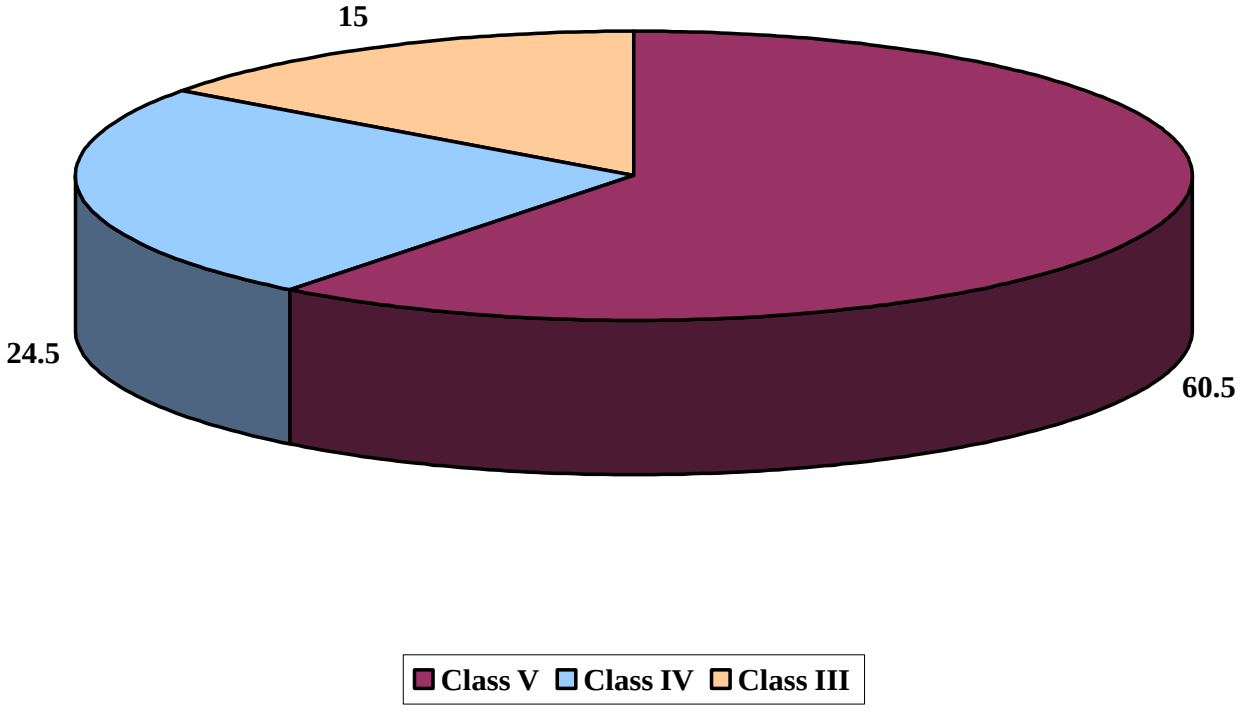


TABLE - 3

OBSTETRIC CODE

Obstetric Code	Group A		Group B	
	Number	%	Number	%
Primi	60	60%	54	54%
G_2	30	30%	24	24%
G_3	7	7%	16	16%
G_4	3	3%	6	6%
Total	100	100%	100	100%
Statistical Value	SD = 67.88		SD =66.46	
	t = 1.063 p = 0.480			

Parity was comparable in both the groups of patients. There was no significant difference.

60% of patients in group A and 54% of patients in group B were primigravida. 40% of patients in group A and 46% of patients in group B were multigravida.

There is a higher incidence of preterm labour in first pregnancies.

OBSTETRIC CODE

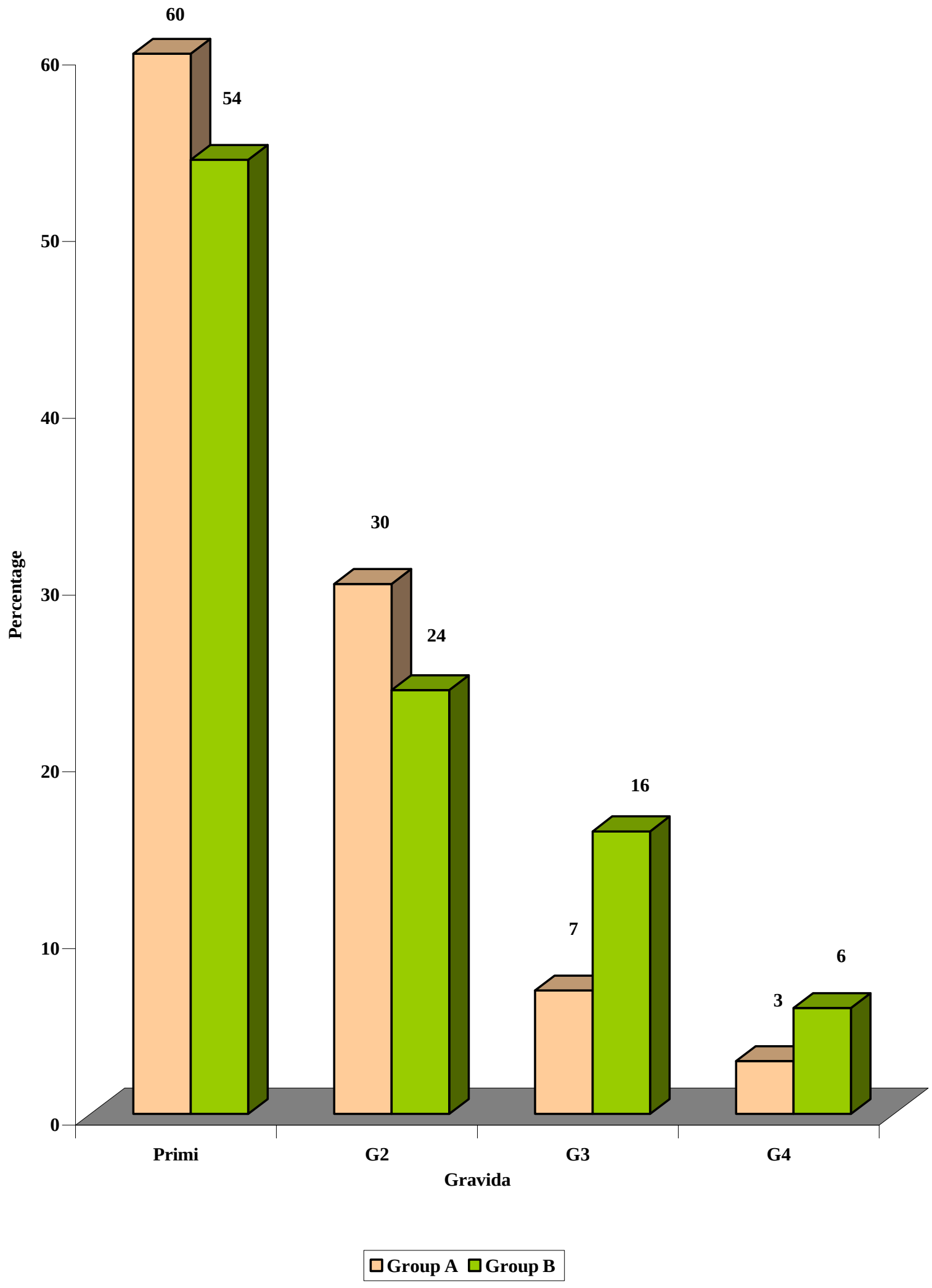


TABLE - 4

BOOKING STATUS

Booking Status	Group A		Group B	
	Number	%	Number	%
Booked	43	43%	46	46%
Unbooked	57	57%	54	54%
Total	100	100%	100	100%
Statistical Value	SD = 66.85		SD =63.47	
	t = 1.20 p = 0.312			

Antenatal booking status did not differ significantly among both the groups of patients.

57% of patients in Group A and 54% in Group B were unbooked. In patients with no antenatal care preterm labour was more common.

BOOKING STATUS

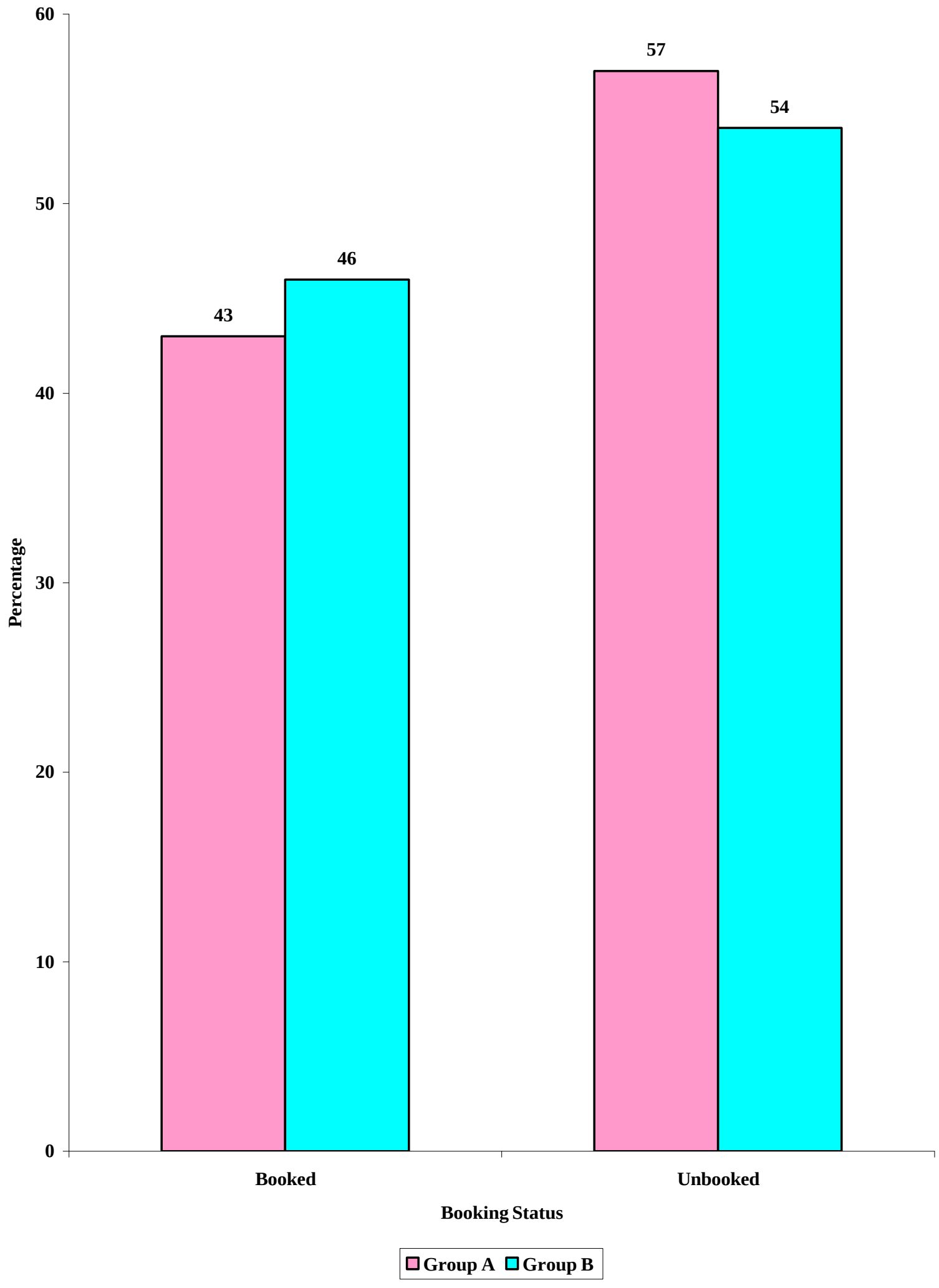


TABLE - 5

GESTATIONAL AGE

Gestational Age	Group A		Group B	
	Number	%	Number	%
28 – 30 wks	26	26%	29	29%
31 – 32 wks	35	35%	29	29%
33 – 34 wks	39	39%	42	42%
Total	100	100%	100	100%
Statistical Value	χ^2 =18.43		χ^2 =49.66	
	t = 1.93 p = 0.03			

Treatment delivery interval varied significantly with gestational age at the time of admission in both the groups of patients.

On an average, majority of patients in both the groups (40.5%) had a gestational age of 33-34 weeks, with a better neonatal outcome. 32% of patients belonged to gestational age group of 31-32 weeks. 27.5% of patients belonged to gestational age group of 28-30 weeks.

Late preterm labour is more common than early preterm labour.

GESTATIONAL AGE

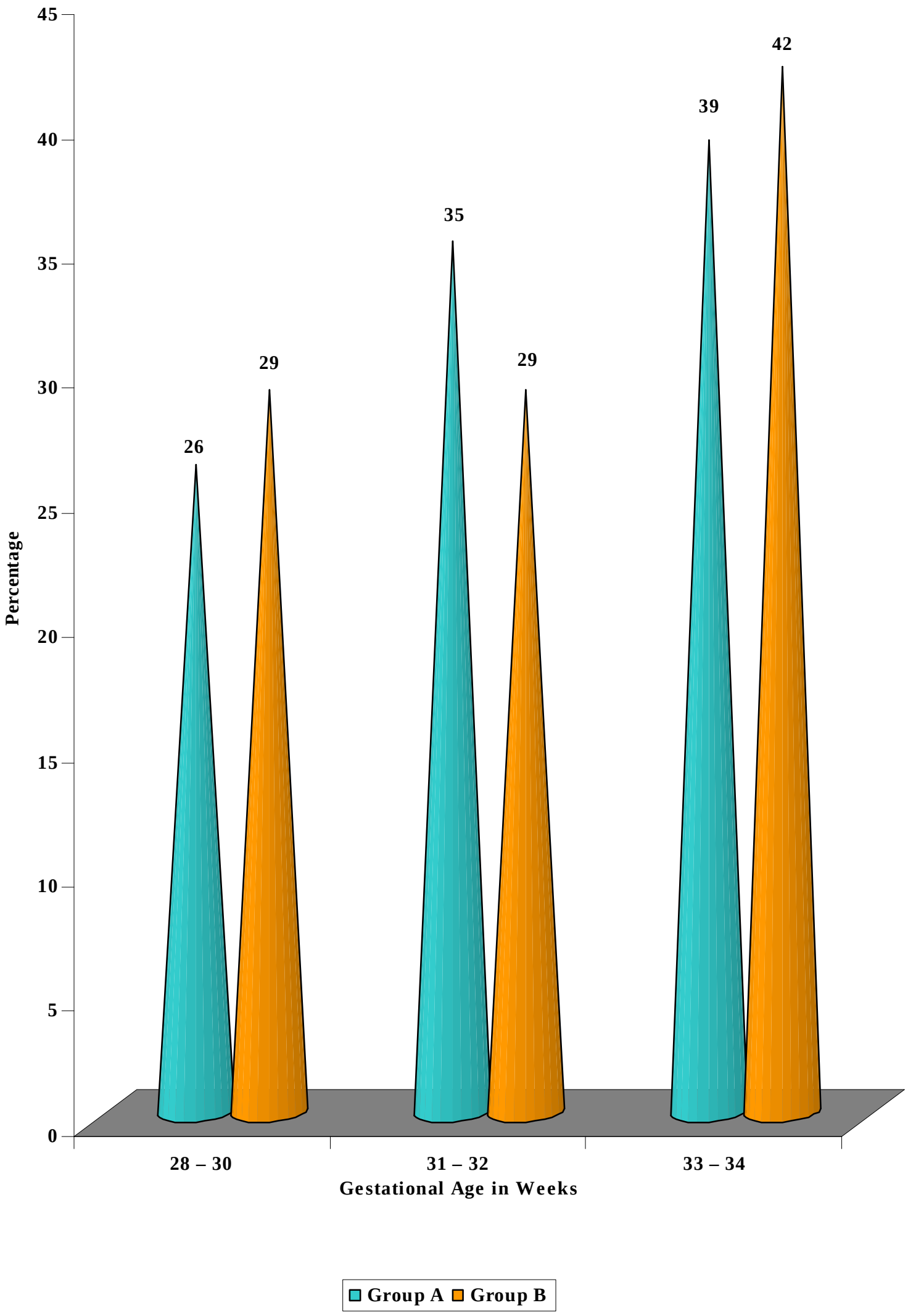


TABLE - 6

PAST OBSTETRIC HISTORY

Past Obstetric History	Group A		Group B	
	Number	%	Number	%
History of preterm labour	9	9%	8	8%
History of 2nd trimester abortion	6	6%	4	4%
Statistical Value	SD = 20.84		SD = 22.72	
	t = 12.66 p = 0.300			

Majority of patients in both the groups were primigravida and experienced preterm labour for the first time. There was no significant difference in the past obstetric history among both the groups of patients.

PAST OBSTETRIC HISTORY

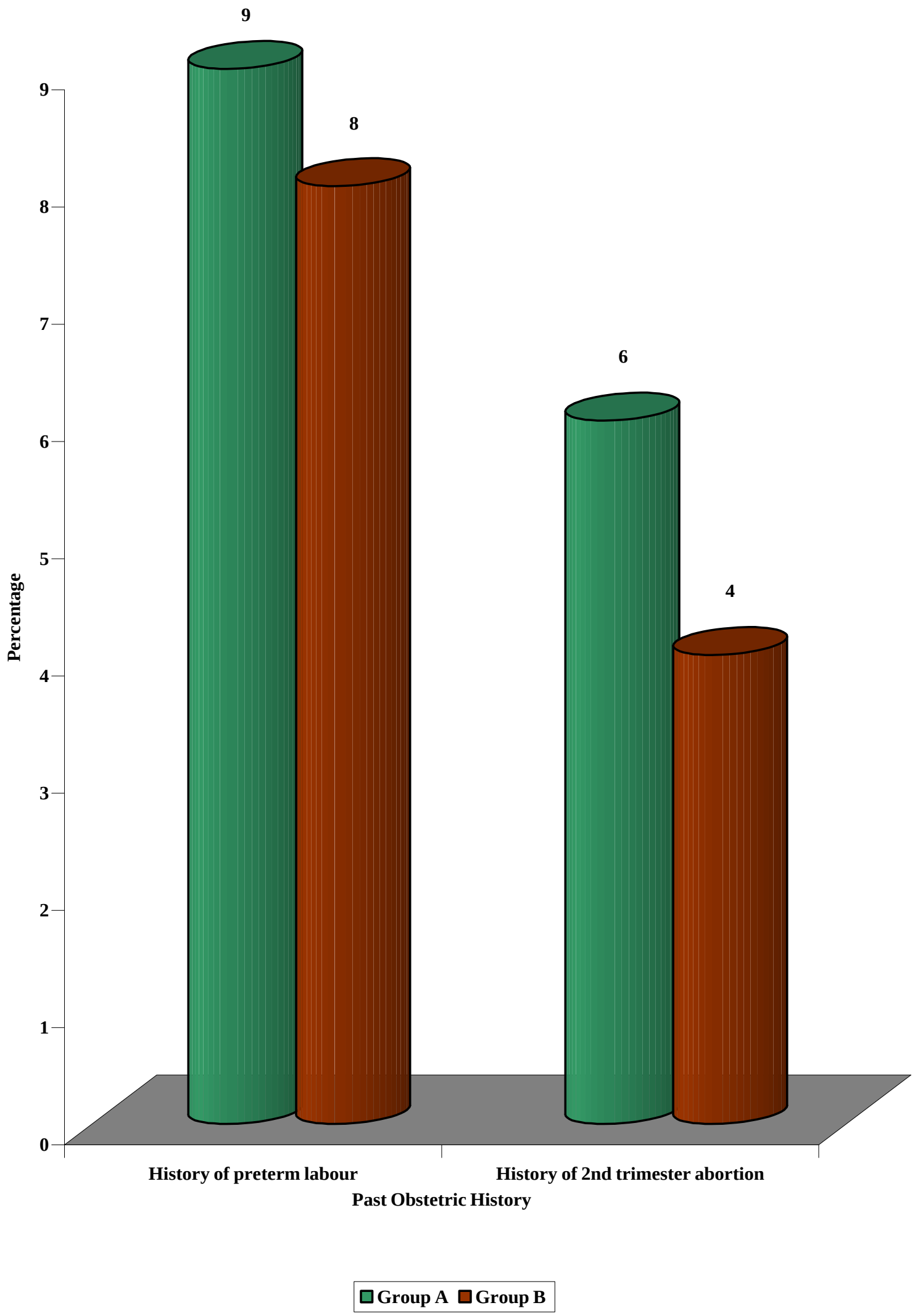


TABLE - 7

MATERNAL SIDE EFFECTS

Side Effects	Group A		Side Effects	Group B	
	Number	%		Number	%
Flushing	21	21%	Headache	14	14%
Headache	09	09%	Dizziness	9	9%
Nausea / Vomiting	09	09%	Palpitations	5	5%
Drowsiness	11	11%	Hypotension	2	2%
Pulmonary Edema	02	02%	Nausea / Vomiting	5	5%
Total	52	52%	Total	35	35%
Statistical Value	t = 2.48			t = 2.487	

Group A

Flushing was the commonest side effect in patients who received intravenous magnesium sulphate. Majority of patients in this group developed minor side effects. Two patients developed pulmonary edema, requiring treatment discontinuation and both delivered at <48 hours.

None of the patients developed signs and symptoms of toxicity. No case of postpartum haemorrhage was observed in those who delivered within 48 hours.

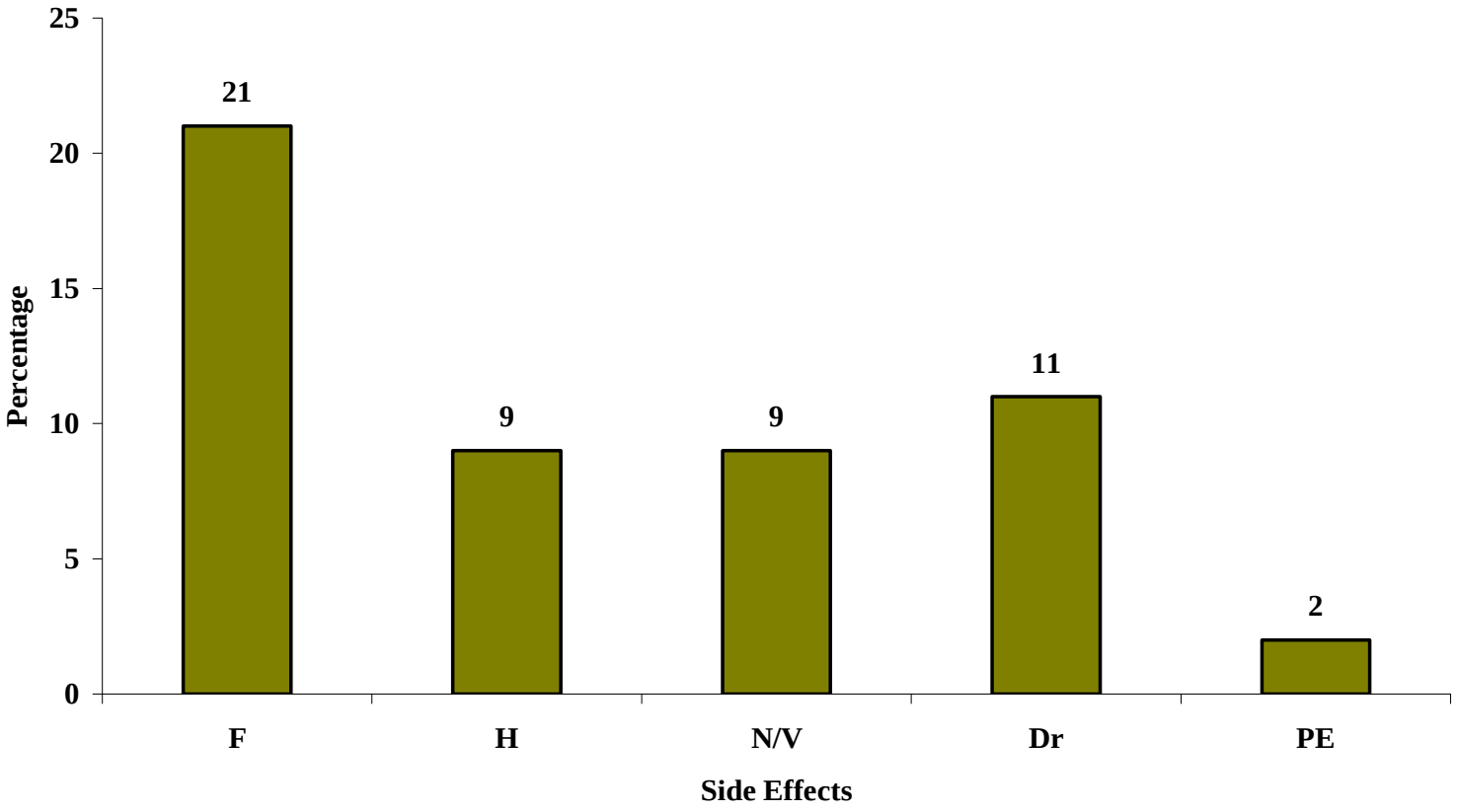
Group B

No severe adverse effects were noted except hypotension that developed in 2 patients requiring treatment discontinuation and one among them delivered at < 48 hrs.

There was **no maternal mortality** among both the groups of patients.

MATERNAL SIDE EFFECTS

Group A



Group B

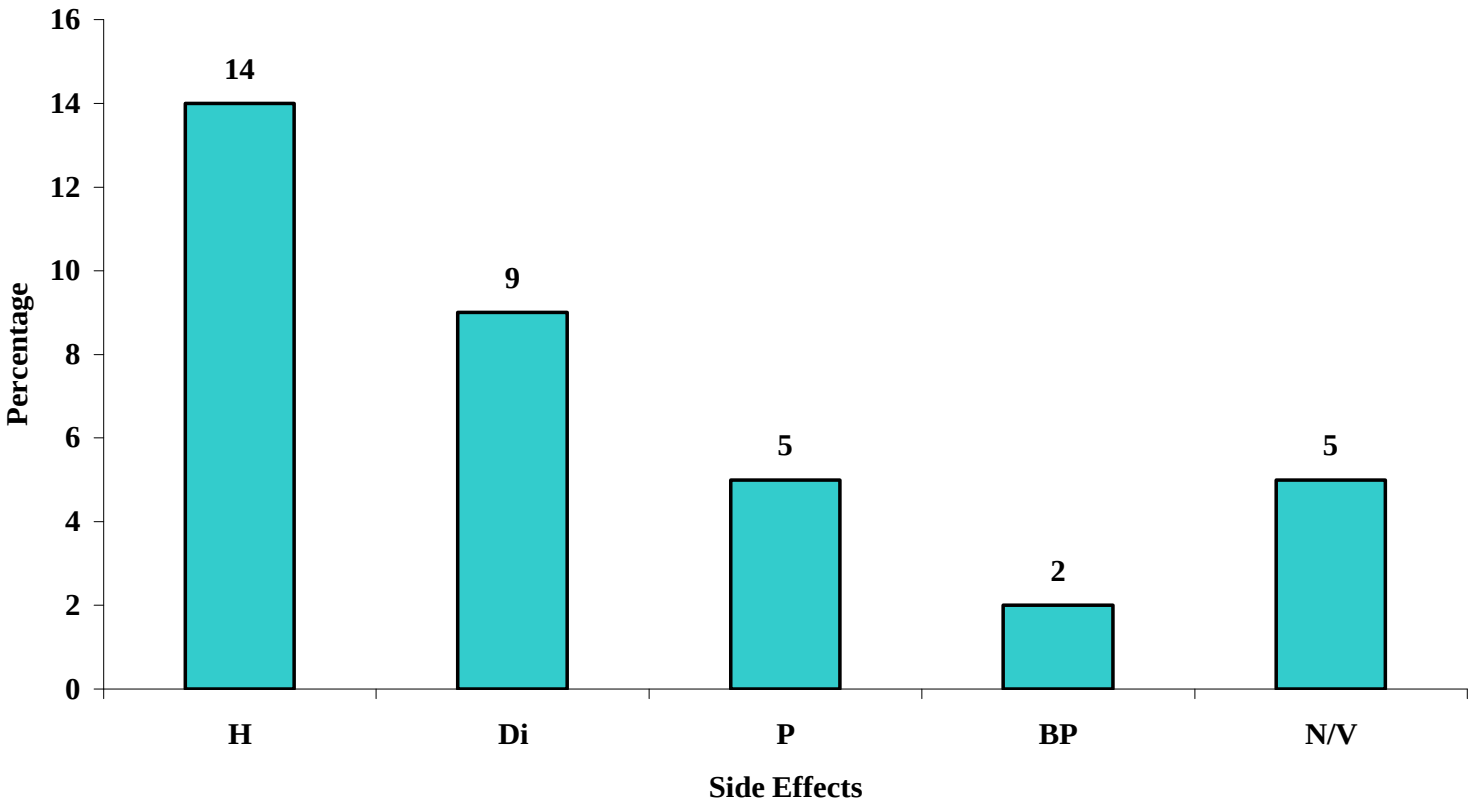


TABLE - 8

PRESENTATION

Presentation	Group A		Group B	
	Number	%	Number	%
Cephalic	85	85%	87	87%
Breech	13	13%	11	11%
Transverse Lie	2	2%	02	02%
Total	100	100%	100	100%
Statistical Value	SD = 85.10		SD = 67.88	
	t = 10.40	p = 0.487		

Majority of patients in both the groups were in cephalic presentation.

PRESENTATION

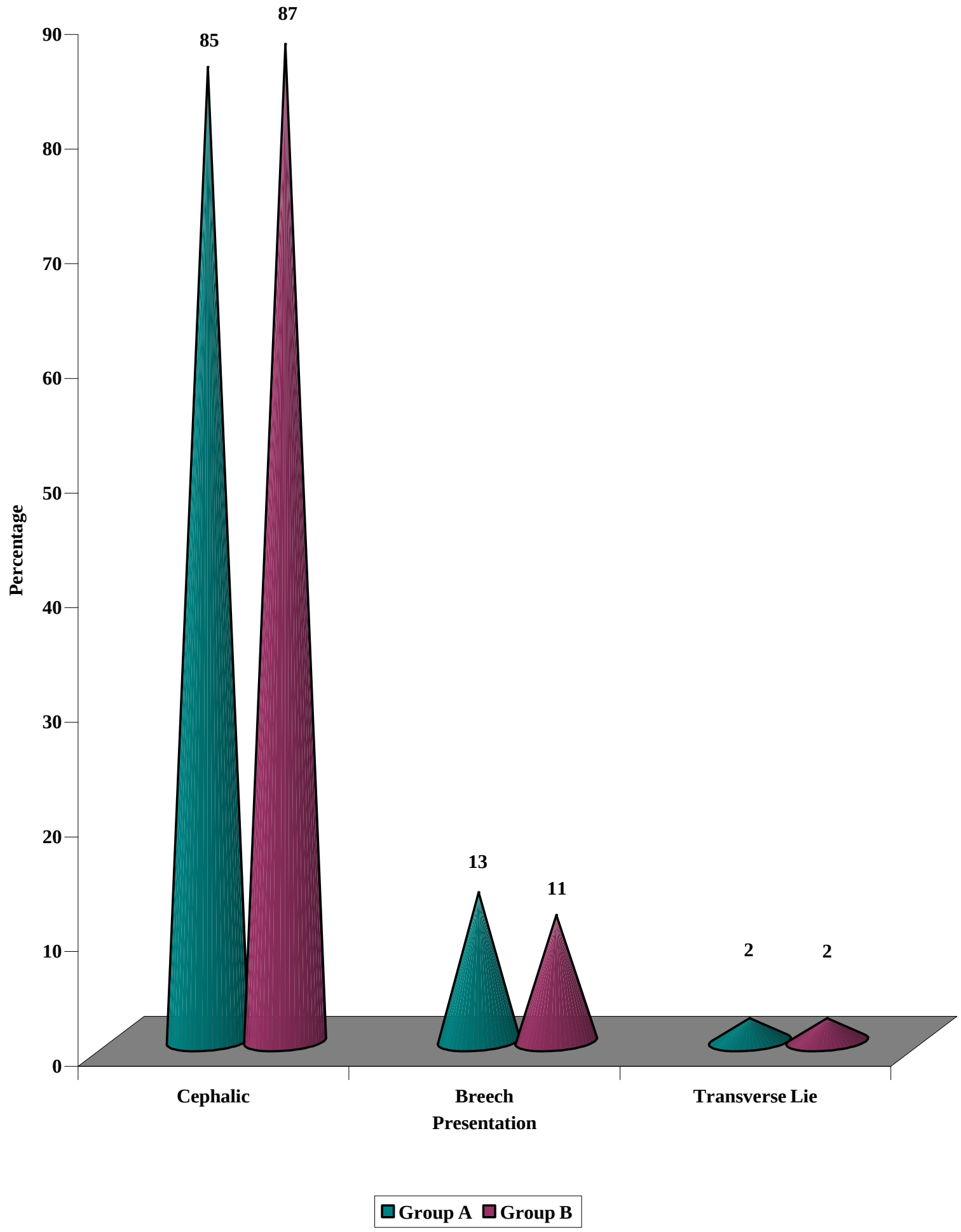


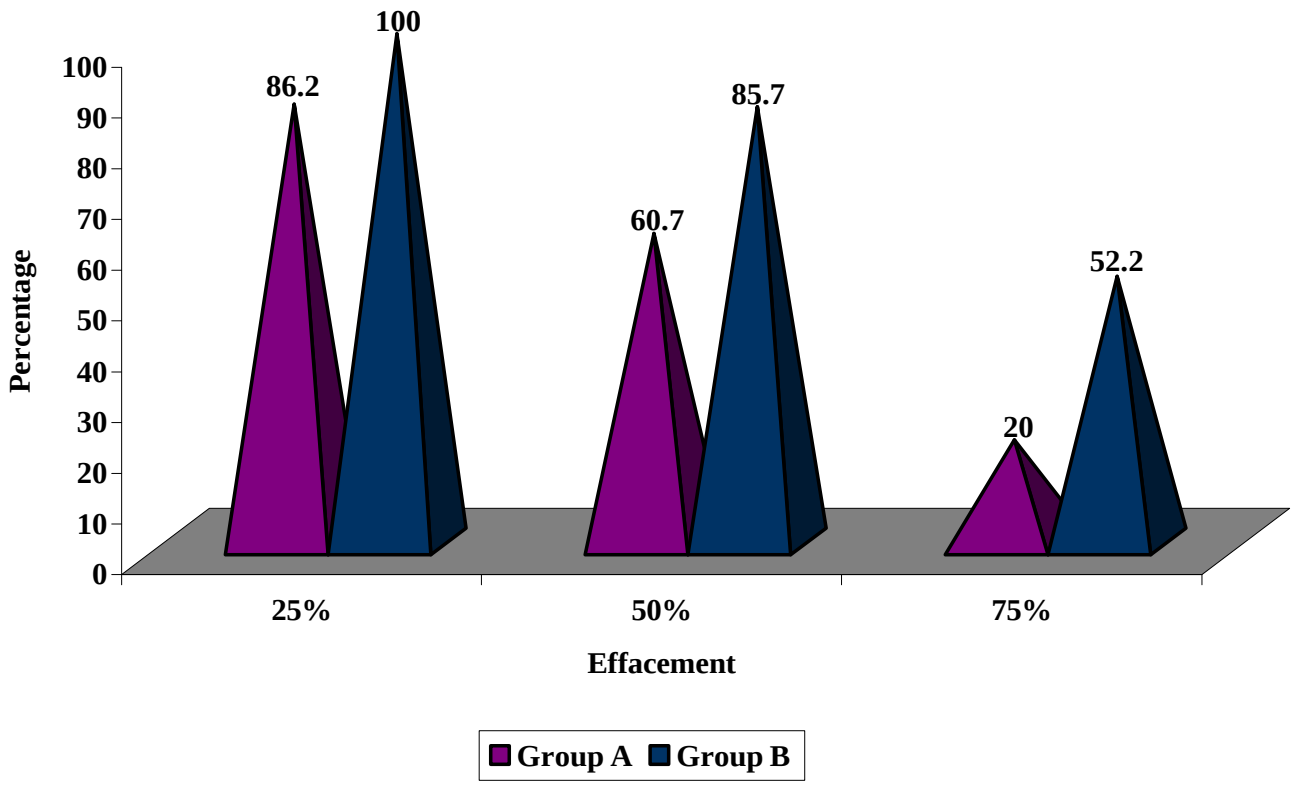
TABLE - 9

CERVICAL EFFACEMENT

Cervical Effacement	Group A			Group B		
	Total No.	Success	Failure	Total No.	Success	Failure
25%	29	25(86.2%)	4(13.8%)	35	35(100%)	-
50%	56	34(60.7%)	22(39.3%)	42	36(85.7%)	6(14.3%)
75%	15	3(20%)	12(80%)	23	12(52.2%)	11(47.8%)
Total	100	62	38	100	83	17
Statistical Value	SD = 25.71 p = 0.0031					

In patients with 25% cervical effacement, 86.2% of patients in Group A and 100% of patients in Group B had successful tocolysis. With 50% cervical effacement, prolongation of pregnancy was observed in 60.7% of patients in Group A and 85.7% of patients in Group B. In patients with 75% cervical effacement 20% of patients in Group A and 52.2% in group B had their pregnancy prolonged by more than 48 hours, which is statistically highly significant (p = 0.0031).

CERVICAL EFFACEMENT IN SUCCESS GROUP



CERVICAL EFFACEMENT IN FAILURE GROUP

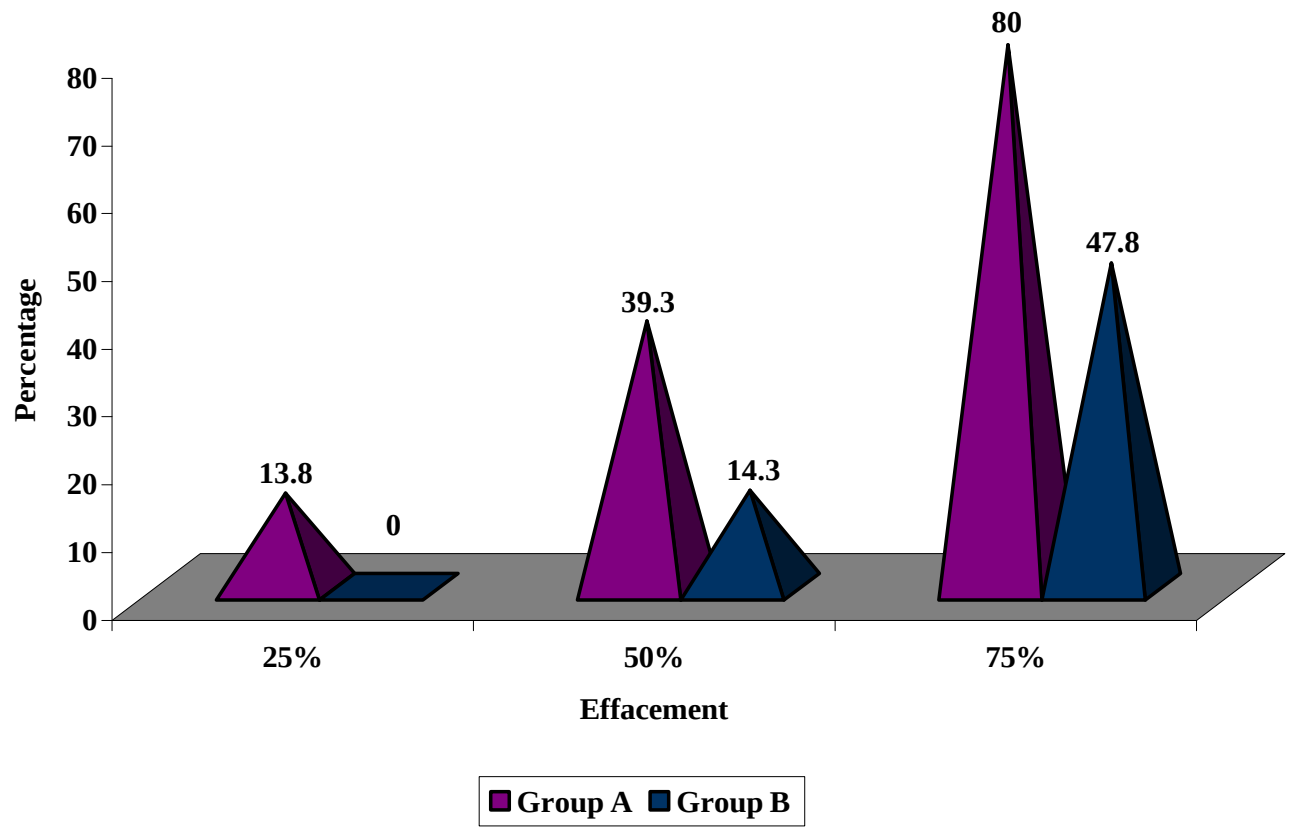


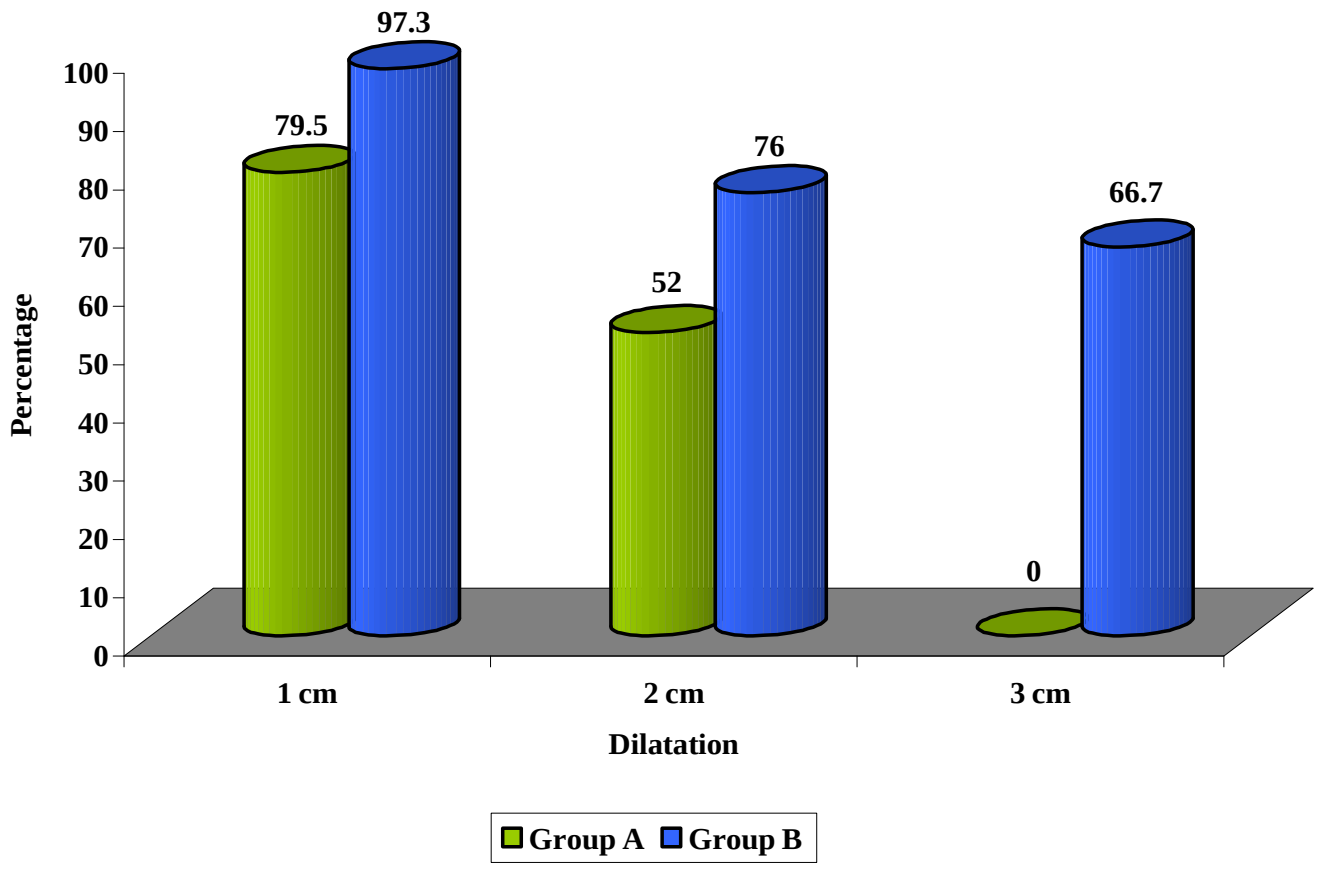
TABLE - 10

CERVICAL DILATATION

Cervical Dilatation	Group A			Group B		
	Total No.	Success	Failure	Total No.	Success	Failure
1cm	44	35(79.5%)	9(20.5%)	37	36(97.3%)	1(2.7%)
2 cm	52	27(52%)	25(48%)	54	41(76%)	13(24%)
3 cm	4	-	4(100%)	9	6(66.7%)	3(33.3%)
Total	100	62	38	100	83	17
Statistical Value	SD = 12.02		p = 0.0012			

When the cervix was 1cm dilated 79.5% of patients in group A and 97.3% of patients in group B had successful tocolysis. With 2cm cervical dilatation successful tocolysis was observed in 52% in group A and 76% in group B. And when cervix was 3cm dilated prolongation of pregnancy > 48hrs was observed in none of the patients in group A and 66.7% of patients in group B which is statistically highly significant (p = 0.0012).

CERVICAL DILATATION IN SUCCESS GROUP



CERVICAL DILATATION IN FAILURE GROUP

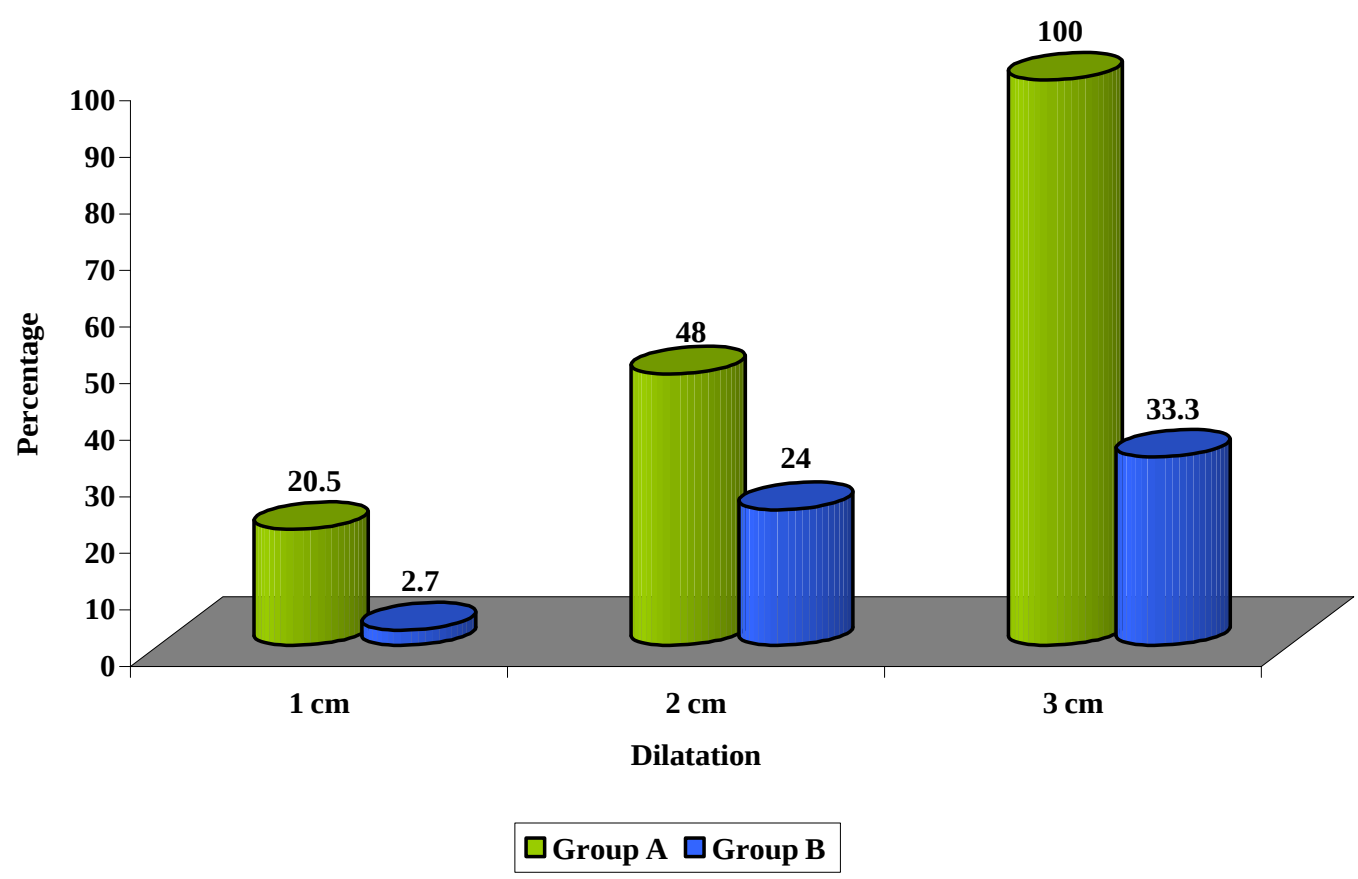


TABLE - 11

SUCCESS OF ACUTE TOCOLYSIS

Duration	Group A		Group B	
	Number	%	Number	%
< 48 hrs	38	38%	17	17%
> 48 hrs	62	62%	83	83%
Total	100	100%	100	100%
Statistical Value	χ^2 =19.36		χ^2 =21.36	
	t = 21.32 p = 0.003			

Acute tocolysis was successful in 62% of patients in group A and 83% of patients in group B, which is statistically highly significant (p = 0.003)

SUCCESS OF ACUTE TOCOLYSIS

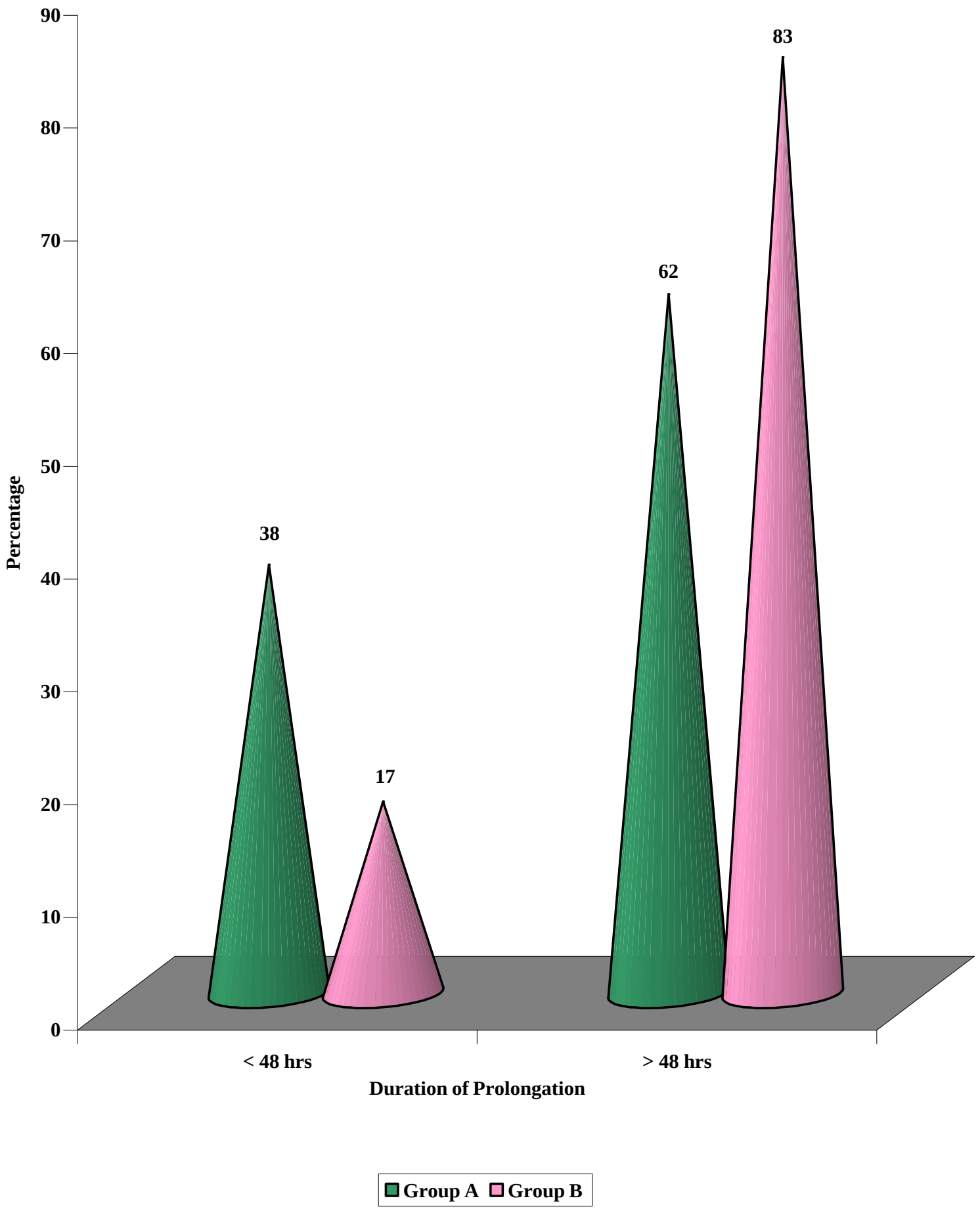


TABLE - 12

DURATION OF PROLONGATION

Prolongation Period	Group A		Group B	
	Number	%	Number	%
< 48 hrs	38	38%	17	17%
48 - 72 hrs	28	28%	31	31%
72 hrs - 1 wk	21	21%	34	34%
1 -2 wks	10	10%	12	12%
> 2 wks	3	3%	6	6%
Total	100	100%	100	100%
Statistical Value	χ^2 =61.76		χ^2 =62.06	
	t = 13.65 p = 0.000			

Among the success group, prolongation of pregnancy beyond 72 hrs was observed in 21% in group A and 34% in group B. Prolongation beyond 1 week was observed in 10% in group A and 12% in group B, beyond 2 weeks in 3% in group A and 6% in group B which is statistically highly significant (p=0.000).

Mean duration of prolongation of pregnancy was 1.8 days and 4.29 days in group A and group B respectively.

DURATION OF PROLONGATION

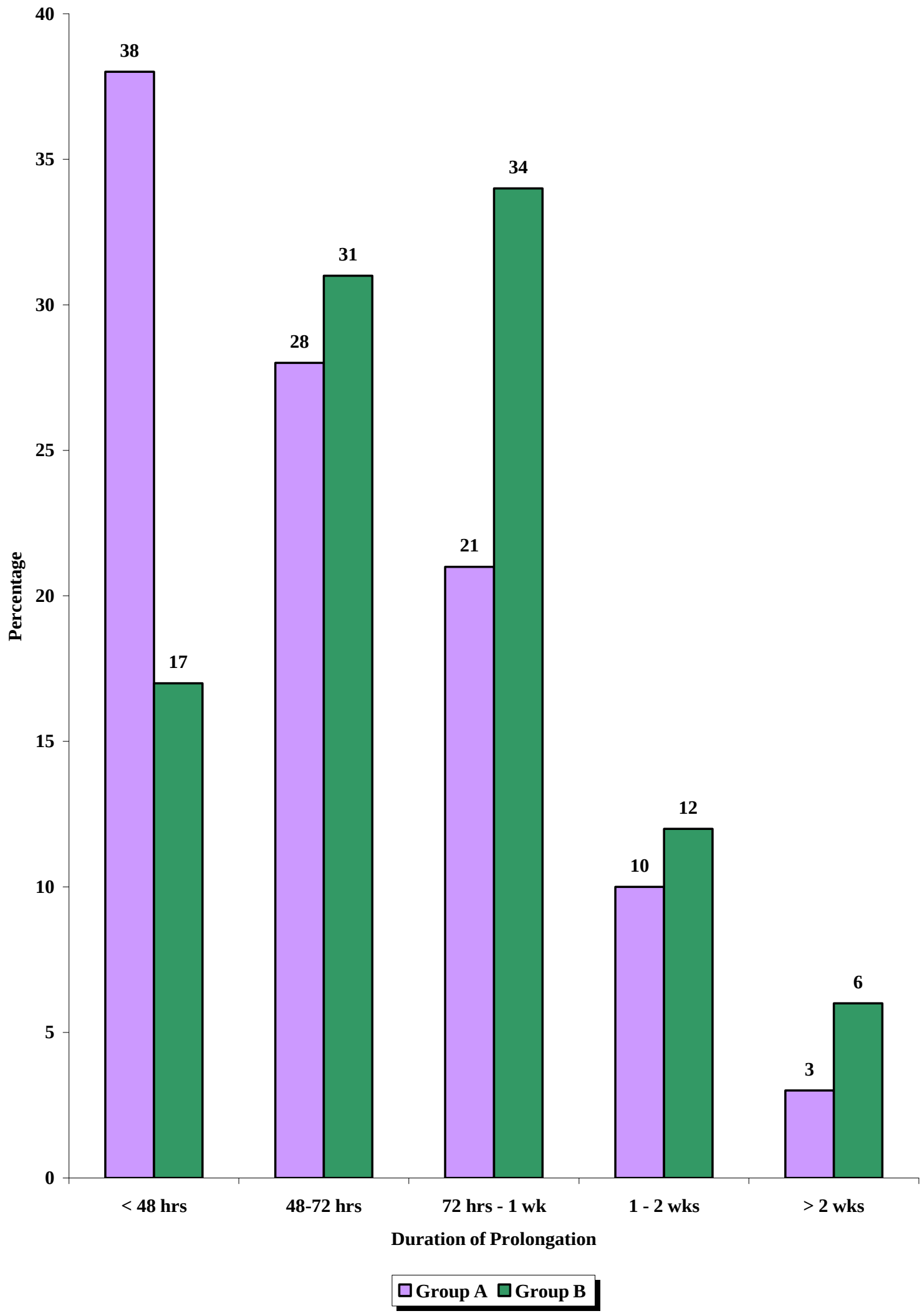


TABLE - 13

PROLONGATION OF PREGNANCY

Gestational Age	Group A		Group B	
	Number	%	Number	%
< 37 wks	97	97%	94	94%
> 37 wks	3	3%	6	6%
Total	100	100%	100	100%
Statistical Value	χ^2 =66.36		χ^2 =65.66	
	t = 19.43 p = 0.000			

Prolongation of pregnancy beyond 37 weeks was observed in 3% of patients in group A and 6% of patients in group B, which is statistically highly significant (p = 0.000).

PROLONGATION OF PREGNANCY

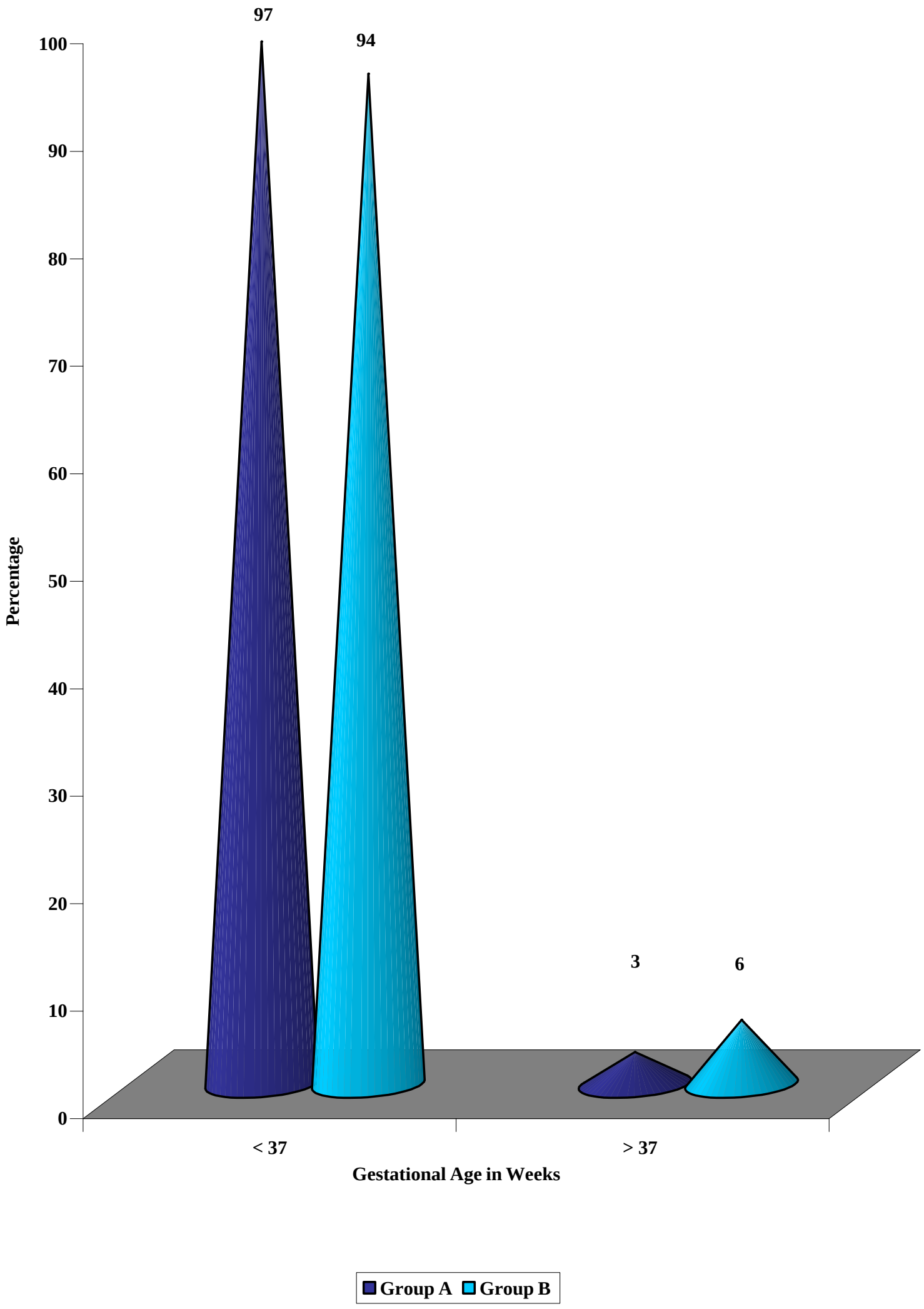


TABLE - 14

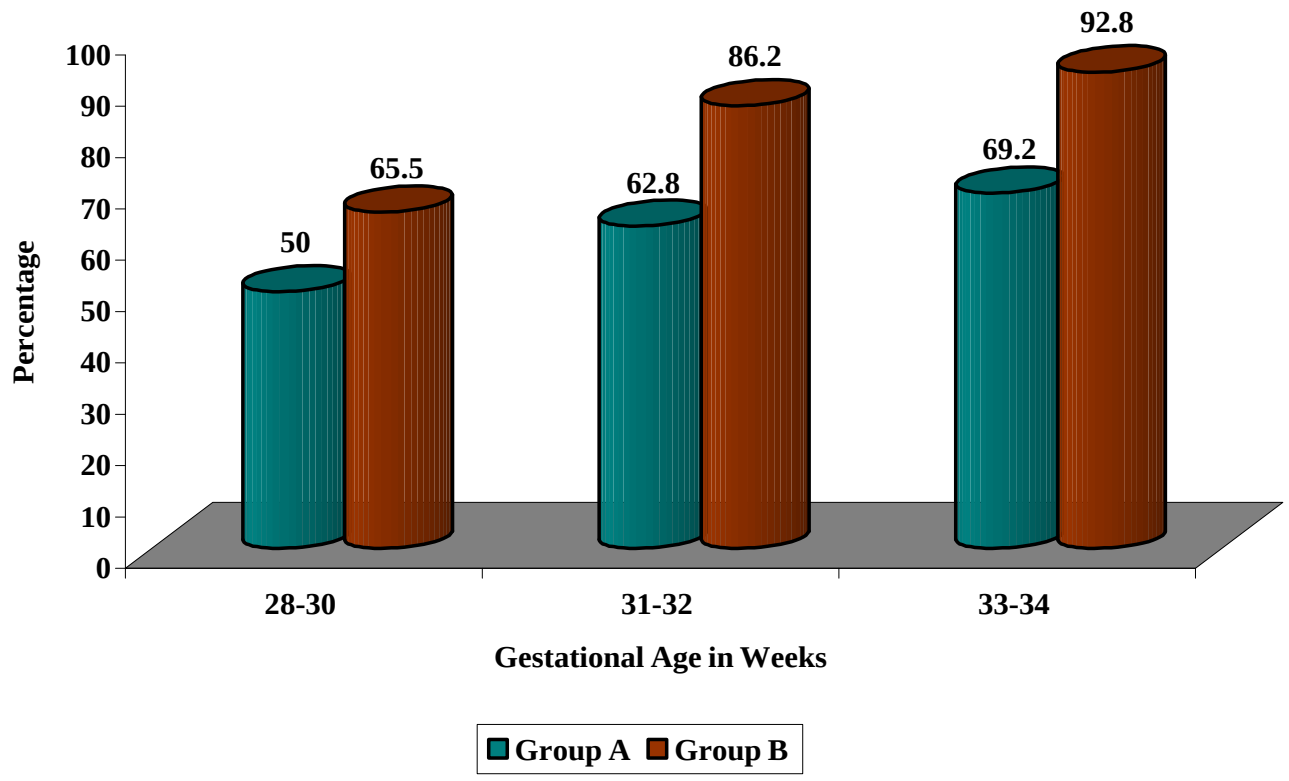
RESPONSE ACCORDING TO GESTATIONAL AGE

Gestational Age	Group A			Group B		
	Number	Success	Failure	Number	Success	Failure
28-30 wks	26	13(50%)	13(50%)	29	19(65.5%)	10(35.5%)
31-32 wks	35	22(62.8%)	13(37.2%)	29	25(86.2%)	4(13.8%)
33-34 wks	39	27(69.2%)	12(30.8%)	42	39(92.8%)	3(7.2%)
Total	100	62	38	100	83	17
Statistical Value	SD = 12.44	χ^2 =16.32		SD = 12.44	χ^2 =21.44	
	p = 0.003			t=1.872		

Between 33 and 34 weeks of gestation tocolysis was successful in 69.2% of patients in group A and 92.8% of patients in group B which is statistically highly significant (p = 0.003).

Even at earlier gestational age it was successful in more number of patients in group B, when compared to group A.

RESPONSE ACCORDING TO GESTATIONAL AGE IN SUCCESS GROUP



RESPONSE ACCORDING TO GESTATIONAL AGE IN FAILURE GROUP

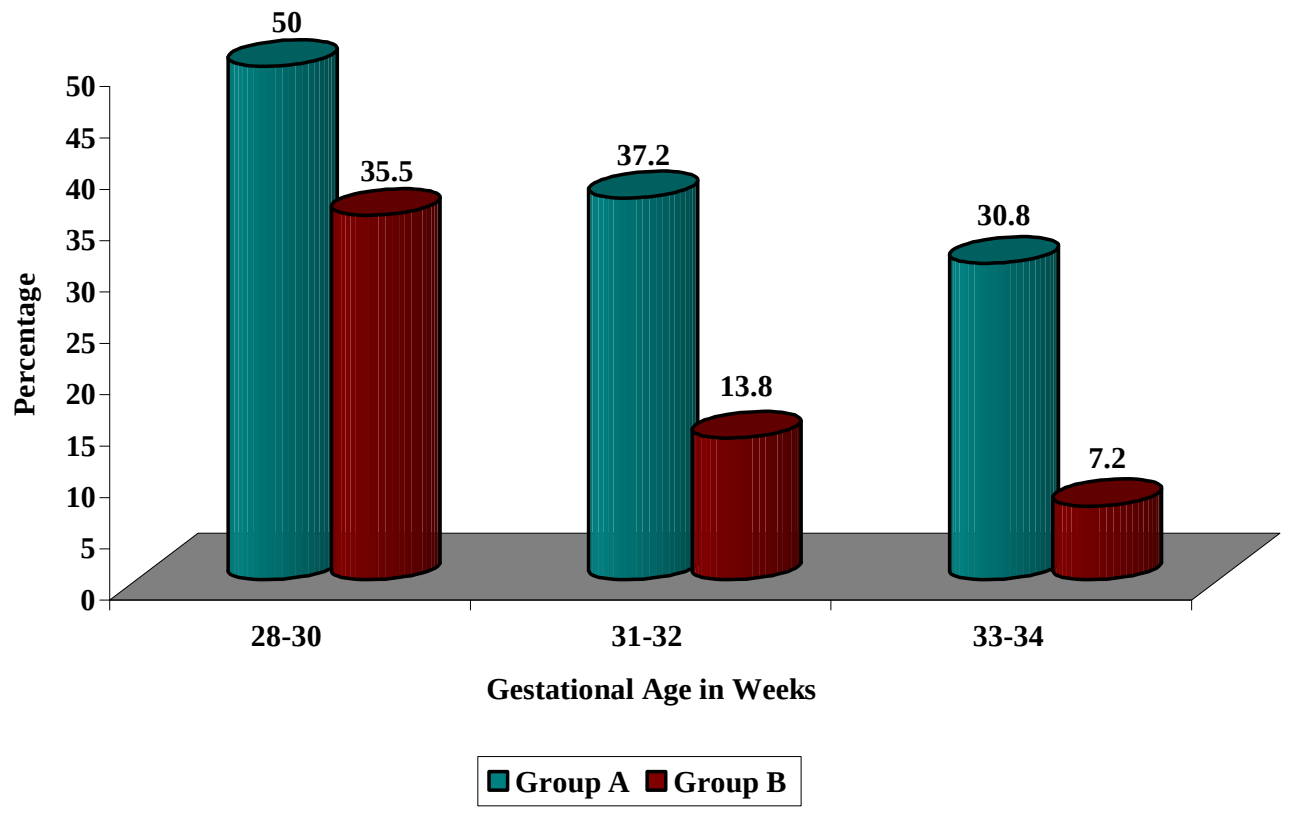


TABLE – 15

DOSE REQUIRED TO STOP CONTRACTIONS AMONG SUCCESS GROUP

Group A

Dose of $MgSo_4$	Number	%
4g	2	3.22%
8g	3	4.80%
12g	5	8.10%
16g	30	48.40%
> 16g	22	35.48%
Total	62	100%
Statistical Value	t = 1.493	

Group B

Dose of nifedipine	Number	%
20 mg	40	48.2%
40 mg	37	44.6%
50 mg	6	7.2%
Total	83	100%
Statistical Value	t = 7.518	

Group A

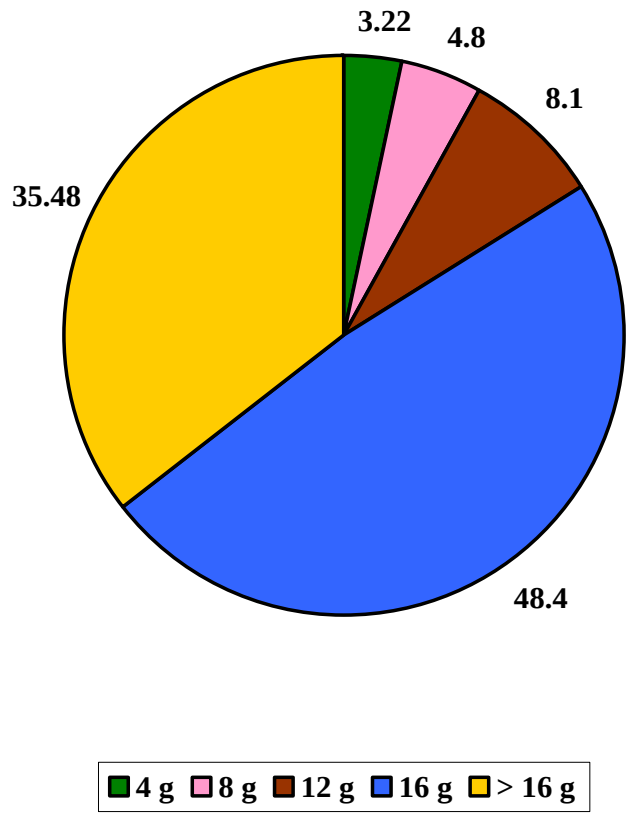
Among the success group, majority of patients in group A (48.4%) required 16g of $MgSO_4$ to stop contractions. 3.22% required 4g, 4.8% required 8g, 8.10% required 12g and 35.48% of patients required > 16g to stop uterine contractions.

Group B

In group B, 48.2% of patients required 20mg, 44.6% required 40mg and 7.2% required 50mg of nifedipine to stop uterine contractions.

DOSE REQUIRED TO STOP CONTRACTIONS AMONG SUCCESS GROUP

GROUP A



GROUP B

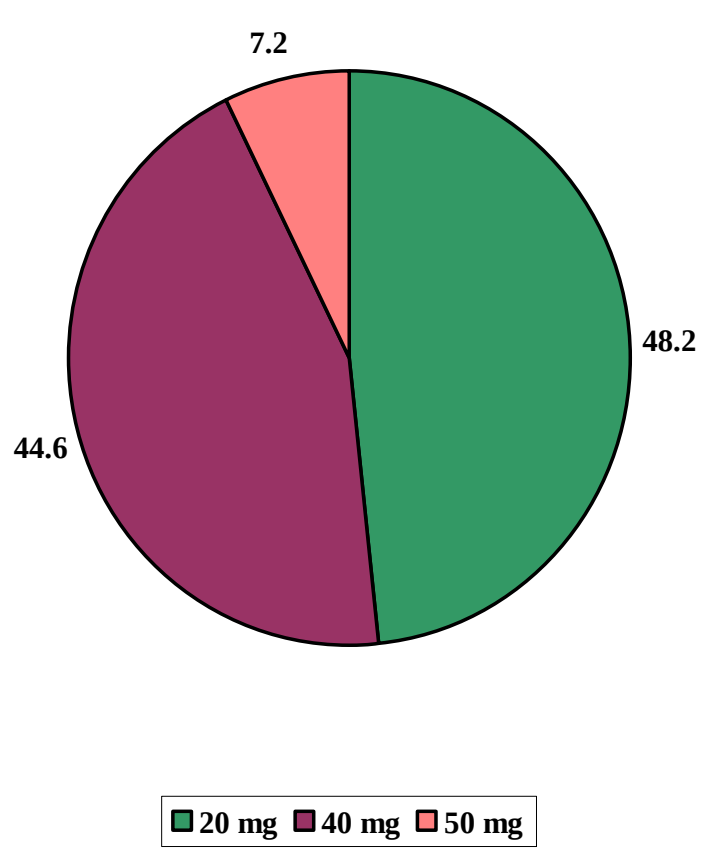


TABLE – 16

MODE OF DELIVERY

Mode of Delivery	Group A		Group B	
	Number	%	Number	%
Vaginal	98	98%	97	97%
Caesarean Section	2	2%	3	3%
Total	100	100%	100	100%
Statistical Value	t = 1.042		t = 1.064	

98% and 97% of patients delivered vaginally in group A and group B respectively. 2% of patients in group A and 3% of patients in group B underwent caesarean section for transverse lie and breech presentation.

MODE OF DELIVERY

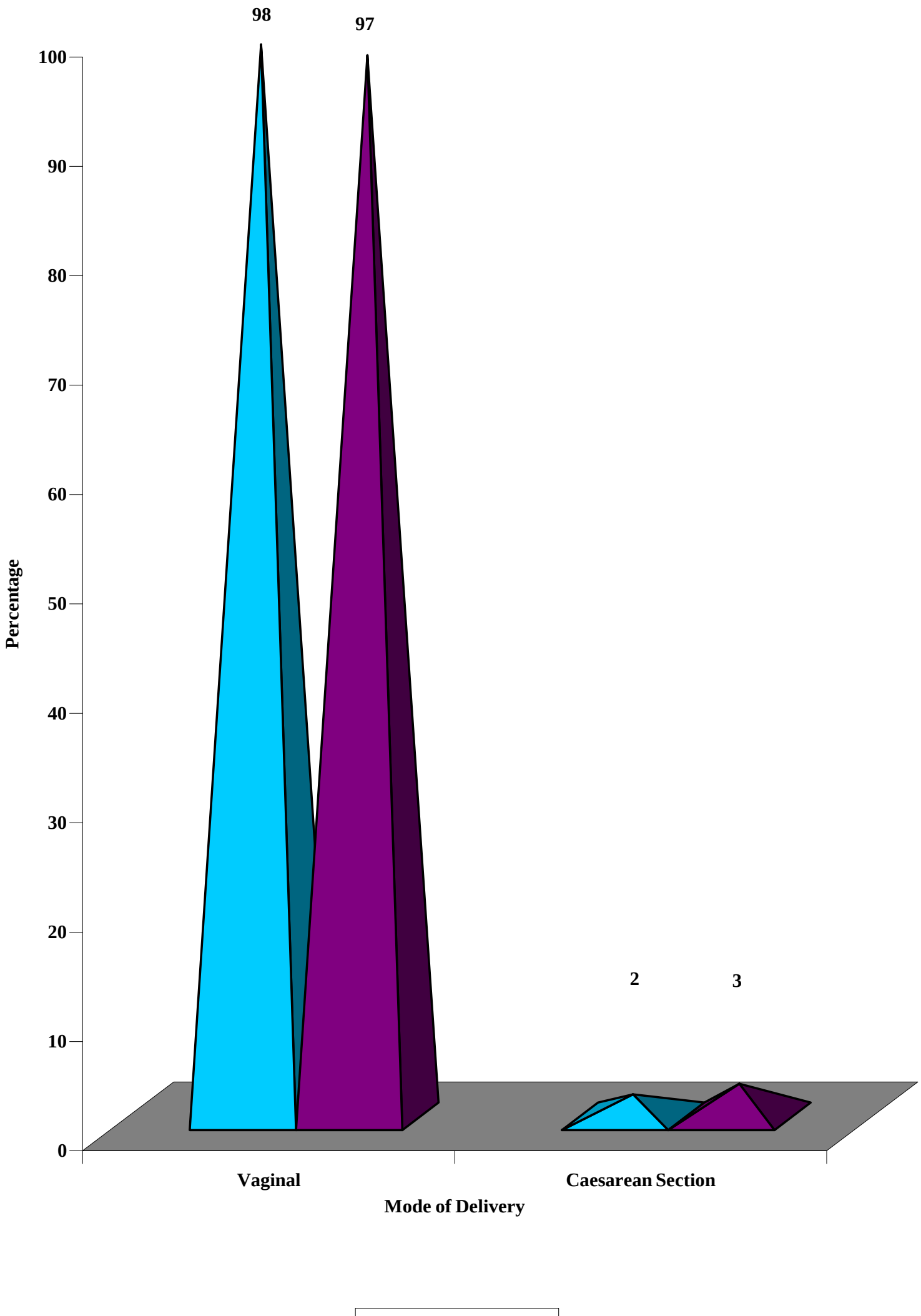


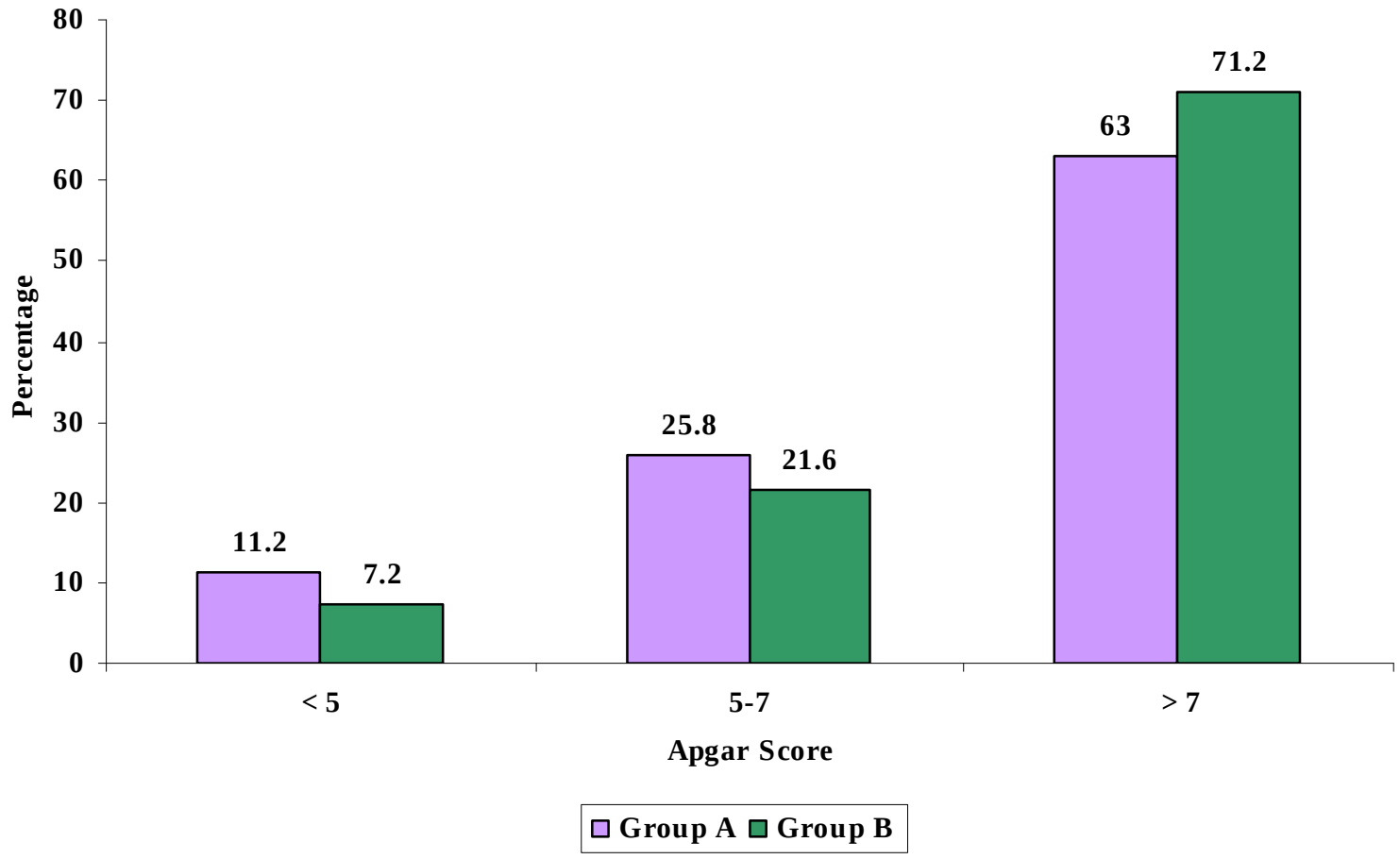
TABLE – 17

APGAR SCORE

5' Apgar	Group A		Group B	
	Success	Failure	Success	Failure
< 5	7 (11.2%)	21 (55.2%)	6 (7.2%)	9 (53%)
5 – 7	16 (25.8%)	12 (31.5%)	18 (21.6%)	5 (29.4%)
> 7	39 (63%)	5 (13.3%)	59 (71.2%)	3 (17.6%)
Total	62	38	83	17
Statistical Value	$\chi^2 = 74.23$		$\chi^2 = 77.63$	
	t = 19.55 p = 0.000			

Apgar score of >7 was found in 76.3% of babies in group A and 88.8% of babies in group B, which is statistically highly significant (p = 0.000).

APGAR SCORE IN SUCCESS GROUP



APGAR SCORE IN FAILURE GROUP

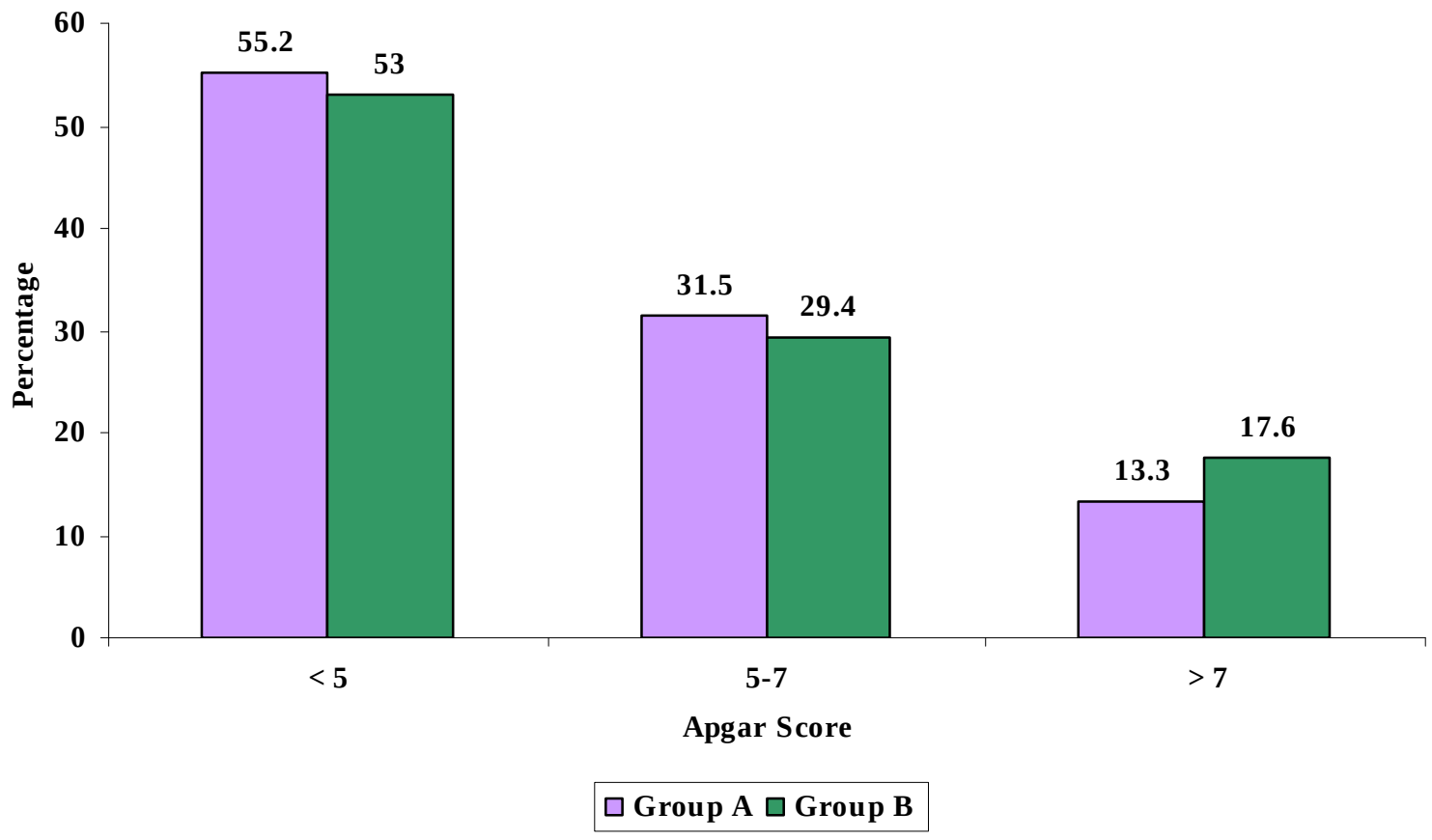


TABLE – 18

BIRTH WEIGHT OF BABY

Weight of baby (kg)	Group A		Group B	
	Number	%	Number	%
< 2	44	44%	36	36%
2 – 2.5	53	53%	58	58%
> 2.5	3	3%	6	6%
Total	100	100%	100	100%
Statistical Value	$\chi^2 = 25.73$		$\chi^2 = 31.67$	
	t = 1.008 p = 0.000			

Majority of the babies had a birth weight of 2 - 2.5kg in both the groups.

6% of babies in group B had a birth weight > 2.5kg, compared to 3% in group A which is statistically highly significant (p=0.000)

BIRTH WEIGHT OF BABY

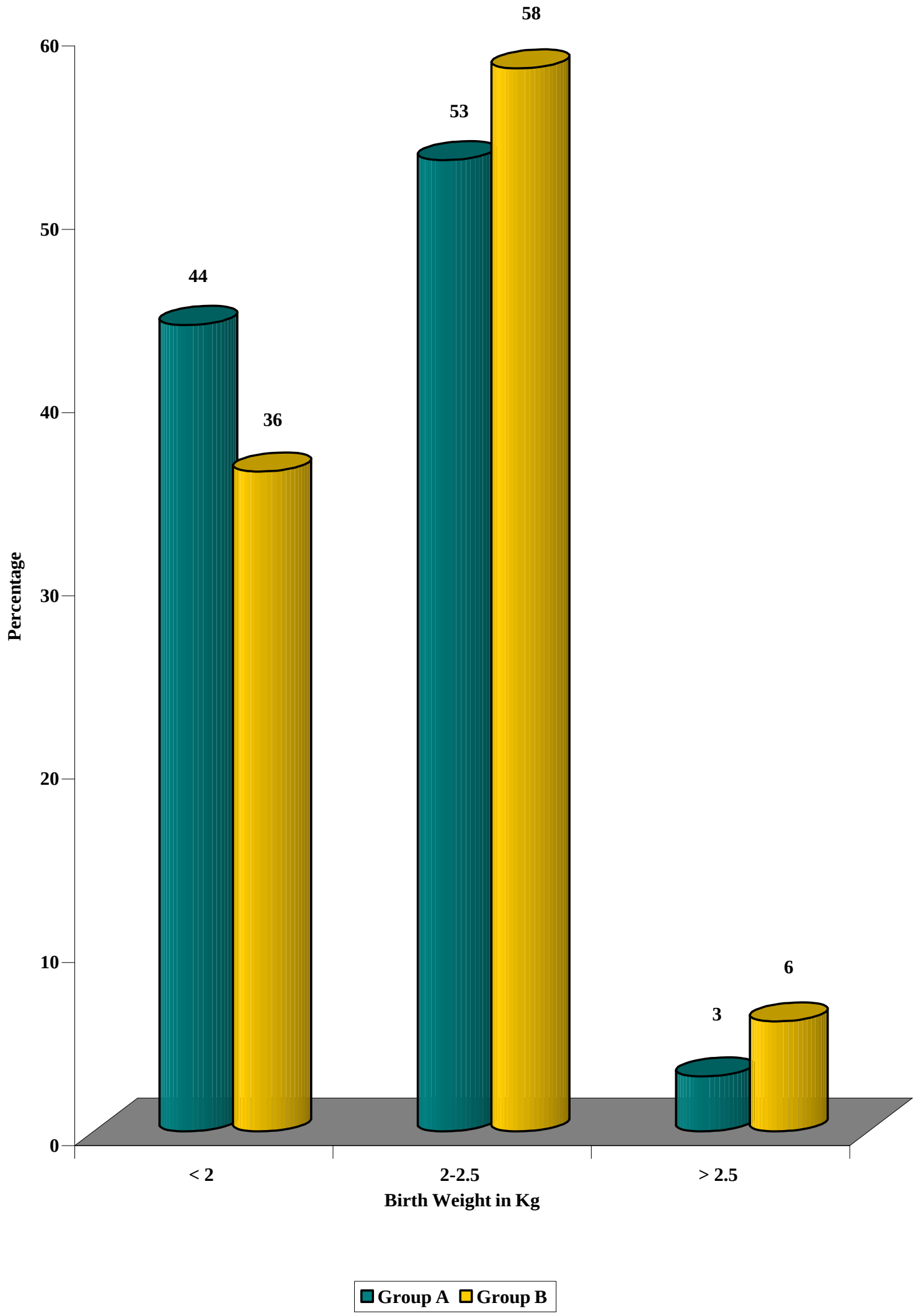


TABLE – 19

NEONATAL MORBIDITY

Complication	Group A	Group B
I – Hypothermia	13	9
II – RDS/Apnoea of immaturity	12	6
III – Sepsis	7	3
IV – Feeding problems	20	14
V – hyperbilirubinemia	22	16
VI – Intraventricular haemorrhage	3	2
Statistical Value	SD = 19.33	SD =18.67
	p = 0.001	

Some of the most common complications of preterm babies have been studied.

RDS, sepsis and intraventricular haemorrhage which are the most worrisome complications of preterm babies were found less commonly in group B, compared to group A and is statistically highly significant (p = 0.001).

In group A, hypotonia and hyporeflexia were observed in 5 babies, respiratory depression in 2 babies and apgar score < 5 in 28 babies.

Fetal tachycardia was observed in 2 patients in group B and FHR returned to baseline values 3 hours after commencing therapy.

NEONATAL MORBIDITY

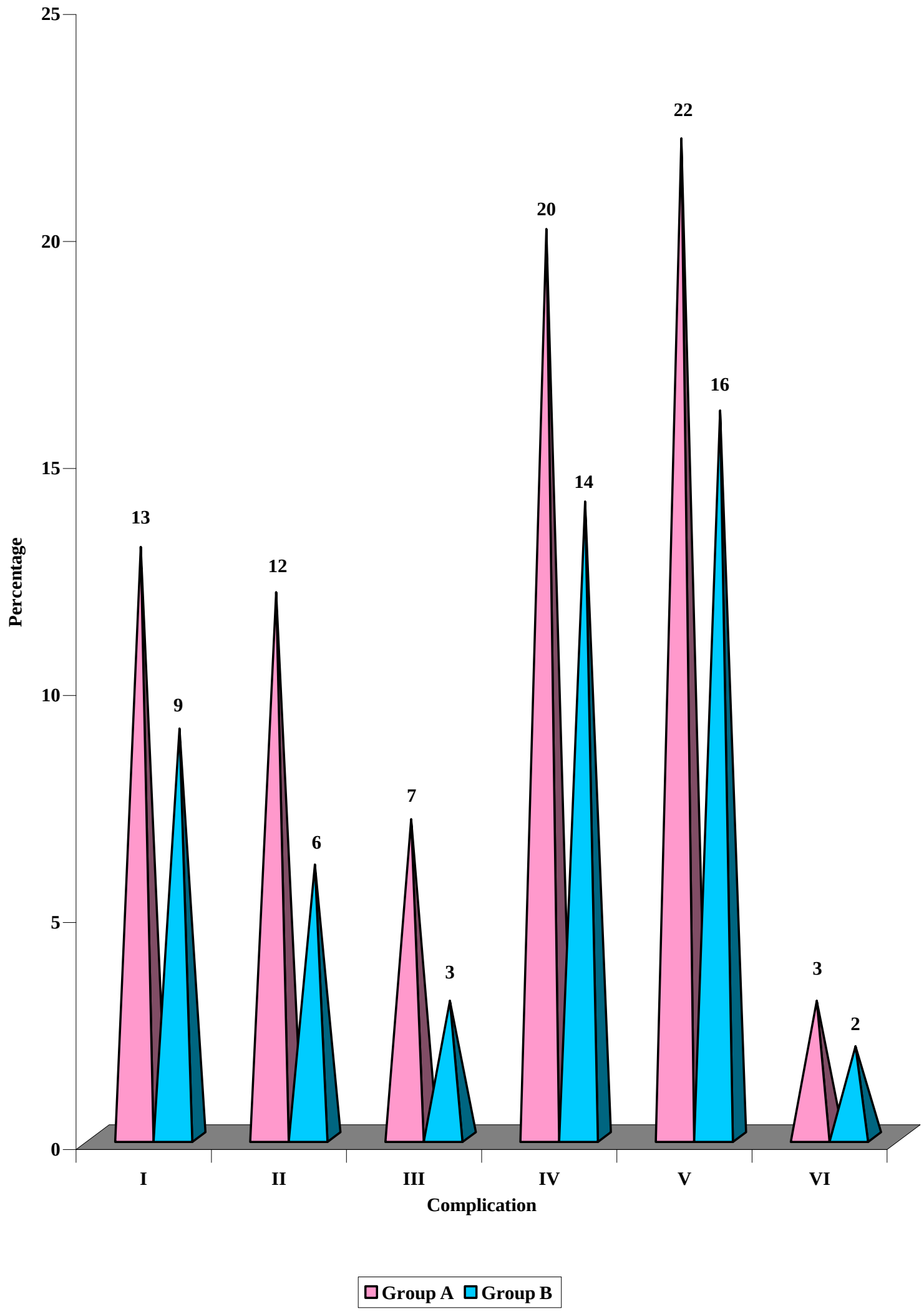


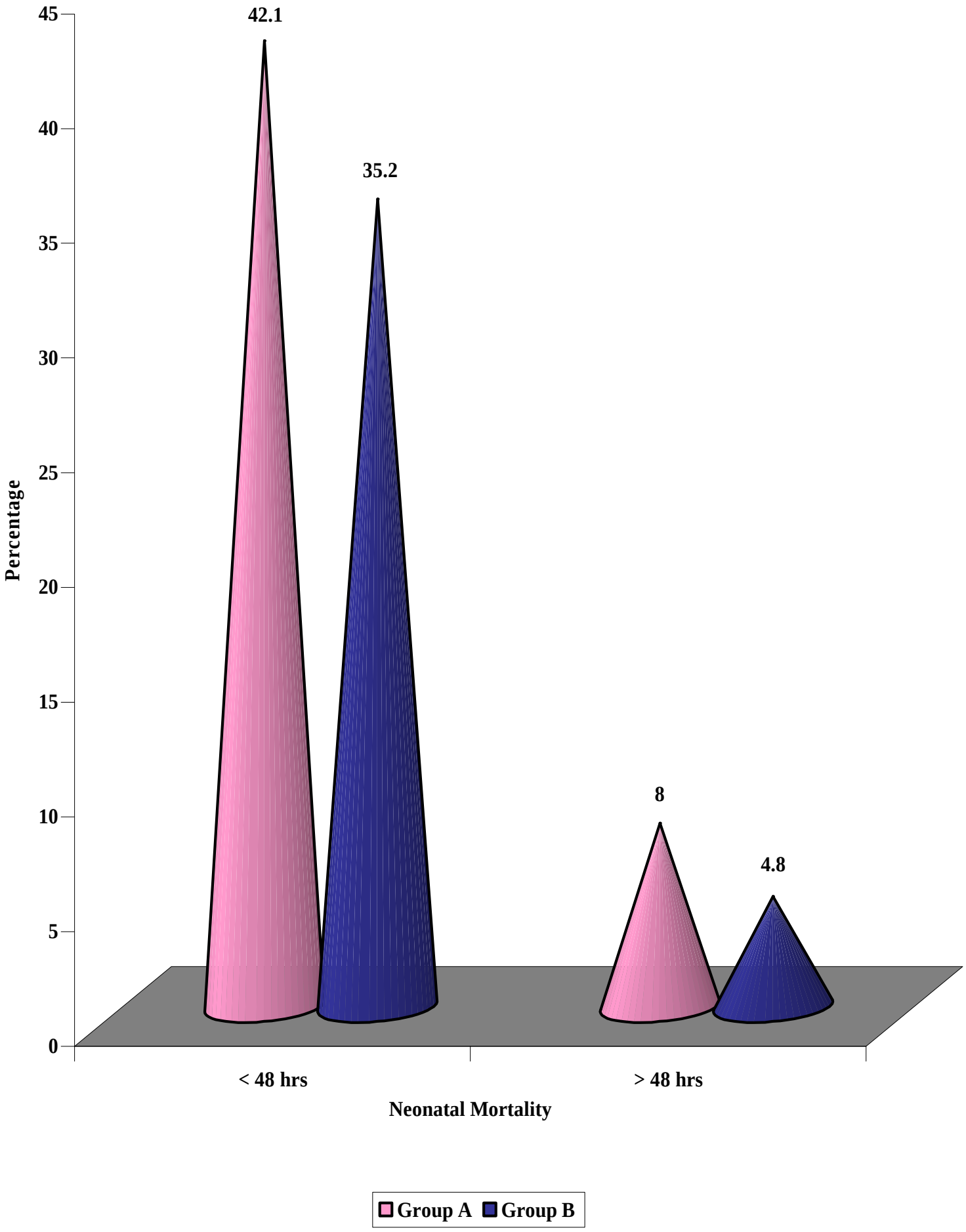
TABLE – 20

NEONATAL MORTALITY

Neonatal mortality	Group A		Group B	
	Number	%	Number	%
< 48 hrs	16	42.1%	6	35.2%
> 48 hrs	5	8%	4	4.8%
Total	21		10	
Statistical Value	SD = 38.61		χ^2 = 38.001	
	t = 19.01 p = 0.000			

In success group, neonatal mortality was 8% in group A and 4.8% in group B. In failure group it was 42.1% in group A and 35.2% in group B which is statistically highly significant (p = 0.000)

NEONATAL MORTALITY



DISCUSSION

DISCUSSION

Preterm labour and delivery accounts for major proportion of neonatal deaths. The goal of therapy of preterm labour is the drug which is effective and with minimum adverse effects.

In our study maternal demographic and clinical characteristics at randomisation were similar between the 2 groups regarding maternal age, socioeconomic status, parity, booking status, gestational age at admission and past obstetric history. Similarly vaginal and caesarean delivery rates were not significantly different.

In each study group, the gestational age at admission varied considerably. In our study the gestational age range was between 28 and 34 weeks. There is no consensus on a lower gestational age limit for the use of tocolytic agents. In a study conducted by **Krishna et.al (1996)**, gestational age at admission varied from 18-34 weeks. In another study done by **Lyell DJ et al (2011)** patients in the gestational age group of 24-34 weeks were assigned randomly to magnesium sulphate or nifedipine. Several metaanalysis excluded women with cervical dilatation of more than 4cm, while in our study the limit was 3cm.

Significant difference between the 2 groups was found in prolongation time after tocolytic administration, gestational age at delivery, incidence of low apgar scores, difference in birth weight and time spent by the newborn in NICU as in the study conducted by **Yasuyuki Kawagoe et al (2011).**

In a study conducted by **Ali Akbar Taherian et al in 2006**, group 1 received nifedipine 10mg tablet orally and repeated every 20 minutes (maximum dose of 40mg in the first hour). If contractions subsided, then the nifedipine maintenance dose would be 10 - 20mg 6^{th} hrly. Group 2 received magnesium sulphate, loading dose of 4 grams iv over 15 minutes, then a maintenance dose of 2-3g/hr as iv infusion. In our study both the drugs were used at a much lower dose; with 48.4% of patients requiring 16 grams of $MgSo_4$ and 48.2% of patients requiring 20mg of nifedipine to stop contractions among the success group.

Tsatsaris et al in 200l, has concluded that nifedipine was more effective in delaying delivery for atleast 48 hours and was more likely to prolong pregnancy beyond 34 weeks when compared with any other tocolytics.

Mild and severe maternal and neonatal adverse effects were more frequent with $MgSo_4$ **(Larmon JE et al).** In a study conducted by **Sakhavar et al (2008),** the most common side effect of $MgSo_4$ was hyperthermia (53.4%) and most of the patients in this group experienced some kind of adverse effects. In a study conducted by **Ferguson et al (1990),** nifedipine caused headache in 5-6% and palpitations in 0-6% of patients. In our study, the commonest side effects were flushing (21%) and headache (14%) in group A and group B respectively.

Nifedipine has been reported to reduce neonatal morbidity (RDS & IVH) compared to other tocolytics **(Papatonis et al, 2000).**

Nifedipine has an excellent safety profile. Oral nifedipine could be a suitable and more convenient alternative to intravenous magnesium sulphate in arresting preterm labour. Magnesium sulphate is not as effective as nifedipine and is unpleasant for women.

SUMMARY

SUMMARY

In this prospective randomised controlled trial of comparison of safety and efficacy of intravenous magnesium sulphate and oral nifedipine it was observed that:

1. Preterm labour was common in the age group of 20-24 years in both the groups.
2. Preterm labour occurred with a higher frequency in primigravida, belonging to class V socioeconomic status with inappropriate antenatal care.
3. Between 31-32 weeks of pregnancy there were 35% of patients in group A and 29% of patients in group B. Between 33-34 weeks there were 39% of patients in group A and 42% in group B.
4. Previous history of preterm delivery was present in 9% of patients in group A and 8% in group B. History of second trimester abortion was present in 6% of patients in group A and 4% in group B.
5. In group A, the commonest side effect was flushing which occurred in 21% of patients.

   In group B, the commonest side effect was headache which was observed in 14% of patients.

On an average, patients with side effects were 52% in group A and 35% in group B. In both the study groups there was **no maternal mortality.**

6. Cephalic presentation was found in 85% of patients in group A and 87% in group B. Others had breech presentation with a small percentage of patients in transverse lie.
7. When the cervix was 25% effaced, prolongation of pregnancy beyond 48 hours was observed in 86.2% of patients in group A and 100% in group B. With 50% cervical effacement, successful tocolysis was observed in 60.7% of patients in group A and 85.7% in group B. With 75% cervical effacement, successful tocolytic effect was seen in 20% of patients in group A and 52.2% in group B.
8. When the cervix was 1cm dilated, successful tocolysis was observed in 79.5% of patients in group A and 97.3% in group B. With 2cm cervical dilatation, prolongation of pregnancy beyond 48hours was observed in 52% and 76% of patients in group A and B respectively. When the cervix was 3cm dilated, none of the patients in group A had prolongation of pregnancy beyond 48 hours and 66.7% of patients in group B had successful tocolysis.
9. Success of acute tocolysis defined as delay of delivery for more than 48hours was observed in 62% of patients in group A compared with 83% in group B.

10. Duration of prolongation beyond 2 weeks was observed in 3% of patients in group A and 6% in group B, and all these patients delivered beyond 37weeks of gestation with very good neonatal outcome.
11. Between 33-34 weeks of gestation, prolongation of pregnancy beyond 48 hours was observed in 69.2% of patients in group A and 92.8% of patients in group B. In the gestational age group of 28-30 weeks success of tocolysis was observed in 50% and 65.5% of patients in group A and group B respectively.
12. Among patients with successful tocolysis 4g of $MgSo_4$ was required in 3.22% of patients, 16g in 48.4% and >16g in 35.48% of patients in group A.

    In group B, among the success group 20 mg of nifedipine was required in 48.2% of patients. 40 mg and 50 mg were required in 44.6% and 7.2% of patients respectively.
13. 98% and 97% of patients delivered vaginally in group A and group B respectively.
14. 76.3% and 88.8% of babies belonging to group A and group B respectively had an apgar score of > 7.
15. Babies weighing 2-2.5kg were delivered in 53% and 58% of patients in group A and group B respectively. Birth weight of >2.5kg was observed in 3% of patients in group A and 6% in group B.

16. RDS / apnoea of immaturity, the most frequent complication of prematurity was seen in 12% of patients in group A and 6% in group B. Septicemia developed in 7% and 3% of patients in group A and group B respectively. Intraventricular haemorrhage the most dreaded complication developed in 3% of patients in group A and 2% in group B.
17. Among the success group neonatal mortality was observed in 8% and 4.8% of patients in group A and group B respectively.

CONCLUSION

CONCLUSION

Tocolytics do appear to delay delivery long enough for successful administration of corticosteroids one of the few interventions of clear benefit.

The perfect tocolytic, which is uniformly effective and has no fetomaternal side effects doesn't exist. Currently available data suggest that magnesium sulphate administration doesn't increase the risk of fetal and neonatal mortality. Though $MgSo_4$ is effective in preventing preterm labour, nifedipine is safe, well tolerated, with few fetomaternal side effects, non invasive and more effective with low purchase price **(RCOG, 2011)**. However, close monitoring is still recommended to avoid and reduce any associated morbidity when these tocolytic agents are used.

In our study, nifedipine was more effective and rather safe tocolytic agent associated with more rapid arrest of contractions, more frequent successful prolongation of pregnancy, better side effect profile, no serious maternal adverse reactions, resulting in good neonatal outcome and significantly fewer admissions of newborn to the NICU, when compared to magnesium sulphate.

Using nifedipine simplifies tocolytic administration and decreases hospital stay without increasing the risk of prematurity.

Preterm labour and delivery is the major cause of perinatal morbidity and mortality especially in developing countries like India. Preventable and

treatable cause of preterm labour should be identified and dealt with for better maternal and neonatal outcome.

To conclude, the use of nifedipine as the first line tocolytic is safe when compared to $MgSo_4$. But severe maternal hypotension can occur and close monitoring of vitals is warranted.

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PROFORMA

COMPARISON OF SAFETY AND EFFICACY OF INTRAVENOUS MAGNESIUM SULPHATE AND ORAL NIFEDIPINE IN TREATMENT OF PRETERM LABOUR

PROFORMA

NAME: AGE: I.P. NO.:

ADDRESS:

OCCUPATION: SOCIO ECONOMIC STATUS:

DATE & TIME OF ADMISSION:

DATE & TIME OF DELIVERY:

DATE OF DISCHARGE:

OBSTETRIC CODE: G P L A D

LMP: EDD:

GESTATIONAL AGE IN WEEKS:

BOOKED/ UNBOOKED IMMUNISED/ NOT IMMUNISED

HISTORY OF PRESENTING ILLNESS:

- Lower abdominal pain
- Fetal movements
- Vaginal discharge
- Bleeding PV
- Draining PV
- Fever
- UTI/ URI
- Other infections
- Coitus
- Trauma

MENSTRUAL HISTORY:

MARITAL HISTORY:

OBSTETRIC HISTORY:

Previous obstetric outcome

PAST OBSTETRIC HISTORY:

- H/O Preterm labour
- H/O Abortion: Induced/ Spontaneous
- Stillbirth

PAST MEDICAL HISTORY

Diabetes/ Hypertension/ TB/ Heart Disease/ Jaundice/ Epilepsy/ Renal disease/ STD

PERSONAL HISTORY

Smoking /Alcohol

GENERAL EXAMINATION:

Febrile Pallor Pedal Edema Icterus

VITALS:

Temp: PR: BP: RR: Ht: Wt: BMI:

SYSTEMIC EXAMINATION:

CVS: RS: CNS:

OBSTETRIC EXAMINATION:

P/A

- Symphysio fundal height
- Uterine size in weeks
- Contractions
- Presenting part
- Position
- Liquor
- FHR
- EFW

P/V

- Cervix
    - Effacement
    - Dilatation
    - Consistency
    - Position
- Station
- Membrane
- Pelvis
- Draining pv / bleeding pv
- Show
- Bishop’s score

INVESTIGATIONS

- Urine: Sugar/Albumin/Microscopy
- Urine culture & sensitivity:
- Complete hemogram:
- Blood grouping & typing:
- Blood sugar:
- Blood urea:
- Serum creatinine:
- Serum electrolytes:
- Vaginal swab culture & sensitivity:
- ECG
- CTG
- USG
    - ✓ Singleton/ Multiple
    - ✓ Cephalic/ Breech
    - ✓ Gestational Age
    - ✓ AFI

- ✓ Placenta
- ✓ Congenital anomalies
- ✓ EFW

GROUP A

INTRAVENOUS MgSO4

4g bolus followed by 1g/hr infusion

GROUP B

ORAL NIFEDIPINE

20mg stat followed by 20mg after 30minutes if contractions persist, then 10mg 8th hrly

TIME	DOSE	CONTRACTION	PR (b/min)	BP (mm Hg)	RR (breaths/ min)	KNEE JERK	URINE OUTPUT	FHR

PERIOD OF TOCOLYSIS:

SIDE EFFECTS:

GROUP A

- Flushing
- Nausea/vomiting
- Headache
- Drowsiness
- Muscle weakness
- Pulmonary edema
- Respiratory depression
- Neonatal complication

GROUP B

- Dizziness
- Palpitations/ tachycardia
- Transient hypotension
- Headache
- Flushing
- Nausea
- Peripheral edema
- Fetal hypoxia

FETAL OUTCOME:

- Mode of delivery:
- GA at delivery:
- Alive/ Stillborn:
- Sex:
- Birth Weight:
- APGAR: 1’

  5’
- Treatment- Delivery interval:
- Neonatal complications: Admitted/ Not admitted
  - Hypothermia
  - RDS/Apnoea of immaturity
  - Sepsis
  - Feeding problems
  - Hyperbilirubinemia
- Neonatal death:

  YES / NO

  CAUSE:

MASTER CHART

S. No.	Name	Age	IP No.	SES	DOA	Obstetric Code	Booking Status	GA at Admn (weeks)	H/O PTL	H/O 2nd TA	Group	Maternal S/E	Presentation	Cx Effacement %	Cx dilatation (cm)	Station	GA at Del(weeks)	TDI (days)	Birth Wt. (kg)	Apgar	Neonatal outcome
1.	Saraswathi	24	31781	IV	5.7.11	G2	UB	32	-	-	A	-	C	50	2	-1	32^{+2}	2	2.1	7	
2.	Periyakka	18	31946	V	6.7.11	P	B	29	-	-	A	N/V	C	25	1	-3	29^{+3}	3	1.1	4	I
3.	Thamaraiselvi	21	32516	V	9.7.11	G2	UB	30	-	+	B	-	C	75	2	-1	30	6 hrs	1.5	4	
4.	Saranya	27	32924	III	9.7.11	P	UB	28	-	-	A	-	B	50	2	-2	28	1 day	1.05	4	II, VI, VII
5.	Rani	20	33498	V	12.7.11	P	B	30	-	-	A	F	C	25	1	-1	30^{+4}	4	1.2	3	
6.	Kiruthika	22	33961	V	15.7.11	P	UB	32	-	-	A	-	C	50	2	-3	32	10 hrs	2.2	4	V, IV
7.	Pichaimani	22	34403	V	15.7.11	P	UB	29	-	-	B		C	50	2	-3	29^{+2}	2	1.2	3	I
8.	Devika	19	34772	III	21.7.11	P	B	33	-	+	A	Dr	C	25	2	-3	33	12 hrs	2.2	6	V
9.	Ranjini	27	34792	III	26.7.11	G3	B	34	-	-	B	-	C	75	2	-1	34	4 hrs	2.2	8	
10.	Parveen Begum	26	35047	V	31.7.11	P	B	33	-	-	B		B	75	3	0	33	6 hrs	2.3	8	IV, V
11.	Nagarani	29	35665	III	1.8.11	P	B	32	-	-	A	-	C	25	1	-2	32^{+2}	2	1.8	8	
12.	Dhanalakshmi	23	35676	IV	6.8.11	P	UB	32	-	-	B	H	C	25	1	-2	33^{+2}	9	2.1	6	
13.	Vasanthi	24	36105	V	6.8.11	G2	UB	28	-	-	A	-	C	50	1	-3	29	7	1 kg	5	II, VII
14.	Bahadur Nisha	20	36282	V	11.8.11	G2	UB	28	-	-	A	N/V	C	50	2	-1	29^{+3}	10	1.1	4	II
15.	Jeyanthi	25	36299	IV	16.8.11	P	B	30	-	-	A	-	C	25	1	-2	31^{+2}	9	1.2	6	V, III
16.	Sathya	23	36316	V	18.8.11	P	UB	31	-	-	B	Di	C	50	2	-3	31	18 hrs	2	4	VII, III
17.	Nadiya	28	36420	V	18.8.11	P	UB	33	-	-	B	-	C	25	1	-2	34^{+4}	11	2.3	8	
18.	Jeevitha	30	36962	V	22.8.11	G3	B	30	-	-	B	-	C	25	1	-3	31^{+2}	9	1.5	5	
19.	Bhanupriya	32	37086	IV	24.8.11	G2	B	32	-	-	A	-	C	50	1	-1	32	20 hrs	1.8	4	I, V
20.	Punitha	23	37595	V	29.8.11	P	UB	32	-	-	B	-	C	75	2	0	32	6 hrs	2.1	6	I
21.	Anjalai	19	37684	V	29.8.11	P	UB	33	-	-	A	-	C	50	2	-3	33	12 hrs	2.2	7	IV
22.	Bhuvana	20	38450	V	12.9.11	P	UB	34	-	-	B	H	C	25	1	-3	37	21	2.6	9	
23.	Priya	27	38752	IV	15.9.11	P	UB	30	-	-	A	-	C	50	2	-3	30	22 hrs	1.3	4	I, IV
24.	Priyadharshini	26	38799	V	19.9.11	G3	B	30	-	-	B	-	C	50	2	-3	30	12 hrs	1.3	6	VII, II
25.	Jeyakodi	24	38826	V	19.9.11	P	B	31	-	-	B	Di	C	25	1	-3	32^{+5}	12	1.8	6	
26.	Krishnammal	25	39120	III	22.9.11	G2	UB	29	-	-	A	-	B	50	2	-2	29	15 hrs	1.2	3	I, VII, II
27.	Vasanthi	21	39374	IV	23.9.11	P	B	32	-	-	A	-	C	50	1	-2	32^{+3}	3	2.1	5	V
28.	Mariyammal	24	39382	V	25.9.11	G2	B	33	-	-	B	-	C	25	2	-2	33^{+3}	3	2.3	8	
29.	Abhirami	23	39778	IV	30.9.11	P	UB	28	-	-	B	-	C	50	2	-3	28^{+3}	3	1.1	3	VII, III
30.	Premalatha	23	39793	V	30.9.11	G3	UB	30	-	-	B	BP	C	75	3	-1	30	4 hrs	1.4	4	II, VII
31.	Indhra	22	39769	III	31.9.11	P	B	33	-	-	A	-	C	25	2	-3	33^{+2}	2	2.2	8	IV

32.	Kavitha	31	39840	IV	1.10.11	G2	B	32	+	-	A	H,F	C	75	3	0	32	18 hrs	2.2	6	IV
33.	Sasikala	30	39900	V	6.10.11	P	UB	34	-	-	A	-	B	50	1	-2	34	15 hrs	2.4	8	
34.	Usha	19	40290	V	8.10.11	P	UB	31	-	-	B	P	C	50	2	-2	31^{+3}	3	1.6	6	V
35.	Selvam	24	40464	IV	8.10.11	P	UB	32	-	-	4	-	C	50	2	-1	33	7	2.3	8	
36.	Akila	20	40955	V	18.10.11	P	UB	33	-	-	B	-	C	25	2	-3	33^{+5}	5	2.4	8	I
37.	Rasathi	24	41289	V	20.10.11	P	B	31	-	-	B	-	C	50	2	-1	31^{+3}	3	1.6	8	
38.	Sampoornam	33	41559	V	25.10.11	G2	UB	34	+	-	B	H	C	75	2	-1	34	6 hrs	2.4	8	II, V
39.	Chinnaponnu	25	41658	III	12.11.11	G2	B	32	-	-	A	F	C	25	1	-3	32^{+3}	3	2.2	6	IV, I
40.	Sudha	27	41748	V	14.11.11	G2	UB	32	-	+	A	N/V, F	C	50	2	-3	32	12 hrs	2.3	3	
41.	Amirtham	30	42317	V	14.11.11	P	UB	33	-	-	B	P	TL	25	2	-2	33^{+3}	3	2.1	8	V
42.	Suseela	26	42357	V	19.11.11	P	UB	33	-	-	A	F, Dr	C	50	1	-2	33	1 day	2.3	8	V
43.	Malathi	28	42368	IV	20.11.11	P	B	28	-	-	B	Di	C	50	2	-3	28	16 hrs	1.2	3	VII, VI
44.	Bakkiyam	22	42251	V	24.11.11	P	UB	31	-	-	A	N/V	C	50	2	-2	31	10 hrs	2	5	VII, II
45.	Sangeetha	21	42359	III	24.11.11	P	B	30	-	-	B	-	C	25	1	-3	31^{+6}	13	1.2	5	
46.	Kalamani	23	42446	V	24.11.11	G2	B	32	-	-	A	H, F	C	50	2	-3	33^{+1}	8	2.2	6	I, V
47.	Nithya	21	42897	IV	26.11.11	P	B	34	-	-	B	Di	C	75	3	0	34^{+2}	2	2.2	8	
48.	Suryadevi	19	43011	V	28.11.11	P	B	33	-	-	A	-	C	50	2	-1	33^{+2}	2	2.4	8	IV
49.	Vijayalakshmi	26	43020	V	29.11.11	G4	UB	34	+	-	B	-	C	50	2	-3	34^{+3}	3	2.3	8	
50.	Menaka	25	43077	V	29.11.11	G3	UB	33	-	-	A	-	C	75	2	-3	33	6 hrs	2.4	7	
51.	Nandinidevi	25	43086	IV	3.12.11	P	UB	34	-	-	B	BP	C	50	2	-2	34^{+3}	3	2.3	8	
52.	Rani	28	43208	V	4.12.11	G3	B	34	-	-	A	-	C	25	1	-2	34^{+3}	3	2.5	8	
53.	Parveen	22	43234	V	4.12.11	G2	UB	31	-	-	B	-	C	50	2	-2	31^{+6}	6	1.6	6	II, I
54.	Anandi	37	43239	V	5.2.11	G2	UB	31	-	-	B	-	C	25	2	-3	31^{+4}	4	1.5	7	
55.	Radhika	29	43410	IV	7.12.11	P	UB	30	-	-	A	-	C	50	1	-2	30^{+3}	3	1.4	4	VII, VI, IV
56.	Valli	30	43707	V	7.12.11	G2	UB	34	-	-	B	N/V	C	25	1	-2	37	21	2.7	9	
57.	Devi	21	43753	V	10.12.11	P	B	33	-	-	B	-	C	50	1	-3	33^{+3}	3	2.4	8	
58.	Shanthi	23	43852	IV	12.12.11	G2	UB	29	-	-	B	-	B	25	1	-2	30^{+4}	11	1.1	4	VII, II
59.	Nandini	25	44011	V	12.12.11	P	UB	34	-	-	A	Dr	C	50	2	-3	34	15 hrs	2.5	8	I, V
60.	Sharadha	27	44021	V	15.12.11	G3	B	32	-	-	B	H	C	50	2	-3	32	15 hrs	2.3	6	
61.	Indrani	27	44311	III	17.12.11	G2	B	33	-	-	B	-	C	25	2	-2	33^{+3}	3	2.4	8	
62.	Sulochana	23	44536	V	17.12.11	G2	B	32	+	-	A	-	C	50	1	-2	32	1 day	2.4	4	IV, V
63.	Padmavathi	22	44625	V	20.12.11	P	B	31	-	-	B	Di	C	75	2	-1	31	4 hrs	2	5	
64.	Mookayee	27	44713	V	21.12.11	P	UB	30	-	-	A	F	C	50	1	-2	30^{+2}	2	1.3	3	I, VI, VII

65.	Palaniyammal	20	44758	V	22.12.11	P	UB	28	-	-	B	-	C	75	3	-3	28	4 hrs	1 kg	3	III, VII
66.	Deepa	20	44895	III	24.12.11	P	UB	33	-	-	A	-	B	50	1	-3	33^{+5}	5	2.4	8	V, IV
67.	Ramya	18	45861	IV	24.12.11	P	UB	30	-	-	B	F	C	50	2	-2	30	6 hrs	1.2	4	VII, VI
68.	Kokila	30	45883	III	26.12.11	P	B	34	-	-	A	-	C	25	1	-1	35	7	2.4	8	
69.	Alagammal	28	45903	IV	28.12.11	G3	UB	29	-	+	B	-	C	25	1	-3	30^{+2}	9	1.1	3	
70.	Gayathri	31	45982	V	28.12.11	P	B	30	-	-	A	-	B	75	3	-2	30	6 hrs	1.4	3	III, VII
71.	Juliet	24	45999	IV	29.12.11	G3	B	34	-	-	B	-	C	75	2	-2	34^{+2}	2	2.2	8	
72.	Kavitha	24	46214	V	29.12.11	P	UB	34	-	-	A	-	C	75	2	-2	34	12 hrs	2.5	7	
73.	Noorjahan	21	46350	V	31.12.11	P	UB	32	-	-	A	H	C	50	2	-3	32	15 hrs	2.2	4	III, VII
74.	Malarmani	24	32	IV	1.1.12	P	B	28	-	-	B	-	C	50	2	-3	28^{+3}	3	1.1	4	VII, V
75.	Sendamarai	20	49	V	1.1.12	P	B	30	-	-	A	N/V	B	25	1	-3	30^{+3}	3	1.3	3	II, VII
76.	Ilakkiya	26	129	III	2.1.12	P	UB	32	-	-	B	H	C	50	1	-3	32^{+4}	4	2.1	8	
77.	Vimalarani	28	248	IV	4.1.12	G2	UB	34	+	-	A	-	C	50	1	-3	34^{+2}	2	2.4	8	
78.	Amirthavalli	27	540	V	6.1.12	G3	UB	33	-	-	B	N/V	C	25	2	-2	33^{+6}	6	2.1	8	IV, V
79.	Sudha	22	592	V	6.1.12	P	B	30	-	-	A	-	C	50	2	-3	30	18 hrs	1.2	3	I, IV, V
80.	Palaniyammal	23	702	III	7.1.12	G2	B	34	-	-	B	Di	C	75	2	-2	34^{+3}	3	2.2	8	
81.	Mariyayee	20	1096	V	10.1.12	P	UB	31	-	-	A	F	C	50	1	-3	31^{+6}	6	1.4	5	V
82.	Eshwari	25	1131	IV	11.1.12	G2	UB	32	-	-	B	-	B	75	2	-1	32	6 hrs	2	7	
83.	Reshma	30	1236	V	11.1.12	G4	UB	33	-	+	A	F, N/V	C	50	1	-2	33^{+3}	3	2.4	8	IV, V
84.	Maheswari	19	1323	IV	12.1.12	P	B	32	-	-	B	P	C	50	1	-3	32^{+4}	4	2.1	8	
85.	Kamalam	31	1628	V	15.1.12	G4	UB	34	+	-	A	F	C	25	2	-3	37^{+1}	22	2.7	8	I, V
86.	Annakamu	30	1720	III	16.1.12	G4	B	29	-	-	B	-	C	25	1	-3	29^{+5}	5	1.2	4	
87.	Pushpam	21	1730	V	16.1.12	P	B	30	-	-	A	-	C	25	1	-2	30^{+4}	4	1.5	4	I
88.	Megala	24	1799	IV	19.1.12	P	UB	31	-	-	A	-	C	50	1	-2	31^{+6}	6	1.7	6	
89.	Sivaranjini	36	1963	V	22.1.12	P	UB	33	-	-	B	N/V	C	25	2	-3	33^{+3}	3	2.2	8	I
90.	Umarani	22	1986	V	23.1.12	G2	B	34	-	-	A	F	B	50	2	-3	34^{+3}	3	2.4	8	
91.	Kalaiselvi	20	2484	III	23.1.12	G2	B	32	+	-	B	-	C	50	1	-3	32^{+4}	4	2	8	I
92.	Latha	23	2496	IV	26.1.12	P	B	33	-	-	A	-	C	75	2	-2	33^{+2}	2	1.9	8	
93.	Sumathi	25	2566	V	27.1.12	G3	UB	29	-	-	B	P	C	50	1	-2	29^{+6}	6	1.1	5	VII, II
94.	Reena	28	3303	III	27.1.12	G4	UB	32	-	-	A	-	C	50	2	0	33	7	2.2	8	IV
95.	Nagalakshmi	18	3365	IV	28.1.12	G2	UB	31	+	-	A	F, PE	C	25	1	-1	31	1 day	1.8	4	II, VII
96.	Annalakshmi	22	3399	V	29.1.12	P	UB	30	-	-	B	-	B	50	3	-3	30^{+3}	3	1.3	6	

97.	Mariyapriya	23	3437	V	29.1.12	P	B	34	-	-	B	-	C	75	3	-1	34^{+2}	2	2.1	8	IV
98.	Manjula	21	3749	V	29.1.12	P	UB	34	-	-	A	H	C	50	2	-2	34	15 hrs	2.4	8	V
99.	Meena	25	4100	V	2.2.12	P	B	33	-	-	B	-	C	25	1	-2	34^{+1}	8	2.2	8	
100.	Kaveri	28	4123	IV	4.2.12	G3	UB	32	-	-	A	-	TL	50	2	-3	32	10 hrs	1.9	6	
101.	Divya	28	4213	V	5.2.12	G2	B	34	-	+	B	-	C	25	2	-3	34^{+5}	5	2.3	8	
102.	Chandra	21	4300	V	10.2.12	G2	UB	29	-	-	A	-	C	50	1	-2	29	12 hrs	1.2	3	III, VII
103.	Padma	27	4359	V	10.2.12	G4	UB	28	-	-	B	-	C	25	1	-3	28^{+4}	4	1.2	6	
104.	Subbulakshmi	29	4400	V	11.2.12	P	B	31	-	-	B	-	C	25	2	-3	31^{+3}	3	2	8	I
105.	Vanitha	21	4452	V	15.2.12	P	UB	33	-	-	A	-	B	50	2	-3	33^{+3}	3	2.2	8	I
106.	Neelamani	30	4808	V	17.2.12	P	UB	33	-	-	B	-	C	50	1	-3	33^{+6}	6	2.1	8	
107.	Mumtaj	32	4829	V	20.2.12	P	B	32	-	-	A	Dr	C	75	2	-2	32	6 hrs	1.8	4	IV
108.	Nagarathinam	28	4965	IV	20.2.12	G2	B	34	+	-	A	N/V	C	50	2	-1	34^{+2}	2	2.5	8	I
109.	Muthulakshmi	26	5273	IV	25.2.12	P	UB	30	-	-	B	H	C	50	1	-2	30^{+5}	5	1.1	8	IV
110.	Flora	24	5299	III	28.2.12	G2	UB	34	-	-	A	-	C	75	3	-2	34	4 hrs	2.5	8	
111.	Yashodha	28	5371	V	1.3.12	G2	UB	34	-	-	B	-	C	50	2	-2	34^{+4}	4	2.3	8	IV
112.	Chitra	27	5420	III	1.3.12	P	B	28	-	-	A	-	C	75	2	-1	2.8	4 hrs	1.2	3	II, VII
113.	Rajeshwari	20	6434	IV	2.3.12	P	B	31	-	-	B	Di	C	50	2	-1	31^{+3}	3	1.5	7	
114.	Renukadevi	23	6512	V	4.3.12	G3	B	30	-	+	B	-	B	25	1	-3	31^{+2}	9	1.4	6	
115.	Jyothi	21	6591	V	4.3.12	P	UB	31	-	-	A	F	C	25	1	-3	32	7	1.7	6	IV, V
116.	Poornima	26	6926	IV	4.3.12	G3	UB	32	+	-	A	-	C	25	1	-3	33^{+3}	10	1.9	8	
117.	Geetha	22	7537	V	6.3.12	P	B	30	-	-	B	P	C	75	2	-2	30	4 hrs	1.3	4	VII, V
118.	Manohari	34	7547	V	6.3.12	G2	B	34	-	-	A	-	C	50	1	-2	34^{+2}	2	2.3	8	IV, V
119.	Sukanya	18	7639	V	7.3.12	P	UB	33	-	-	B	H	C	50	1	-3	33^{+6}	6	2.2	8	I
120.	Alamelu	22	7789	V	8.3.12	P	UB	34	-	-	A	-	C	50	2	-1	35^{+5}	12	2.4	8	
121.	Thangammal	27	7812	V	10.3.12	G4	UB	32	-	-	B	-	C	25	1	-3	33	7	2.1	8	
122.	Revathi	29	8029	V	10.3.12	G2	B	30	-	-	A	-	B	50	2	-1	30	12 hrs	1.2	4	VII, II
123.	Kanaka	22	8106	V	11.3.12	P	UB	32	-	-	B	-	C	25	1	-3	32^{+5}	5	2.1	8	
124.	Poovathi	28	8255	IV	13.3.12	G2	B	33	+	-	A	-	C	50	1	-2	33^{+4}	4	2.3	6	
125.	Tharanika	27	8273	III	13.3.12	P	UB	31	-	-	B	-	C	50	1	-2	31^{+4}	4	2	8	V
126.	Mariyayee	23	8280	V	15.3.12	G2	B	33	-	-	A	H	C	25	2	-3	33^{+3}	3	1.6	8	
127.	Shakunthala	21	8285	III	17.3.12	P	B	31	-	-	A	-	C	25	2	-1	32	7	1.5	5	
128.	Shivagami	20	8569	III	18.3.12	G2	UB	34	-	-	B	-	C	50	2	-3	35	7	2.2	8	V, IV
129.	Amudha	21	8593	V	18.3.12	P	B	32	-	-	A	-	C	25	1	-2	33^{+2}	9	2.1	6	IV, V

130.	Vaidehi	23	8776	V	19.3.12	P	UB	34	-	-	B	H	C	75	2	-1	34^{+2}	2	2.2	8	
131.	Vijaya	25	8809	V	20.3.12	P	UB	34	-	-	A	F, Dr	C	50	1	-1	37^{+2}	23	2.6	8	
132.	Sheela	21	8969	V	24.3.12	P	B	34	-	-	B	-	C	50	2	-3	34^{+3}	3	2.3	8	V
133.	Renganayaki	27	8983	V	24.3.12	G2	UB	30	-	-	B	-	B	75	3	-2	30	6 hrs	1.1	3	
134.	Divya Bharathi	19	9440	V	27.3.12	P	UB	32	-	-	A	-	C	50	2	-2	32	12 hrs	2.2	6	III, VII
135.	Jerina Parveen	33	9477	V	29.3.12	P	B	30	-	-	B	-	C	75	2	-3	30	4 hrs	1.2	4	VII
136.	Ilaiyarasi	35	9503	V	29.3.12	G3	B	31	-	-	B	-	C	50	2	-3	31^{+3}	3	1.3	8	
137.	Gomathi	26	9508	IV	31.3.12	P	UB	29	-	-	A	H	C	25	1	-3	30	7	1.2	4	II, VII
138.	Lakshmi	28	9617	IV	3.4.12	G3	B	32	+	-	B	N/V	C	75	2	-2	32^{+2}	2	2.1	8	V, IV
139.	Yuvarani	30	9820	V	3.4.12	P	UB	33	-	-	A	-	C	25	1	-2	33^{+5}	5	2.2	8	
140.	Uma	25	10145	V	4.4.12	G2	B	34	-	-	B	-	C	50	2	-3	34^{+2}	2	2.2	8	
141.	Sheela Devi	26	10185	V	5.4.12	G2	B	31	-	-	A	-	C	50	2	-2	31^{+3}	3	1.7	8	
142.	Aruna	25	10196	IV	7.4.12	G2	UB	34	-	-	B	H	C	25	1	-3	35^{+3}	10	2.4	8	
143.	Amudha	25	10525	V	7.4.12	P	B	33	-	-	A	-	B	50	1	-2	33	15 hrs	1.9	7	
144.	Kalpana	22	10591	V	9.4.12	P	UB	33	-	-	B	-	C	25	2	-3	34^{+1}	8	2.2	8	V
145.	Panchavarnam	21	10620	IV	12.4.12	P	UB	32	-	-	A	H	C	50	2	-1	32	18 hrs	2.2	6	IV
146.	Kalaiyarasi	19	10663	IV	12.4.12	P	B	33	-	-	A	Dr	C	50	1	-3	33^{+6}	6	2.3	8	
147.	Chandradevi	28	10773	V	12.4.12	P	UB	31	-	-	B	Di	C	25	1	-3	31^{+6}	6	1.8	8	IV
148.	Bhagyalakshmi	22	10790	V	14.4.12	P	B	31	-	-	A	F	C	50	2	-1	31^{+5}	5	1.6	6	IV, V
149.	Arpudam	36	10830	IV	16.4.12	P	B	30	-	-	B	-	B	25	1	-3	30^{+6}	6	1.3	8	IV, V
150.	Chinnaponnu	26	10883	V	16.4.12	G2	UB	32	-	+	A	N/V	C	50	2	-3	32^{+2}	2	1.7	8	
151.	Pappathi	23	10964	III	17.4.12	G3	UB	29	+	-	B	H	C	25	1	-3	30	7	1.2	5	IV, V
152.	Veeramani	28	10991	V	19.4.12	P	B	28	-	-	B	-	C	75	2	0	28^{+3}	3	1.2	5	
153.	Praveena	23	11590	V	20.4.12	P	UB	34	-	-	A	-	C	25	1	-2	37^{+3}	24	2.3	8	
154.	Kamakshmi	21	11596	V	20.4.12	G2	B	32	-	-	B	P	C	75	2	-1	32^{+2}	2	2.1	8	
155.	Prabhadevi	27	11664	V	23.4.12	G2	UB	28	+	-	A	-	C	25	1	-1	28	1 day	1.1	3	I, III, VII
156.	Roja	33	11693	V	23.4.12	G3	UB	33	-	+	A	-	C	50	1	-3	33^{+4}	4	2.3	8	
157.	Sasikala	22	11790	III	25.4.12	P	UB	30	-	-	B	H	B	50	2	-1	31	7	1.2	6	IV
158.	Kala	24	12097	IV	25.4.12	P	B	34	-	-	B	-	C	75	2	-1	34^{+2}	2	2.5	8	
159.	Vijaya	21	12547	V	26.4.12	P	UB	33	-	-	A	-	C	50	1	-2	33^{+5}	5	2.2	8	
160.	Asha	24	12593	V	26.4.12	G3	UB	32	+	-	B	-	C	50	2	-1	32^{+6}	6	2.2	8	V
161.	Anitha	23	12651	V	27.4.12	G2	B	32	-	-	A	Dr, F	C	25	2	-3	32^{+3}	3	2.2	8	

162.	Parveen	21	12711	IV	28.4.12	P	B	31	-	-	B	-	B	50	1	-2	31^{+5}	5	2	8	IV
163.	Yogarani	21	13776	IV	28.4.12	P	UB	30	-	-	A	-	C	50	2	-3	30	12 hrs	1.4	3	II, VII
164.	Rukmani	27	15776	V	29.4.12	P	UB	32	-	-	B	-	C	75	3	-2	32^{+2}	2	2.5	8	
165.	Jeyamani	28	16876	III	30.4.12	G2	B	34	-	-	A	-	TL	25	2	-2	34^{+3}	3	2.2	8	
166.	Lalitha	33	16942	V	30.4.12	G2	UB	33	-	-	A	H, Dr	C	50	1	-1	34^{+1}	8	1.6	8	V
167.	Allirani	30	16998	IV	31.4.12	P	B	33	-	-	B	-	C	25	1	-3	33^{+4}	4	2.4	8	
168.	Kathun Beevi	19	17258	V	31.4.12	P	B	34	-	-	B	-	C	50	2	-2	34^{+4}	4	2.5	8	
169.	Elanjiyam	24	17741	V	1.5.12	P	UB	30	-	-	A	F	B	25	2	-2	31^{+6}	13	1.3	5	IV
170.	Arukkani	22	17846	III	2.5.12	G2	UB	28	-	-	B	P	C	50	2	-2	29^{+6}	13	1 kg	6	IV, V
171.	Vembu	20	18241	V	2.5.12	P	UB	31	-	-	A	-	C	25	2	-3	32^{+4}	11	2.2	8	IV
172.	Malarkodi	22	18361	V	2.5.12	P	B	34	-	-	B	-	B	50	2	-1	34^{+6}	6	2.4	8	
173.	Sulochana	22	18424	IV	4.5.12	G2	B	33	-	-	A	Dr, F	C	50	1	-1	33^{+4}	14	2.1	8	
174.	Jeyalakshmi	25	18444	V	4.5.12	P	UB	30	-	-	A	-	C	25	1	-2	31^{+5}	12	2.1	5	V
175.	Radha	27	18512	V	5.5.12	P	B	29	-	-	B	H	C	50	1	-2	29^{+4}	4	1.2	6	
176.	Eshwari	24	18567	V	6.5.12	P	B	33	-	-	A	-	C	75	2	0	33	3 hrs	2.3	5	
177.	Gowri	26	18742	V	7.5.12	P	UB	32	-	-	B	P	C	50	2	-2	32^{+5}	5	2.1	8	
178.	Gomathi	26	18861	IV	8.5.12	G2	UB	29	-	-	A	H	C	75	2	-2	29	6 hrs	1.2	4	II, VII
179.	Stellamary	23	19203	III	8.5.12	G2	UB	33	-	-	B	-	C	50	1	-2	33^{+4}	4	2.2	8	
180.	Anbumani	22	19381	III	9.5.12	P	B	32	-	-	A	Dr, PE	C	25	1	-3	32	1 day	2.3	7	V, VII
181.	Chitra	19	19421	IV	10.5.12	G2	B	34	+	-	B	Di	C	25	1	-2	37^{+2}	23	2.6	9	
182.	Nilofer	31	19549	III	12.5.12	P	UB	33	-	-	A	-	B	50	2	-1	33^{+6}	6	2.3	8	
183.	Thamarai	28	19687	V	12.5.12	G2	UB	30	-	+	A	N/V	C	75	2	-2	30^{+2}	2	1.5	6	IV
184.	Maheswari	27	19939	V	15.5.12	G2	UB	34	-	-	B	-	C	50	2	-2	34^{+3}	3	2.4	8	
185.	Anjalai	28	19982	IV	22.5.12	G3	B	32	-	-	A	-	C	75	2	-1	32^{+2}	2	2.1	8	
186.	Renuka	26	20006	IV	24.5.12	G2	B	33	-	-	B	-	C	75	3	-2	33^{+3}	3	2.3	8	V
187.	Rekha	29	20406	V	24.5.12	P	B	34	-	-	B	-	C	50	1	-2	37^{+3}	24	2.7	9	
188.	Jeyachandra	20	20442	III	25.5.12	P	UB	33	-	-	A	-	C	50	2	-2	33^{+5}	5	2.1	8	
189.	Kamakshi	26	20452	V	26.5.12	P	UB	32	-	-	A	-	B	50	2	-1	32^{+3}	3	2.2	8	IV
190.	Arul Devi	28	29835	V	30.5.12	G2	B	32	+	-	B	-	C	25	2	-2	32^{+3}	3	2	8	
191.	Geetha	27	20891	IV	6.6.12	G2	B	34	-	-	A	F	C	75	2	-3	34	8 hrs	2.4	8	
192.	Lakshmi	30	21372	V	10.6.12	G3	B	34	-	-	B	N/V	C	25	1	-2	37^{+2}	23	2.8	9	IV, V
193.	Malathi	23	21447	V	10.6.12	G3	B	31	-	-	A	-	C	75	2	-2	31^{+2}	2	1.4	8	

194.	Rajeshwari	22	21984	III	12.6.12	P	UB	30	-	-	A	Dr	C	50	1	-2	30	12 hrs	1.3	3	V, VII
195.	Malar	19	22474	IV	12.6.12	P	UB	32	-	-	B	-	C	50	2	-2	32^{+2}	2	2	8	
196.	Tamilarasi	34	22496	IV	14.6.12	G4	UB	34	-	-	B	H	TL	50	1	-1	37^{+4}	25	2.6	8	
197.	Selvarani	23	22512	V	22.6.12	P	UB	31	-	-	A	-	C	25	1	-3	31^{+3}	3	1.2	8	
198.	Hemapriya	33	22617	V	24.6.12	P	B	29	-	-	A	F	C	75	3	-2	29	3 hrs	1.1	3	III, VII
199.	Annalakshmi	21	23011	V	24.6.12	G3	UB	33	-	-	B	-	C	25	1	-2	33^{+4}	4	2.1	8	
200.	Muthuselvi	36	23311	IV	27.6.12	G4	UB	30	-	-	B	-	B	25	2	-2	30^{+5}	5	1.5	6	

ABBREVIATIONS

SES	-	Socioeconomic Status
DOA	-	Date of Admission
GA at Admn	-	Gestational Age at Admission
H/O 2nd TA	-	History of Second Trimester Abortion
H/O PTL	-	History of Preterm Labour
Mat S/E	-	Maternal Side Effects
GA at Del	-	Gestational Age at Delivery
TDI	-	Treatment Delivery Interval
B. Wt	-	Birth Weight
UB	-	Unbooked
B	-	Booked
F	-	Flushing
H	-	Headache
N/V	-	Nausea / Vomiting
Dr	-	Drowsiness
PE	-	Pulmonary Edema
Di	-	Dizziness
P	-	Palpitations
↓ BP	-	Hypotension

C	-	Cephalic
B	-	Breech
Tr	-	Transverse Lie
I	-	Hypothermia
II	-	Respiratory Distress Syndrome / Apnoea of Immaturity
III	-	Sepsis
IV	-	Feeding Problems
V	-	Hyperbilirubinemia
VI	-	Intraventricular Haemorrhage
VII	-	Neonatal Death