

CLASS IV

SUBSTANCE ABUSE DURING PREGNANCY

ALCOHOL

- CNS DEPRESSANT
- INCIDENCE HIGHEST IN MOTHERS 20-40 YEARS OF AGE
- PREGNANT WOMEN SHOULD AVOID ALCOHOL COMPLETELY
- ADVERSE MATERNAL EFFECTS

- ADVERSE FETAL/NEONATAL EFFECTS

COCAINE AND CRACK

- PREVENTS REUPTAKE OF DOPAMINE, NOREPINEEPHRINE--LEADS TO VASOCONSTRICTION, TACHYCARDIA, HYPERTENSION
- ADVERSE MATERNAL EFFECTS

- ADVERSE FETAL/NEONATAL EFFECTS

MARIJUANA

- NO STRONG RESEARCH INDICATING TERATOGENIC EFFECTS
- SOCIAL FACTORS

HEROIN, METHADONE

- ADVERSE MATERNAL, FETAL/NEONATAL EFFECTS

BARBITURATES

STIMULANTS

CAFFEINE

NICOTINE

PSYCHOTROPICS

DIABETES MELLITUS IN PREGNANCY

PATHOPHYSIOLOGY

1. INSULIN PRODUCTION DECREASE BY PANCREAS
2. WITHOUT ADEQUATE INSULIN, GLUCOSE DOES NOT ENTER CELLS, WHICH BECOME ENERGY DEPLETED
3. BLOOD GLUCOSE LEVELS INCREASE
4. CELLS BREAK DOWN PROTEIN AND FAT STORES FOR ENERGY
5. KETOACIDOSIS--COMBINATION OF KETOSIS AND ACIDOSIS--UNCONTROLLED DIABETES
 - a. KETOSIS--ACCUMULATION OF SUBSTANCES CALLED KETONE BODIES WHICH ARE CHEMICALS THAT BODY MAKES WHEN THERE IS NOT ENOUGH INSULIN IN BLOOD AND IT MUST BREAK DOWN FAT AND PROTEIN INSTEAD OF GLUCOSE FOR ENERGY. KETONES ARE MAINLY ACETONE WHICH ARE TOXIC; BUILD UP IN BLOOD AND SPILL OVER INTO URINE. NORMALLY KETONES REMOVED VIA KIDNEY AND URINE. IN DIABETES, KETONE FORMATION HAPPENS RAPIDLY--END UP IN BLOOD, WHICH MAKES IT ACIDIC. BLOOD GLUCOSE INCREASES
 - b. ACIDOSIS--INCREASED ACIDITY OF BLOOD--Ph LOW

CLASSIFICATIONS--BASED ON CAUSE

TYPE I

TYPE II

TYPE III

TYPE IV

WHITE'S CLASSIFICATION

CLASS A-T

DESCRIBES EXTENT OF DISEASE

DIABETES IN PREGNANCY

1. ESTROGEN, PROGESTERONE, OTHER HORMONES RISE TO STIMULATE INCREASED INSULIN PRODUCTION AND INCREASED TISSUE RESPONSE TO INSULIN
2. STORAGE OF GLYCOGEN IN LIVER PRODUCES ANABOLIC STATE DURING 1ST HALF OF PREGNANCY
3. 2ND HALF OF PREGNANCY PRESENTS WITH INCREASED RESISTANCE TO INSULIN AND DECREASED GLUCOSE TOLERANCE DUE TO:
 - a. SECRETION OF H_pL (INSULIN AGONIST), PROLACTIN, INCREASED CORTISOL AND GLYCOGEN LEVELS
 - b. RESULTS IN CATABOLIC EFFECT
 - c. LEADS TO INCREASING DEMANDS FOR INSULIN PRODUCTION WHICH LEADS TO PROGRESSIVE HYPERGLYCEMIA
 - d. CALLED DIABETOGENIC EFFECT

GESTATIONAL DIABETES

FIRST ONSET IN PREGNANCY
2-3% OF PREGNANCIES

CHALLENGES OF GDM

MATERNAL RISKS

FETAL/NEONATAL RISKS

CLINICAL MANAGEMENT

GTT CRITERIA

LAB ASSESSMENT

ANTEPARTAL MANAGEMENT

- DIET
- GLUCOSE MONITORING
-
- INSULIN REQUIREMENTS
- FETAL EVALUATION

INTRAPARTAL MANAGEMENT

- WHEN TO DELIVER
- LABOR MANAGEMENT
- INSULIN REQUIREMENTS

POSTPARTAL REQUIREMENTS

- INSULIN REQUIREMENTS
- BREAST FEEDING
- NEONATAL EVALUATION

ANEMIA IN PREGNANCY

IRON DEFICIENCY

SICKLE CELL

FOLIC ACID DEFICIENCY

HIV/AIDS IN PREGNANCY

PATHOPHYSIOLOGY

RISKS TO MOTHER AND FETUS

ANTEPARTUM, INTRAPARTUM, AND POSTPARTUM CARE

BLEEDING DISORDERS IN PREGNANCY

MAJOR CAUSE OF BLEEDING IS ABORTION (MISCARRIAGE)

- THREATENED
- IMMINENT
- COMPLETE
- MISSED

- HABITUAL
- SEPTIC

ECOTOPIC PREGNANCY
EGG IMPLANTS BEFORE REACHING UTERUS

CAUSES:

- CONGENITAL ANOMALIES OF TUBE
- PID
- PREVIOUS ECTOPIC

MANAGEMENT

- MEHOTREXATE
- SALPNIGOSTOMY
- SALPNIGECTOMY

GESTATIONAL TROPHOBLASTIC DIEASE

1 IN 1500/2000 PRENANCIES

ABNORMALLY DEVELOPED PLACENTA--GRAPELIKE TUMORS, TROPHOBLASTIC TISSUE GROWS

CAN DEVELOP INTO CHORIOCARCINOMA

COMPLETE--EMPTY EGG--NO MATERNAL GENETIC MATERIAL

PARTIAL--EGG/SPERM NEVER GO THROUGH 1ST MEOTIC DIVISION

CHORIOADENOMA DESTRUENS--INVOLVES MYOMETRIUM

INCOMPETENT CERVIX

PREMATURE DILATION OF CERVIX

CAUSED BY:

- INFECTION
- TRAUMA
- CONGENITAL CERVICAL ANOMALIES
- PREVIOUS INDUCED ABORTIONS

SHIRODKAR CERCLAGE

- REINFORCEMENT

- REMOVED WHEN LABOR OCCURS

HYPEREMESIS GRAVIDARUM

- EXCESSIVE VOMITING--USUALLY 1ST TRIMESTER
- MAY BE RELATED TO INCREASED HCG
- NOT TREATED--CAN LEAD TO METABOLIC ACIDOSIS

PREMATURE RUPTURE OF MEMBRANES

PROM

PPROM

MATERNAL/FETAL/NEONATAL RISKS

PRETERM LABOR

LABOR THAT OCCURS BETWEEN WEEKS 20-37

- PREVELANCE
- RESEARCH
- RECURRENT
- NONRECURRENT
- SCREENING
 - FETAL FIBRINECTIN
 - SALIVARY ESTRIOL
 - TVS

MANAGEMENT

- HOME UTERINE ACTIVITY MONITOR
- TOCOLYSIS
 - B-ADRENERGIC (B-MIMETIC) AGONISTS
 - MGSO₄
 - PROSTAGLANDIN SYNTHESIS INHIBITORS
 - BETAMETHASONE (FETUS)

HYPERTENSIVE DISEASE IN PREGNANCY

PREGNANCY INDUCED HYPERTENSION (PIH)

- PREECLAMPSIA/ECLAMPSIA
- CHRONIC HYPERTENSION
- CHRONIC HYPERTENSION WITH SUPERIMPOSED PREECLAMPSIA/ECLAMPSIA
- TRANSIENT HYPERTENSION

PREECLAMPSIA

DISEASE OF THEORIES

MOST COMMON HYPERTENSIVE DISEASE IN PREGNANCY

PATHOPHYSIOLOGY

- CAUSE UNKNOWN
- 5-7% OF ALL PREGNANCIES
- PROGRESSIVE

NORMAL PREGNANCY: LOWER PVR AND INCREASED RESISTANCE TO ANGIOTENSIN II, A DECAPEPTIDE AND POTENT VASOCONSTRICTOR

AFTER 20 WEEKS BP INCREASES PROBABLY DUE TO LOSS OF RESISTANCE TO ANGIOTENSIN II

PROSTACYCLIN (A PROSTOGLANDIN AND POTENT VASODILATOR) IS FOUND TO DECREASE IN PREECLAMPSIA

THROMBOXANE (A PROSTAGALNDIN AND POTENT VASOCONSTRICTOR) IS USUALLY FOUND IN EQUAL RATIO WITH PROSTACYCLIN

IN PREECLAMPSIA, *RATIO CHANGES*--MORE THROMBOXANE AND RESISTANCE TO ANGIOTENSIN II--LEADS TO *INCREASED VASOCONSTRICTION*

PREECLAMPSIA PRESENTS WITH VASOCONSTRICTON, INCREASED BP, DECREASED PLACENTAL PERFUSION, VASOSPASM, DECREASED GFR AND URINE OUTPUT

PROTEINURIA CAUSED BY STRETCHING OF CAPILLARY WALLS OF GLOMERULAR ENDOTHELIAL CELLS, ALLOW PROTEIN TO ESCAPE IN URINE

EDEMA CAUSED BY FLUID SHIFTS TO EXTRACELLULAR SPACES DUE TO VESSEL DAMAGE

PRENATAL FACTORS INCREASING RISK OF PIH

- PRIMIGRAVIDA
- ESSENTIAL HYPERTENSION
- AGE EXTREMES (UNDER 17, OVER 35)
- UNDERWEIGHT OR OVERWEIGHT
- FAMILY HISTORY OF HYPERTENSION
- DIAGNOSIS OF PIH IN PREVIOUS PREGNANCY
- DIABETES MELLITUS

CHARACTERIZED BY:

- DEVELOPMENT OF HYPERTENSION
 - 30MM HG INCREASE IN SYSTOLIC AND 15 MM HG DIASTOLIC OVER BASELINE ON AT LEAST 2 OCCASIONS 6 OR MORE HOURS APART

PROTEINURIA

EDEMA

HEADACHE

MATERNAL RISKS

FETAL/NEONATAL RISKS

CLINICAL MANAGEMENT

ANTEPARTAL

MILD PREECLAMPSIA

MODERATE PREECLAMPSIA

INTRAPARTAL

POSTPARTAL

HELLP SYNDROME

HEMOLYSIS

ELEVATED LIVER ENZYMES

LOW PLATELETS

ECLAMPSIA

Rh SENSITIZATION

Rh WOMAN EXPOSED TO Rh BLOOD, EITHER FROM TRANSFUSION OR A PRIOR PREGNANCY PRODUCES IMMUNOGLOBULIN (Ig) ANTIBODY (ANTIRhD)

INDIRECT COOMBS

DIRECT COOMBS

RhoGAM

ERYTHROBLASTOSIS FETALIS

HYDROPS FETALIS

ABO INCOMPATIBILITY

- USUALLY OCCURS WITH TYPE O MOTHER AND TYPE A OR B FETUS
- HIGH ANTI-A OR ANTI-B ANTIBODIES
- ANTENATAL ASSESSMENT

TORCH INFECTION

TOXOPLASMOSIS

OTHER

RUBELLA

CYTOMEGALOVIRUS

HERPES SIMPLEX

DYSFUNCTIONAL UTERINE CONTRACTIONS

HYPERTONIC LABOR

HYPOTONIC LABOR

- LABOR MANAGEMENT
- MATERNAL RISKS
- FETAL/NEONATAL RISKS

PRECIPITOUS LABOR

- LABOR LESS THAN 3 HOURS

POSTTERM PREGNANCY

- PREGNANCY 42 WEEKS PAST 1ST DAY OF LAST MENSTRUAL PERIOD
- MATERNAL RISKS
- FETAL/NEONATAL RISKS

MALPOSITION

- OCCIPUT POSTERIOR
- PERSISTENT OCCIPUT POSTERIOR
- LABOR MANAGEMENT
- MATERNAL/NEONATAL RISKS

MALPRESENTATION

- BROW
- FACE
- BREECH
- SHOULDER
- TRANSVERSE LIE
- COMPOUND PRESENTATION

MACROSOMIA

NEWBORN WEIGHT > 4000GMS

OFTEN SEEN IN:

- DIABETIC MOTHERS
- GRAND MULTIPARITY
- POSTTERM GESTATION
- LARGE PARENTS

MATERNAL RISKS

FETAL/NEONATAL RISKS

MULTIPLE GESTATION

- MONOZYGOTIC
 - SAME SEX (IDENTICAL)
 - ONE OVA
- DIZYGOTIC
 - TWO OVA
 - SAME OR DIFFERENT SEXES (FRATERNAL)
- MONOCHORIONIC
- DICHORIONIC

FETAL DISTRESS

CONTRIBUTING FACTORS:

- CORD COMPRESSION
- UTERO-PLACENTAL INSUFFICIENCY
- PREEXISTING MATERNAL OR FETAL DISEASE

FETAL DISTRESS WARNING SIGNS

- MECONIUM STAINED AMNIOTIC FLUID
- OMINOUS FHR PATTERNS
- PERSISTENT LATE DECELERATIONS
- PERSISTENT SEVERE VARIABLE DECELERATIONS
- PROLONGED DECELERATIONS

- DECREASED VARIABILITY

CLINICAL MANAGEMENT

FETAL DEATH

INTRAUTERINE FETAL DEATH (IUFD)

POSSIBLE CAUSES

- PREECLAMPSIA
- ABRUPTIO PLACENTAE
- PLACENTA PREVIA
- DIABETES
- CONGENITAL ANOMALIES
- INFECTION
- ISOIMMUNE DISEASE
- NUCAL CORD
- UNKNOWN CAUSES

PROLONGED RETENTION OF FETUS MAY LEAD TO:

DESSEMINATED INTRAVASCULAR COAGULOPATHY (DIC)

ABRUPTIO PLACENTAE

PREMATURE SEPARATION OF PLACENTA FROM UTERINE WALL

THREE CLASSIFICATIONS:

- MARGINAL
- CENTRAL
- COMPLETE

CLINICAL MANAGEMENT

PLACENTA PREVIA

IMPLANTATION OF PLACENTA IN LOWER UTERINE SEGMENT

THREE CLASSIFICATIONS:

- LOW PLACENTAL IMPLANTATION
- PARTIAL PLACENTA PREVIA
- TOTAL PLACENTA PREVIA

CLINICAL MANAGEMENT

DIFFERENCES BETWEEN ABRUPTIO PLACENTAE AND PLACENTA PREVIA

PROLAPSED UMBILICAL CORD

PROLAPSED CORD: WHEN CORD PRECEDES FETAL PRESENTING PART

DECREASED BLOOD FLOW IN CORD LEADS TO FETAL DISTRESS

MAY RESULT AFTER RUPTURE OF MEMBRANES

CLINICAL MANAGEMENT

AMNIOTIC FLUID EMBOLISM

AMNIOTIC FLUID MAY LEAK INTO CHORIONIC PLATE AND INTO MATERNAL CIRCULATORY SYSTEM THROUGH:

- TEAR IN AMNION OR CHORION
- PLACENTAL SEPARATION
- CERVICAL TEAR

CLINICAL PRESENTATION

- CHEST PAIN
- DYSPNEA
- CYANOSIS
- HYPOTENSION
- TACHYCARDIA
- MASSIVE HEMORRHAGE

CLINICAL MANAGEMENT

HYDRAMNIOS >2000ML AMNIOTIC FLUID

CAUSE UNKNOWN--20% ASSOCIATED WITH CONGENITAL ANOMALIES

TWO TYPES:
CHRONIC

ACUTE

CLINICAL MANAGEMENT

OLIGOHYDRAMNIOS

SEVERELY REDUCED AMOUNT OF AMNIOTIC FLUID

OCCURS AT TIMES WITH:

- POSTMATURITY
- IUGR
- FETAL RENAL MALFORMATION

CLINICAL MANAGEMENT

CEPHALOPELVIC DISPROPORTION (CPD)

FETUS LARGER THAN PELVIC DIAMETERS

PELVIC MEASUREMENTS

PROLONGED LABOR

CLINICAL MANAGEMENT

LACERATIONS

1ST DEGREE

2ND DEGREE

3RD DEGREE

4TH DEGREE

PLACENTA ACCRETA

ATTACHMENT OF PLACENTA DIRECTLY TO THE UTERINE WALL WITHOUT INTERVENING DECIDUA BASALIS

MAY PENETRATE MYOMETRIUM

CLINICAL MANAGEMENT

VERSION

EXTERNAL CEPHALIC VERSION:

EXTERNAL MANIPULATION USED TO CONVERT BREECH TO CEPHALIC PRESENTATION

INTERNAL VERSION

USED TO DELIVER SECOND TWIN DURING VAGINAL BIRTH IF NOT DESCENDING OR IN DISTRESS--RARE

AMNIOTOMY

ARTIFICIAL RUPTURE OF MEMBRANES (AROM)

AMNIHOOK--MUST BE AT LEAST 2 CM DILATED

AFTER 3 CM AROM WILL PROBABLY SHORTEN LABOR (IS A STIMULATION OF LABOR)

FHR ASSESSED BEFORE AND AFTER AROM

CERVICAL RIPENING

PROSTAGLANDIN GEL

CYTOTEC

HAVE BEEN SHOWN TO CAUSE CERVICAL RIPENING, SHORTER LABORS, DECREASED USE OF OXYTOXIN

BALLOON

LABOR INDUCTION

STIMULATION OF UTERINE CONTRACTIONS BEFORE SPONTANEOUS ONSET OF LABOR

INDICATED INDUCTION:

- DIABETES
- RENAL DISEASE
- PIH
- PROM
- POSTTERM GESTATION
- IUFD
- IUGR
- ISOIMMUNIZATION

FETAL MATURITY

CERVICAL READINESS

OXYTOCIN STIMULATION

ELECTIVE INDUCTION--CONTROVERSY

AMNIOINFUSION

USED INTRAPARTALLY TO INCREASE FLUID VOLUME DUE TO OLIGOHYDRAMNIOS

DECREASES PRESSURE ON CORD

PROMOTES INCREASED PERFUSION TO FETUS

CAN DILUTE MODERATE TO HEAVY MECONIUM FLUID

USED IN PRETERM LABOR WITH PPROM

CAN BE USED WHEN SEVERE VARIABLE DECELERATIONS PRESENT

EPISIOTOMY

SURGICAL INCISION TO PERINEUM TO ENLARGE OUTLET

RESEARCH

PREVENTATIVE MEASURES

TWO TYPES:

- MEDIAN
- MEDILATERAL

FORCEPS DELIVERY

PROVIDES TRACTION, ROTATION

THREE CATEGORIES:

- OUTLET FORCEPS
- LOW FORCEPS
- MID FORCEPS

ADVERSE EFFECTS

VACUUM ASSISTED BIRTH

SUCTION CUP APPLIED TO FETAL HEAD; PUMP CREATES NEGATIVE PRESSURE AND ALONG WITH TRACTION AND UTERINE CONTRACTIONS FACILITATES DESCENT OF FETAL HEAD

ADVERSE EFFECTS

CESAREAN BIRTH

C-BIRTH RATE VARIES--CAN BE AS HIGH AS 55% AT SOME INSTITUTIONS

INDICATIONS:

- COMPLETE PLACENTA PREVIA
- CPD
- ABRUPTION
- ACTIVE GENITAL HERPES
- PROVEN FETAL DISTRESS
- PROLAPSED CORD
- BENIGN AND MALIGNANT TUMORS
- CERVICAL CERCLAGE
- FAILURE TO PROGRESS

CONTROVERSIAL INDICATIONS:

- BREECH
- PREVIOUS C-BIRTH
- MAJOR CONGENITAL ANOMALIES

SKIN INCISIONS

- TRANSVERSE (PFANNENSTIEL)
- VERTICAL

UTERINE INCISIONS

- TRANSVERSE
- SELHEIM (LOWER UTERINE SEGMENT)
- CLASSIC (UPPER SEGMENT OF CORPUS)

PREPARATION FOR C-BIRTH

REPEAT C-BIRTH

EMERGENCY C-BIRTH

VBAC VAGINAL BIRTH AFTER CESAREAN

MORE CONSUMER DEMAND

INDICATIONS:

PREVIOUS C-BIRTH, LOW TRANSVERSE UTERINE INCISION

MUST BE IN AN INSTITUTION WHERE C-BIRTH CAN BE PERFORMED WITHIN 30 MINUTES WITH PHYSICIAN AVAILABLE

CONTRAINDICATED WITH PREVIOUS CLASSIC UTERINE INCISION

NONRECURRING INDICATIONS

