

### **Discussion**

In this study the occurrence of HMD in term babies was high (11.5%) in comparison with other studies like a study in England<sup>(6)</sup> where the incidence is 0.1%.

Delivery by CS showed increased risk of HMD in both emergency and elective CS, but the risk is low in neonate delivered by NVD. These results are similar to many other studies which had demonstrated the protective nature of NVD in lowering the incidence of HMD. In Norwegian population –based cohort study they found cesarean delivery to be a major risk factor associated with the development of HMD<sup>(7)</sup>. A hospital based case control study in Beirut showed that after controlling for other factors HMD was twice as likely in infants delivered by cesarean delivery<sup>(8)</sup>. Another study done in Pakistan showed that HMD is three folds higher in CS group than those delivered vaginally<sup>(9)</sup>. Some authors have suggested that the mechanism for decreased HMD in vaginally delivered neonates is associated with endogenous prostaglandin production stimulated by uterine activity<sup>(10,11)</sup>. Some have suggested labor results in the release of lung surfactant into the airways, other theories to explain HMD in cesarean delivered infants include persistent fetal circulation as well as increased retention of pulmonary fluid in neonate delivered by CS.<sup>(12)</sup> During vaginal delivery about one third of fetal lung fluid is removed by squeezing the babies chest, this removal is missing during delivery by

CS<sup>(13)</sup>. Others have suggested that a beta-adrenergic surge during labor may be responsible for the ultimate fetal lung expulsion of surfactant in preparation for birth<sup>(14-16)</sup>.

In this study HMD was nearly the same in those delivered by emergency CS and in those delivered by elective CS, while in other studies like Curet et al, and Kim A found cesarean delivery before labor associated with higher incidence of HMD compared with cesarean done after the onset of labor pain<sup>(17-19)</sup>. This can be explained by the fact that the time of delivery that matters because that is what determines lung functional development – a view borne out by Gabert et al<sup>(20)</sup> who showed that CS was not associated with HMD when the lecithin –sphingomyelin ratio offered a good prognosis. In this study HMD was inversely proportional to body weight of neonate, similar results were obtained by many other studies like in Australian study showing that higher risk of HMD was associated with body weights lower than 2.5kg and the risk is lower in those Weighing > 3.5 kg<sup>(9)</sup>.

This was explained by the fact that HMD is thought to be caused by high lung alveolar surface tension, causing atelectasis and lack of pulmonary surfactant, which is a combination of lecithin, phosphatidylglycerol, cholesterol and surfactant apoproteins. The production of surfactant by fetal lung begins by week 20 but does not reach the surface of the fetal lung until much later<sup>(15)</sup>. In this study males are