Gathering laboratory results to history and clinical examination would improve the sensitivity and specificity of ferritinemia for this highly dangerous disease and allow decisive therapeutic decisions.

Conclusion In addition to its "traditional" interest in deficits or iron overload, ferritin may offer some beneficial major diagnostic benefits for life threatening cases like perinatal hemochromatosis.

Methods We retrospectively analysed Tinzaparin doses and anti-Xa levels from all children admitted to PICU (January 2012–December 2013). Hospital policy is to determine the first aXa level after 3–4 doses and 4 h post dose, targeting 0.5–1.5 IU/ml. Results There were 31 episodes of newly started Tinzaparin in 28 children. Mean age was 57 (SD±62) months. First anti-Xa levels were determined at 3.45 (SD±1.9; range 1–10.8) days after the first dose and were sub therapeutic in 25 of 31 (81%): mean 0.33 (SD± 0.15) IU/ml. Tinzarin dose was increased in 12/25 (48%) patients and further anti-Xa levels were determined. In 15 patients further levels were not available due to transition to vit K antagonists or PICU discharge. Therapeutic anti-Xa levels (0.69 (SD± 0.27) IU/ml), were eventually reached in PICU in 16 patients after a mean of 8.8 (SD± 7.1 range 3–12) days.

Conclusion Tinzaparin dosing in PICU patients only leads to therapeutic anti-Xa levels in critically ill children after a mean of 8.8 (SD± 7.1 range 3–12) days after the first dose and were sub therapeutic in 25 of 31 (81%): mean 0.33 (SD± 0.15) IU/ml. Tinzarin dose was increased in 12/25 (48%) patients and further anti-Xa levels were determined. In 15 patients further levels were not available due to transition to vit K antagonists or PICU discharge. Therapeutic anti-Xa levels (0.69 (SD± 0.27) IU/ml), were eventually reached in PICU in 16 patients after a mean of 8.8 (SD± 7.1 range 3–10) days.

Objective Tinzaparin leads to adequate anti-Xa levels in paediatric intensive care patients following congenital heart surgery.

Methods Tinzaparin is used in paediatric intensive care (PICU) following cardiac surgery as a bridge to oral anticoagulation. Low Molecular Weight Heparins (LMWH), such as Tinzaparin are thought to lead to immediate anticoagulation with adequate anti-Xa levels 2–4 hours after the first dose. Dosing following international guidelines is in adequate age and guided by anti-Xa levels. However, little is known about LMWH dosing in PICU patients. We conducted a retrospective study to evaluate tinzaparin dosing.

Results There were 31 episodes of newly started Tinzaparin in 28 children. Mean age was 57 (SD±62) months. First anti-Xa levels were determined at 3.45 (SD±1.9; range 1–12) days after the first dose and were sub therapeutic in 25 of 31 (81%): mean 0.33 (SD± 0.15) IU/ml. Tinzarin dose was increased in 12/25 (48%) patients and further anti-Xa levels were determined. In 15 patients further levels were not available due to transition to vit K antagonists or PICU discharge. Therapeutic anti-Xa levels (0.69 (SD± 0.27) IU/ml), were eventually reached in PICU in 16 patients after a mean of 8.8 (SD± 7.1 range 3–10) days.

Conclusion Tinzaparin dosing in PICU patients only leads to target anti-Xa levels after more than 8 days. Levels need to be determined after the first dose so that doses can be adequately increased.

Objective To define the demographic characteristics, clinical features and outcome of patients with brain death and to emphasise the importance of organ donation in children.

Setting Centre I: 14-bed, tertiary care Paediatric Intensive Care Unit (PICU).

Methods Data were collected from September 2009 to October 2012 retrospectively. Twenty children who were diagnosed as brain death were included. Data including demographics, disease leading to brain death, duration of brain death evaluation, ancillary tests to confirm the brain death, complications and outcome, duration of hospitalisation, status of survival and organ donation were collected for statistical evaluation.

Results The mean age were 6.2 ± 5.3 (median:3.8) years. Male/ female ratio was 1.85. Disease leading to brain death was traumatic brain injury in 11(55%) patients. The mean duration of brain death evaluation was 6.7 ± 6.4 (median:4) and 1.7 ± 1 (median:1) days in Centres I and II respectively. The duration of hospitalisation was 12.5 ± 10.7 (median:7.5) days. Electroencephalography (EEG) was applied in 18(90%) patients. Complications included diabetes incipitus in 9(45%) cases. Duration of survival was 9.8 ± 9.4 (median:6) days. One of the patients’ parents give consent to organ donation in Centre I while 4 parents accepted organ donation in Centre II. The study demonstrated that duration of brain death evaluation in Centre I was longer when compared to Centre II (p < 0.05). There was no difference between centres in obtaining concepts of organ