randomized into: pentoxifylline/hypothermia group (n = 10; received hypothermia and intravenous pentoxifylline once daily for 3 successive days) and hypothermia group (n = 10 received hypothermia and an equivalent dose of placebo). Cranial ultrasound and amplitude EEG were done at enrollment. Serum malondialdehyde (MDA) was measured before enrollment and at day 7.

**Results:** MDA is higher in stage III HIE compared to stage II and in non-survivors compared to survivors (P < 0.001). Mortality in the hypothermia group is double the pentoxifylline/hypothermia group. We found no statistical difference between the two groups regarding short-term clinical outcome or serum MDA.

**Conclusion:** Pentoxifylline combined with hypothermia therapy of great value in HIE neonates.

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**Thyroid hormone dysfunction in critically ill fullterm neonates with sepsis**

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**Introduction:** Neonates are usually susceptible to sepsis with non-specific clinical manifestations. Neonatal sepsis is an alarming condition resulting in high morbidity and mortality. Alteration in thyroid hormone levels is usually observed in hospitalized patients with critical illness. With increasing severity of illness, the levels of total thyroxine (TT4), free thyroxin (fT4) and thyroid stimulating hormone (TSH) may also decrease.

**Objectives:** This study aims to assess the thyroid hormone levels and CRP levels in neonates with sepsis and correlating these levels with disease severity.

**Patients and Methods:** This case control study was carried out over 12 months on 50 critically ill fullterm newborns admitted to the Neonatal Intensive Care Unit (NICU) in Ain-Shams University Hospitals. Fifty healthy fullterm newborns served as controls. All cases underwent detailed history taking including maternal medical conditions, maternal infections, maternal drug intake, maternal hypo- and hyperthyroidism. Presence of PROM, meconium staining, Apgar scoring at one minute and at 5 minutes, neonatal resuscitation and congenital malformations. Neonates were diagnosed using sepsis score. CBC, blood culture and CRP were done for all neonates on 3rd day and on 10th day of antibiotic therapy. Serum total T3 (TT3), T4 (TT4), and TSH were determined and compared with age matched reference values.

**Results:** From the 50 sick neonates; 32 (64%) were survivors and 18 (36%) were non-survivors. 52% had PROM, 46% needed ventilation, 64% were discharged. A mortality rate of 36% was recorded. On day 3; there was low T3 with mean of 58.77 ± 17.07 ng/dl, low T4 (mean= 2.10 ± 2.57ug/dl) and high TSH levels (6.73 ± 2.08uU/ml). However, on day 10; serum T3 returned to be within normal range, with mean of 114.38 ± 26.5ng/dl, serum T4 returned to normal range (11.72 ± 2.54ug/dl) and TSH was lowered to half its value to reach normal levels (mean= 3.17 ± 2.57 uU/ml). T4 was significantly correlated to the septic clinical parameters, the diastolic BP, the WBC count, hemoglobin level, Neutrophil count and the CRP levels (P values < 0.05). TSH was significantly correlated to the septic clinical parameters, the HR, hemoglobin levels, and monocyte count (P values < 0.05).

**Conclusion:** Hypothyroxinemia has considerable prevalence in neonatal intensive care setting and is related with critical illness as neonatal sepsis.

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**Left ventricle myocardial performance in down syndrome children with clinically and anatomically normal hearts: relationship to oxidative stress**

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Down syndrome (DS) represents a human disorder in which oxidative stress is implicated in many organs pathophysiologies. Very scarce data exist concerning left ventricle (LV) performance in DS children with clinically and anatomically normal hearts, especially in the pediatric age group. Tissue Doppler derived myocardial performance index (TDI-Tei index) proved to be a reliable method for ventricular performance evaluation. Myeloperoxidase (MPO) enzyme plays a crucial role in inflammation and in oxidants production and is a marker of cardiovascular risk.

**Aim:** to evaluate LV myocardial performance in DS children with clinically and anatomically normal hearts using tissue Doppler derived myocardial performance index (TDI-Tei index) and correlate it with plasma myeloperoxidase as a marker of oxidative stress in those children.

**Patients and Methods:** This cross sectional study include 120 DS children recruited from Children s Hospital, Ain Shams university Out patients clinic and echocardiography unit(mean age 8.35± 4.25 years)who were subjected to: thorough history taking, clinical general and cardiac examination, laboratory investigations (CBC, ALT, serum creatinine, TSH, FT3 and T4).12 lead ECG as well as 2D, Mmode, color, pulsed and continuous wave Doppler echocardiography.DS children with congenital or acquired heart diseases, dysrhythmias, anemia, pulmonary hypertension, thyroid, renal diseases, diabetes were excluded from the study. The remaining 50 DS children with anatmically and clinically normal hearts (gpI) were compared to 50 age and sex matched healthy children as control(gpII). Studied groups were subjected to: plasma myeloperoxidase (MPO) level assessment (ELISA) and LV TDI-Tei indexes evaluation (VividE9, Vingmed, GE, Horten, Norway).

**Results:** DS children (gpI) had normal LV systolic functions by conventional echocardiography (EF 68.2± 3.9% VS 67± 4.5% in gpII).LVTDI-Tei index was significantly increased in gpI compared to GPII(0.46± 0.02 VS 0.32± 0.08(P < 0.001). Plasma MPO was significantly increased in gpI compared to gp II(64, 48± 31.6ng/ml VS 50.4± 30.2ng/ml, p < 0.001). A significant positive correlation was found between plasma MPO and LV TDI-Tei index (in gpI (r = 0.877, p = 0.001).

**Conclusion:** Subclinical LV dysfunction evidenced by increased TDI Tei index was detected in DS children with anatomically and clinically normal hearts. This dysfunction correlated with oxidative stress assessed by plasma myeloperoxidase level which antioxidants augmentations and tissue Doppler myocardial performance screening and follow up for those children for early detection and protect management before reaching overt LV dysfunction.”