

None of our patients in either group had received multiple doses of surfactant. Detailed analysis of patients receiving surfactant was not a stated objective; however, the major baseline characteristics were similar in both the groups. It was aimed to keep the tidal volumes lower by using lower PIP and optimal PEEP to maximally recruit lungs in synchronized intermittent mandatory ventilation (SIMV) group; however, tidal volume was not measured.

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Early term – Continuing Conundrum of Immaturity

In the past decade, the focus in newborn care has been on the concept of “late preterm”. The short term and long term complications of preterm birth between 34^{0/7} and 36^{6/7} weekly have become well recognized. Most doctors and patients assume that once “term” gestation is reached; the outcome is uniformly optimal [1]. However recent reports have highlighted the differential outcomes even in term infants born before 39 weeks and after. Respiratory distress, need for ventilation, hypoglycemia, sepsis, and NICU admissions, are higher in the infants born between 37^{0/7} and 38^{6/7} as compared to after 39 weeks [2-3]. Tita, *et al.* [4] determined the risk of adverse outcomes such as mechanical ventilation, newborn sepsis, hypoglycemia, admission to the neonatal ICU, and hospitalization for 5 days or more to be increased by a factor of 1.8 to 4.2 for births at 37 weeks and 1.3 to 2.1 for births at 38 weeks compared to 39 weeks. Long term neurodevelopmental outcomes and need for special education has also been found to be higher [5]. We reviewed our 2011 annual data (**Table I**) and compared the outcomes in 3 groups: late preterm (34^{0/7} to 36^{6/7} weeks), 37 weeks and 39 weeks. A delay of 2 weeks after 37 weeks added 300 g to the weight and significantly reduced NICU admissions, hypoglycemia and jaundice requiring phototherapy.

This heterogeneity in outcomes in the group of “term” births has generated 2 categories: “early term” births (37^{0/7} and 38^{6/7} weeks) and “full term” births (39^{0/7} to 41^{6/7} weeks). This new definition of “early term” emphasizes the continuing immaturity and potential for adverse outcomes. Though spontaneous onset of labor before “full term” or indicated delivery due to maternal-fetal complications are inevitable, the growing menace of

TABLE I NEONATAL OUTCOMES BY GESTATIONAL AGE

	34-36wk (n=287)	37wk (n=329)	39wk (n=633)
Mean birthweight (g) [§]	2279	2769	3024
LSCS (%)	48.8	33.4	26.7
NICU admission (n)%*	46.3 (133)	18.2 (60)	12.9 (82)
Hypoglycemia (n)% [#]	11.5 (33)	4.5 (15)	1.7 (11)
Need for phototherapy (n)%*	44.6 (128)	25.8 (85)	19.7 (125)
Respiratory distress (n)%*	13.4(38)	3.6 (12)	3.6 (23)

* Comparison between morbidities at gestational age 37 weeks and 39 weeks; *P<0.05; #P<0.05; §P<0.005.

delivering high risk mothers early – as soon as term gestation is reached – needs to be limited. As recommended, induction or scheduled cesarean section without any obstetric reason should be after 39 weeks. This has even become accepted as a quality indicator of obstetric care.

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Hypocalcemia and Tachycardia Induced Cardiomyopathy

Kourti, *et al.* [1] have highlighted important aspects of tachycardia induced cardiomyopathy and cardiogenic shock in their letter. We wish to share our experience in handling a child with tachycardia induced cardiomyopathy and cardiogenic shock resulting from hypocalcemia.

We describe a 4-month-old infant, who presented to pediatric emergency in cardiogenic shock and supraventricular tachycardia. He presented with cold and mottled peripheries, cyanosed and in hypotensive shock. He was given a saline bolus at 10 mL/kg over 30 minutes and started on IV adrenaline. ECG revealed SVT which was reverted to normal rhythm with the use of second dose of IV adenosine at 0.2 mg/kg. Transthoracic echocardiography revealed a dilated left ventricle with hypokinesia with markedly reduced left ventricular ejection fraction of 22%, without any congenital cardiac defects. Troponin T was negative by card test and serum CPK-MB levels were also normal. His ionized calcium was low (0.2 mmol/L). Serum magnesium levels were normal. Child was started on Inotropes (Milrinone and dobutamine) and calcium chloride was given to correct his hypocalcemia. Adrenaline was tapered off followed by dobutamine and milrinone. No antiarrhythmics were

given as maintenance therapy. His calcium levels improved to 1.1 mmol/L and gradually his LVEF improved to 60%. At discharge, child was asymptomatic and hemodynamically stable.

In managing these patients, electrolytes like calcium and magnesium are important part of work-up, apart from those described by the authors. Both hypocalcemia and hypomagnesemia have been reported as causes of arrhythmias in children as well adults [2,3]. Since this child had two such episodes, he also needs to undergo electrophysiological study to look for any conduction pathway defect, which is rare but a important cause to look for in patients with repeated arrhythmias.

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Specific Learning Disability – The Road to Disability Act

We would like to thank Dr Unni [1] for highlighting his concerns regarding specific learning disability (SLD) and bringing such an important yet not so common issue into limelight. The greatest hurdle lies in identifying these children so that only the deserving child gets the benefit if SLD forms part of Disability Act. The developed countries were early to recognize and quick to act on such issues. India is a vast multilingual country. It is not possible to apply any Western tool directly to our children

due to different social and educational structure and the norms will vary. The SLD battery test developed and validated by National Institute of Mental Health and Neurosciences (NIMHANS) [2], does not have proper norms. Moreover the tool does not cover the cognitive aspect. We need a foolproof tool to find the prevalence of SLD in public and private schools. First generation learners should be excluded or else the prevalence will be high as environment plays an important factor in the development of the child.

For disability benefit or relaxation in education board norms, who will certify children with SLD? This