# CLASS V

**ASSESSMENT OF FETAL WELL BEING**

**MATERNAL ANTENATAL RISK FACTORS**

- MATERNAL AGE> 16, < 35 YEARS OLD
- Rh ISOIMMUNIIZATION
- HISTORY OF UNEXPLAINED STILLBIRTH
- SUSPECTED IUGR
- POSTDATES GESTATION
- PIH, DIABETES MELLITUS, CARDIAC DISEASE
- TORCH DISEASES
- HIV/AIDS
- SUBSTANCE ABUSE
- PHYSICAL ABUSE
- MULTIPLE GESTATION
- LOW SOCIOECONOMIC LEVEL OF MOTHER
- ENVIRONMENTAL HAZARDS

**ULTRASOUND**

TRANSABDOMINAL

ENDOVAGINAL

USE OF HIGH FREQUENCY WAVES TO VISUALIZE STRUCTURES OF VARYING DENSITIES

- NONINVASIVE, PAINLESS
- RESEARCH HAS FOUND NO HARMFUL EFFECTS  NEW RESEACH???

CLINICAL (DIAGNOSTIC) APPLICATIONS

- FHR, FBM
- EARLY DETECTION OF PREGNANCY
- MEASUREMENT OF BPD
- MULTIPLE GESTATION
- ESTIMATION OF FETAL BIRTH WEIGHT
- DETECTION OF ANOMALIES
- AMNIOTIC FLUID INDEX  (AFI)
- PLACENTA LOCATION

- DETECTION OF IUFD
- DETECTION OF IUGR
- FETAL POSITION, PRESENTATION
- USED WITH AMNIOCENTESIS, PBS, BPP, DOPPLER FLOW STUDIES

**DOPPLER BLOOD FLOW VELOCITY**

ULTRASOUND BEAM DIRECTED AT UMBILICAL ARTERY

ASSESSMENT OF PLACENTAL FUNCTION BY MEASURING BLOOD FLOW CHANGES IN MATERNAL AND FETAL CIRCULATION

SIGNAL REFLECTED OFF CIRCULATING RBCS—CREATES WAVELIKE PATTERN

**NONSTRESS TEST**

ACCELERATIONS IN FETAL HEART RATE WITH FETAL MOVEMENT INDICATES ADEQUATE OXYGENATION AND INTACT CNS (FETAL WELL BEING)

REACTIVE TEST

NON-REACTIVE TEST

**BIOPHYSICAL PROFILE**

FIVE COMPONENTS

- NST
- FETAL BREATHING MOVEMENTS
- FETAL TONE
- FETAL MOVEMENTS
- AMNIOTIC FLUID INDEX

**AMNIOCENTESIS**

NEEDLE INSERTED THROUGH ABDOMINAL WALL INTO UTERUS DURING ULTRASOUND TO OBTAIN AMNIOTIC FLUID SAMPLE FOR ANALYSIS

DIAGNOSTIC CRITERIA

ADVANTAGES, RISKS

**CHORIONIC VILLI SAMPLINF (CVS)**

SMALL SAMPLE OF CHORIONIC VILLI OBTAINED FOR TESTING

ADVANTAGES, RISKS

**PERCUTANEOUS UMBILICAL BLOOD SAMPLING (PUBS)**

BLOOD SAMPLE TAKEN FROM UMBILICAL CORD IN UTERO UNDER ULTRASOUND THROUGH MATERNAL ABDOMEN AND UTERUS

BLOOD DISORDERS

CHROMOSOME ABNORMALITIES

FETAL KAROTYPING

***NEWBORN AT RISK***

RISK FACTORS SAME FOR NEWBORN AS FOR MOTHER

NEWBORN WEIGHT AND GESTTIONAL AGE

LARGE FOR GESTATIONAL AGE (LGA)

SMALL FOR GESTATIONAL AGE (SGA)

BIRTH WEIGHT AND GESTATIONAL AGE ARE USED TOGETHER TO ASSESS NEONATAL MATURITY AND MORTALITY RISK

SGA INFANT MAY BE:

PRETERM

POSTTERM

TERM

LGA INFANT MAY BE:

PRETERM

POSTTERM

**INTRAUTERINE GROWTH RESTRICTION (IUGR)**

FACTORS CONTRIBUTING TO IUGR:

- MATERNAL DISEASE
- MATERNAL FACTORS
- ENVIRONMENTAL FACTORS
- FETAL FACTORS

SYMMETRIC

ASYMMETRIC

COMPLICATIONS RELATING TO IUGR

- POLYCYTHEMIA
- HYPOGYCEMIA
- HEAT LOSS
- ASPIRATION SYNDROME
- PERINATAL ASPHYXIA
- HYPOCALCEMIA
- INTRAUTERINE INFECTIONS
- CONGENITAL MALFORMATIONS

- LEARNING DIFFICULTIES
- GROWTH GAPS

LGA

FACTORS ASSOCIATED WITH LGA

DIABETES

GENETIC PREDISPOSITION

MALE INFANTS

VARIOUS SYNDROMES

MULTIPARITY

***INFANT OF DIABETIC MOTHER (IDM)***

FETUS EXPERIENCES EXCESSIVE GROWTH DUE TO:

- EXPOSURE TO HIGH MATERNAL GLUCOSE LEVELS—GLUCOSE READILY CROSSES THE PLACENTA RESULTING IN FETAL BLOOD SUGAR LEVELS 80% OF MATERNAL LEVELS
- FETUS RESPONDS BY PRODUCING LARGE QUANTITIES OF INSULIN (HYPERINSULINEMIA)
- INCREASED AMOUNTS OF GLUCOSE PRESENT IS RESERVED AS GLYCOGEN STORES AND RESULTS IN MACROSOMIA
- INSULIN ACTS AS A GROWTH HORMONE IN FETUS
- HYPERINSULINEMIA ALSO PRODUCES MACROSOMIA FROM INCREASED HEPATIC GLYCOGEN AND TOTAL BODY FAT STORES
- AFTER DELIVERY, WHAT HAPPENS??

COMPLICATIONS OF IDM

- ❖ HYPOGLYCEMIA
- ❖ HYPOCALCEMIA

- ❖ HYPERBILIRUBENEMIA

- ❖ BIRTH TRAUMA

- ❖ RESPIRATORY DISTRESS SYNDROME—WHITES'S CLASS A-C. EXCESS INSULIN PRODUCTION BY FETUS' PANCREAS RESULTS IN DELAYED SURFACTANT PRODUCTION

- ❖ CONGENITAL BIRTH DEFECTS

CLINICAL MANAGEMENT

BLOOD GLUCOSE LEVELS FROM CORD BLOOD

IDM WITH GLUCOSE LEVEL BELOW 40MG/Dl ARE GIVEN EARLY FEEDING

IF NORMAL GLUCOSE LEVELS NOT MAINTAINED IV GLUCOSE INITIATED

**POSTTERM INFANT**

BORN AFTER 42 WEEKS GESTATION

POSTMATURITY SYNDROME

- HYPOGLYCEMIA
- MECONIUM ASPIRATION
- POLYCYTHEMIA
- CONGENTIAL ANOMALIES
- SEIZURE ACTIVITY
- COLD STRESS

**PRETERM INFANT**

PRETERM AND SGA INFANTS HAVE THE HIGHEST MORTALITY RISK

- ➢ IMMATURITY OF ALL SYSTEMS

- ➢ RESPIRATORY/CARDIOVASCULAR

- THERMOREGULATION
- RENAL
- REACTIVITY/BEHAVIORAL STATES
- NUTRITION AND FLUID REQUIREMENTS

COMPLICATIONS

RESPIRATORY DISTRESS SYNDROME

**INTRAVENTRICULAR HEMORRHAGE (IVH)**

**PATENT DUCTUS ARTERIOSIS (PDA)**

APNEA

ANEMIA

**RETINOPATHY OF PREMATURITY (ROP)**

**BRONCHOPULMONARY DISPLASIA**

**NECROTIZING ENTERCOLITIS (NEC**)

LONG TERM NEEDS AND OUTCOME

NEUROLOGIC DEFECTS

AUDITORY DEFECTS

SPEECH DEFECTS

**INFANT OF SUBSTANCE ABUSING MOTHER**

FETAL ALCOHOL SYNDROME (FAS)

COMPLICATIONS

CHARACTERISTICS

CLINICAL MANAGEMENT

**INFANT OF A DRUG DEPENDENT MOTHER**

RISKS

- INTRAUTERINE ASPHYXIA
- INTRAUTERINE INFECTION
- ALTERATION IN BIRTH WEIGHT
- LOW APGAR SCORES
- IUGR

COMPLICATIONS AFTER BIRTH

RESPIRATORY DISTRESS

JAUNDICE

CONGENITAL ANOMALIES

BEHAVIORAL ABNORMALITIES

WITHDRAWAL

HEROIN

COMPLETE ANE EARLY PRENATAL CARE
METHADONE PROGRAM

**INFANT WITH CONGENITAL ANOMALY**

- CHOANAL ATRESIA
- HYDOCEPHALUS
- CLEFT LIP, PALATE
- TRACHEOESOPHAGEAL FISTULA
- DIAPHRAGMATIC HERNIA
- MYELOMENINGIOCELE, IMPERFORATE ANUS
- OMPHALOCELE, GASTROCHISIS

***NEWBORN WITH CONGENITAL HEART DEFECT***

ACYANOTIC

- PDA

- ASD

- VSD

- COARCTATION OF THE AORTA
- HYPOPLASTIC LEFT HEART SYNDROME

CYANOTIC

- TETROLOGY OF FALLOT
- TRANSPOSITION OF THE GREAT VESSELS

**NEWBORN WITH INBORN ERROR OF METABOLISM**

PKU—LACKS ABILITY TO CONVERT EXCESS PHENYLALANINE, AN ESSENTIAL AMINO ACID THE BODY USES FOR GROWTH, TO TYROSINE. EXCESSIVE ACCUMULATION OF PHENYLALANINE LEADS TO PROGRESSIVE MENTAL REGRESSION

MAPLE SYRUP URINE DISEASE

GALACTOSEMIA

CONGENTIAL HYPOTHYROIDISM

TESTING

PKU GUTHRIE TEST
HEEL STICK
WHEN IS THIS PERFORMED?

**INFANT WITH ASPHYXIA**

CIRCULATORY PATTERNS ASSOCIATED WITH ASPHYXIA

- ✓ FAILURE OF LUNG EXPANSION AND ESTABLISHMENT OF RESPIRATION PRODUCES HYPOXIA, ACIDOSIS, AND HYPERCARBIA

- ✓ THESE BIOCHEMICAL CHANGES CAUSE:

    - HIGH PULMONARY VASCULAR RESISTANCE
    - PULMONARY VASOCONSTRICTION
    - HYPOPERFUSION OF LUNGS

LARGE RIGHT TO LEFT SHUNT THROUGH THE DUCTUS ARTERIOSIS

FORAMEN OVALE OPENS, BLOOD THEN FLOWS RIGHT TO LEFT

METABOLIC ACIDOSIS OCCURS

RESPIRATORY ACIDOSIS MAY OCCUR

FREE FATTY ACIDS AND GLYCEROL INCREASE IN BLOOD

GLYCOGEN STORES MOBILIZED FOR CONTINUOUS GLUCOSE SOURCE FOR BRAIN

HEPATIC AND CARDIAC STORES OD GLYCOGEN CAN BE USED UP REAPIDLY DURING ASPHYXIC ATTACK

PROLONGED ASPHYXIA CAN RESULT IN BRAIN DAMAGE, EATH

RISK FACTORS

- NONREASSURING FETAL HEART RATE PATTERNS
- DIFFICULT BIRTH
- FETAL BLOOD LOSS
- APNEIC EPISODE UNRESPONSIVE TO TACTILE STIMULATION
- INADEQUATE VENTILATION
- PREMATURITY
- STRUCTURAL LUNG ABNORMALITY
- CARDIAC ARREST

CLINICAL MANAGEMENT

INTRPARTAL

BPP

SCALP Ph

FHR TRACING

**INFANT WITH RESPIRATORY DISTRESS**

PRECIPITATING FACTORS

- PREMATURITY
- SURFACTANT DEFICIENCY DISEASE
- PHYSIOLOGIC ALTERATIONS
- HYPOXIA
- RESPIRATORY ACIDOSIS
- METABOLIC ACIDOSIS

CLINICAL MANAGEMENT

**TRANSIENT TACHYPNEA OF THE NEWBORN**

- MATERNAL OVERSEDATION
- PROLAPSED CORD
- BREECH BIRTH
- MATERNAL DIABETES
- MATERNAL BLEEDING
- CESAREAN BIRTH

CLINICAL PRESNTATION

1.

2.

3.

4.

CLINICAL MANAGEMENT

**MECONIUM ASPIRATION SYNDROME**

PATHOPHYSIOLOGY

ALVEOLI DISTENDED DUE TO AIR ALLOWED IN BUT OBSTRUCTION OF AIR OUTFLOW DURING EXPIRATION DUE TO MECONIUM IN LUNGSD—LEADS TO INSPIRED AIR TRAPPING IN ALVEOLI AND AIR LEAK (PHEUMOTHORAX AND PNEUMOMEDIASTNUM 20-30%)

BILE SALTS AND PANCREATIC ENZYMES IN MECONIUM CAUSE A CHEMICAL PNEUMONITIS

RISK FACTORS

PROLONGED LABOR POSTTERM
INFANT OF SUBSTANCE ABUSING MOTHER

BREECH

CLINICAL MANIFESTATIONS

1.

2.

3.

4.

5.

6.

CLINICAL MANAGEMENT

- INITIAL RESUSCITATION
- MECHANICAL VENTILATION
- CHEST X-RAY
- ABGS
- SURFACTANT REPLACEMENT THERAPY
- ECHMO (EXTRAL CORPORAL MEMBRANE OXYGENATATION)

**NEWBORN WITH COLD STRESS**

PATHOPHYSIOLOGY

COLD STRESS RESULTS FROM HEAT LOSS THROUGH:

1.
2.
3.
4.

OXYGEN REQUIREMENTS RISE, GLUCOSE USE INCREASES, ACIDEMIA OCCURS, AND SURFACTANT PRODUCTION DECREASES

AMOUNT OF HEAT LOSS DEPENDS MANY TIMES ON THE ACTION OF THE CARE GIVER

RISK FACTORS

- PRETERM AND SGA—HAVE DECREASED BROWN FAT, ADIPOSE TISSUE, AND GLYCOGEN STORES
- NONSHIVERING THERMOGENESIS—BROWN FAT METABOLISM OFTEN NOT PRESENT
- HYPOXEMIA, INTERCRANIAL HEMORRHAGE, CNS ABNORMALITY, AND HYPOGLYCEMIA INHIBIT NEWBORN TO RESPOND TO COLD STRESS BY NONSHIVERING THERMOGENESIS

CLINICAL MANAGEMENT

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7.

**HYPOGLYCEMIA**

PLASMA GLUCOSE CONCENTRATION OF 40MG/DL OR LESS

NEWBORN MAY BE ASYMPTOMATIC

IDENTIFICATION

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6.

7.

DIFFERENTIAL DIAGNOSIS

- CNS DISEASE
- SEPSIS
- METABOLIC IRREGULARITIES
- POLYCYTHEMIA
- CHD
- DRUG WITHDRAWAL

- INSTABLE TEMPERATURE

- HYPOCALCEMIA

CLINICAL MANAGEMENT

EARLY BREAST/BOTTLE FEEDING IS ONE OF FIRST MAJOR PREVENTATIVE APPROACHES

ASYMPTOMATIC NEWBORNS WITH GLUCOSE LEVELS 30-49MG/DL GIVEN BREAST/FORMULA FEEDINGS, OR ORAL GLUCOSE

PLASMA GLUCOSE MEASUREMENT OBTAINED 30-60 MINUTES AFTER FEEDING

NEWBORNS WITH PLASMA GLUCOSE LEVELS OF 20-25 MG/DL ARE TRATED WITH IV GLUCOSE

RAPID INFUSION OF 20-25% GLUCOSE CONTRAINDICATED—MAY LEAD TO REBOUND HYPOGLYCEMIA

**NEWBORN WITH JAUNDICE**

MAJOR CAUSE IS HEMOLYTIC DISEASE OF NEWBORN

CONSIDERED PATHOLOGIC IF:

- JAUNDICE EVIDENT IN 1ST 24 HOURS OR AFTER 4 DAYS OF LIFE
- SERUM BILIRUBIN >5MB/DL IN ONE DAY
- TOTAL SERUM BILIRUBIN CONCENTRATION EXCEED 12.9MG/DL IN TERM INFANTS;15MG/DL IN PRETERM
- CLINICAL JAUNDICE PERSISTS BEYOND 7 DAYD TERM, 14 DAYS PRETERM

CLINICAL MANAGEMENT

PHOTOTHERAPY

FIBER OPTIC BLANKET

EXCHANGE TRANSFUSION

ALBUMIN INFUSION

**NEWBORN WITH ANEMIA, POLYCYTHEMIA**

PHYSIOLOGIC ANEMIA—EXPECTED, GRADUAL DROP IN HEMOGLOBIN $1^{ST}$ 6-12 WEEKS OF LIFE

CLINICAL MANAGEMENT DEPENDS ON SEVERITY AND IF BLOOD LOSS IS ACUTE OR CHRONIC

POLYCYTHEMIA

BLOOD VOLUME AND HEMACRIT INCREASED

COMMON IN:

- SGA INFANTS
- FULL TERM INFANTS WITH DELAYED CORD CLAMPING
- TWIN TO TWIN TRANSFUSION
- MATERNAL TRANSFUSION
- CHRONIC INTRAUTERINE HYPOXIA

SYMPTOMATIC INFANT RECEIVES PARTIAL EXCHANGE TRANSFUSION

**NEWBORN WITH INFECTION (SEPSIS NEONATORUM)**

RISK FACTORS, PREDISPOSING FACTORS

PREMATURITY
LOW BIRTH WEIGHT
MATERNAL ANTENATAL INFECTION

MOST COMMON CAUSATIVE AGENTS

GRAM NEGATIVE

E-COLI
ENTEROBACTOR
PROTEUS
KLEBSIELLA

GRAM POSITIVE

GROUP B STREP
PSEUDOMONAS
STAPHYLOCOCCUS

NOSOCOMIAL INFECTION

ASSESSMENT

- LETHARGIC OR IRRITABLE
- TEMPERATURE INSTABILITY
- PALLOR, DUSKINESS, CYANOSIS, COOL, CLAMMY SKIN
- FEEDING INTOLERANCE
- HYPERBILIRUBINEMIA

CLINICAL MANAGEMENT

1.

2.

3.

4.

5.

**EARLY POSTPARTAL HEMMORHAGE**

1$^{ST}$ 24 HOURS AFTER BIRTH

UTERINE ATONY

- PROLONGED LABOR
- OVERDISTENTION OF UTERUS

- OXYTOCIN
- RETAINED PLACENTAL FRAGMENTS
- GRAND MULTIPARITY
- PIH
- INTRA-AMNIOTIC INFECTION
- ASIAN OR HISPANIC HERITAGE
- USE OF ANESTHESIA

FULL BLADDER CAN INCREASE RISK OF POSTPARTAL HEMMORHAGE

VULVAR, VAGINAL, PELVIC HEMATOMAS

LACERATIONS OF REPRODUCTIVE TRACT

CLINICAL MANAGEMENT

**LATE POSTPARTAL HEMORRHAGE**

24 HOURS TO 6 WEEKS AFTER BIRTH

USUALLY DUE TO SUBINVOLUTION OF PLACENTAL SITE DUE TO RETAINED PLACENTAL FRAGMENTS

CLINICAL MANAGEMENT

PUERPURAL INFECTION

ENDOMETRITIS

PELVIC CELLULITIS

PERINEAL WOUND INFECTIONS

CESAREAN WOUND INFECTIONS

URINARY TRACT INFECTION

- OVERDISTENTION OF BLADDER
- INABILITY TO VOID
- CYSTITIS

**MASTITIS**

**THROMBOEMBOLITIC DISEASE**

CONTRIBUTING FACTORS

- INCREASE IN CERTAIN BLOOD CLOTTING FACTORS
- POSTPARTAL THROMBOCYTOSIS
  RELEASE OF THROMBOPLASTIN SUBSTANCES FROM TISSUE OF DECIDUA, PLACENTA, AND FETAL MEMBRANES

PREDISPOSING FACTORS

- OBESITY
- INCREASED MATERNAL AGE
- HIGH PARITY
- ANESTHESIA AND SURGERY
- PREVIOUS HISTORY OF THROMBOSIS
- MATERNAL ANEMIA, HYPOTHERMIA, HEART DISEASE
- ENDOMETRITIS
- VARICOSITIES

SUPERFICIAL VEIN THROMBOPHLEBITIS

DEEP VEIN THROMBOSIS

SEPTIC VEIN THROMBOSIS

**POSTPARTAL PSYCHIATRIC DISORDERS**

POSTPARTUM DEPRESSION

POSTPARTUM PSYCHOSIS