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ASSESSMENT OF MYOCARDIAL FUNCTIONS IN NEONATES WITH SEVERE BIRTH ASPHYXIA BY 2D ECHOCARDIOGRAPHY

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ABSTRACT

Background: Birth asphyxia causes multiorgan dysfunction. Cardiovascular effects of birthasphyxia are transient myocardial ischemia, transient tricuspid insufficiency, mitralincompetence, persistent pulmonary hypertension, dilated cardiomyopathy, congestive cardiacfailure and cardiac dysrhythmia. This study aims to assess myocardial functions in neonateswith severe birth asphyxia at a tertiary care centre of southern Rajasthan.

Methods: A hospital-based cross-sectional analytical study was conducted with 90 neonatesfulfilling the criteria of birth asphyxia were admitted in the Neonatal Intensive Care Unit(NICU), of Department of Paediatrics RNT Medical College, Udaipur, Rajasthan. Each neonatewas systematically evaluated according to a pre-designed proforma. Details regarding mothersand neonates were noted, including vital parameters and anthropometric measurements. Athorough systemic examination and relevant laboratory investigations were conducted. 2DECHO was used for assessment of cardiac function. The data were analysed using appropriatestatistical tools and compared with relevant studies.

Results: Abnormal echocardiographic findings were noted in 59 (65.5%) neonates. The mostcommon echocardiographic finding in neonate with hypoxic ischemic encephalopathy (HIE)was Tricuspid regurgitation (TR) in 33.3% neonates (30 out of 90) followed by reduced ejectionfraction in 26.6% neonates (24 out of 90) and right ventricular (RV) hypokinesia (18.9%). Aninverse relationship was observed between severity of HIE and pulmonary hypertension.A highmortality of 23.3% was observed in our study participants.

Conclusion: Echocardiographic findings demonstrated worsening cardiac function with increased HIE severity. 2 D Echo can serve as a critical indicator for assessing and managingcardiac dysfunction in neonates with birth asphyxia.

Keywords: *Birth asphyxia, Hypoxic ischemic encephalopathy, Echocardiography*.

INTRODUCTION

According to the WORLD HEALTH ORGANISATION (WHO), perinatal asphyxia is thefailure to initiate and sustain breathing (1). Peri natal asphyxia is the third major cause ofneonatal mortality in India (2). According to the WHO, around 4 million babies develop birthasphyxia, and asphyxiated newborns may develop severe consequences such as epilepsy,cerebral palsy, developmental delay, and mental retardation. Furthermore, of 1.2 millionneonatal deaths in India, 300,000-350,000 babies die due to perinatal asphyxia mostly withinthe first 3 days of life (3)

American Academy of Paediatrics (AAP) and American College of Obstetrics andGynaecology define birth asphyxia as the presence of all of the following criteria

a) Profound metabolic or mixed acidaemia (pH <7.00) in umbilical artery blood sample, if obtained
b) Persistence of Apgar score of 0–3 for longer than 5 min
c) Neonatal neurologic sequelae (e.g., seizures, coma, hypotonia), and multiple organ involvement (e.g., kidney, lungs, liver, heart, intestines (4)

Birth asphyxia causes multiorgan dysfunction. The brain is commonly affected by Perinatalasphyxia because of hypoxic-ischemic encephalopathy but other organs or systems arefrequently overlooked which also bear the consequences of hypoxic-ischemic insult. The othercommonly affected organ systems in birth asphyxia involve kidneys in about 50% of neonates,cardiovascular system in 25%, pulmonary system in 23% (5)

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Cardiovascular effects of birth asphyxia are transient myocardial ischemia, transient tricuspidinsufficiency, mitral incompetence, persistent pulmonary hypertension, dilatedcardiomyopathy, congestive cardiac failure and cardiac dysrhythmia. Various mechanisms areresponsible for ischemic myocardia damage. Diminished oxidative phosphorylation and ATPproduction impairs ion pump function resulting in the accumulation of intracellular sodium,chloride, water and calcium; extracellular potassium, and excitatory neurotransmitters (eg.glutamate). This in turn causes cellular dysfunction and immediate or delayed cell death.Reperfusion injury to previously ischemic tissue causes further damage by formation of excessreactive oxygen species (6)

Oxidative effect of re-oxygenation done during resuscitation or ventilation may cause severecardiovascular consequence, which is due to degradation of cardiac myosin light chain protein 1 (MLC 1), matrix metalloproteinases 2 (MMP 2). A cardiac myocyte death is caused bydecrease in adenosine triphosphate (ATP) which causes cells to swell and die, releasingglutamate, which can lead to further cell death (7)

Myocardial injury in perinatal asphyxiated neonates with HIE can be appropriately diagnosedthrough significant clinical findings. Several early markers have been used to assess myocardialinjury in asphyxiated newborns including cardiac troponin I (cTnI) and CK MB. The imagingtechniques like 2D echocardiography, computed tomography, and tissue doppler imaging canalso be used (8)

Despite myocardial dysfunction being a common consequence of severe birth asphyxia,assessment of myocardial functions is often not done routinely in NICUs. Hence this study wasplanned to evaluate the impact of birth asphyxia on myocardial functions. Early identificationmight enable prompt and better management of asphyxiated newborns.

MATERIALS AND METHODS

A hospital-based cross sectional analytical study was conducted on 90 neonates with birthasphyxia at the Neonatal Intensive Care Unit of a tertiary care centre at southern Rajasthanover a period of one year after approval by institutional ethics committee. The study's samplesize was calculated based on a previous study by Goel M et al. (2013), (9) who found a 33.6%prevalence of myocardial changes by ECHO among asphyxiated neonates.Term neonates fulfilling the AAP criteria (4) for birth asphyxia were included in the study afterobtaining written informed consent from their care-givers.Preterm babies, low birth weight, intrauterine growth retarded neonates, those with majorcongenital malformations, suspected congenital cardiac diseases, any other diseases leading toshock, neonates getting admitted after 24 hours of birth, and those did not give show signs ofHIE were excluded.

A detailed history was taken including sociodemographic profiles, maternal details, antenatalcourse, obstetric and medical complications were noted in predesigned proforma. Babies' birthhistory, need and type of resuscitation required were noted and detailed examination wasperformed at the time of admission to the NICU. Appropriate management was begun as perunit protocol. Neonates were categorised into three groups based on the Sarnat&Sarnat HIEstaging (10). Comparative assessment of echo findings was done between the three groups.Babies were followed till discharge or death of babies.

Echocardiography was performed within one week of birth using a Philips Affinity-70 Echomachine® with a paediatric S8-2 transducer® by person trained in paediatricechocardiography.Data were entered in MS Excel and analysed using SPSS V.26. The Chi-Square Test wasapplied for proportional comparisons. p value <0.05 was considered significant.

RESULTS

In this study, 90 neonates with birth asphyxia were included. Of these 58(64.4%) were maleand 32 (35.6%) were females. Sixty percent of the newborns weighted between 2.5-3 kg,28.9% weighed 3-3.5 kg and 11.1% weighed more than 3.5 kg. The mean weight of the studyneonates was 2.798 kg. Seventy eightneonates were term and 12 % were postterm. (Table 1)

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Table 1: Distribution of study participants according to perinatal & neonatal history

Variable		N (%)
Sexof baby	Male Female	58 (64.4) 32 (35.6)
Birthweight(inKg)	2.5-3 3-3.5 >3.5	54 (60) 26 (28.9) 10 (11.1)
Meanbirthweight(inKg)±SD	2.798 ± 0.2665	
Gestational Age	Term Post-term	78 (86.7) 12 (13.3)
Typeofdelivery	LSCS	42 (46.7)
Perinatalevents	Meconium-StainedLiquor	12 (13.3)

The most common echocardiographic abnormality observed among the study participants wasTricuspid regurgitation which was seen in 30 (33.3%) neonates. Other common abnormalitiesobserved were reduced ejection fraction (26.6%), RV hypokinesia (18.9%), pulmonaryhypertension (17.7%), LV hypokinesia (11.1%), RA/RV dilatation (8.9%) and mitralregurgitation with global hypokinesia (4.4%).

Twenty-four neonates (26.6%) had reduced ejection fraction. Of these, 7 (7.8%) had mildlyreduced ejection fraction whereas 12 (13.3%) and 5(5.5%) neonates had moderately andseverely reduced ejection fraction respectively. (Table 2)

Table 2: Distribution of echocardiographic findings

Variable	N(%)
Tricuspid regurgitation Mild Moderate Severe	30(33.3) 3(10) 10(33.3) 17(56.6)
Ejection Fraction Slightly reduced (41-55) Moderately reduced (31-40) Markedly reduced (≤ 30)	24(26.6) 7 (7.8) 12 (13.3) 5 (5.5)
Right ventricular hypokinesia	17 (18.9)
Pulmonary Hypertension Mild Moderate Severe	16(17.7) 5 (5.6) 6 (6.7) 5 (5.6)
Left ventricular hypokinesia	10 (11.1)
Right atrial/right ventricular dilatation	8 (8.9)
Mitral regurgitation with global hypokinesia	4 (4.4)

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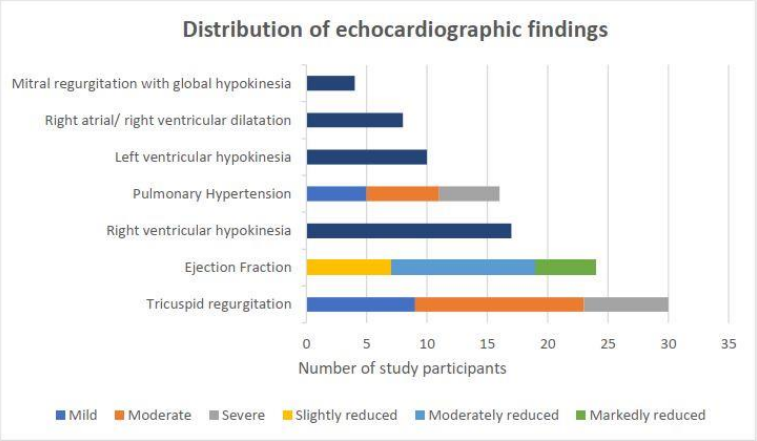

Diagram 1: Distribution of echocardiographic findings

Echocardiographic findings of the neonates were compared with their HIE severity. Of 30neonates with TR, 56.7 % were from HIE III group whereas 33.3% from HIE II and 10% fromHIE I. Most of the neonates (85.7%) with severe TR belonged to HIE III group. None of theneonates from HIE I group showed severe TR. The inverse relationship of HIE severity withTR was statistically significant **(p 0.002).**

Of 30 neonates with reduced ejection fraction, 46.6 % were from HIE III group whereas 23.3%from HIE II and 10% from HIE I. Most of the neonates with markedly reduced ejection fraction(< 30%) belonged to HIE III group. None of the neonates from HIE I group showed markedlyreduced ejection fraction. The inverse relationship of HIE severity with ejection fraction wasstatistically significant **(p 0.002).**

An increase in the incidence of RV hypokinesia was observed with increasing severity of HIEas 64.7% neonates with RV hypokinesia belonged to HIE III group. None of the neonates withHIE I showed RV hypokinesia 2D echo. It was found to be statistically significant **(p value0.00**1)

Similar inverse relationship of HIE severity were also observed with LV hypokinesia (p 0.06)but the relationship was not statistically significant. (Table 3)

Table 3 Relation between severity of HIE and cardiac dysfunction

Variable	Sarnat & Sarnat Staging			p-value
	I N=30(%)	II N=30(%)	III N=30(%)	
Tricuspid regurgitation Mild Moderate Severe	3(10) 3 (10) 0 (0) 0 (0)	10(33.3) 2 (6.7) 7 (23.3) 1 (3.3)	17(56.6) 4 (13.3) 7 (23.3) 6 (20)	0.002
Ejection Fraction Slightly reduced (41-55) Moderately reduced (31-40) Markedly reduced (≤30)	3(10) 3 (10) 0 (0) 0 (0)	7(23.3) 1 (6.7) 6 (23.3) 0 (3.3)	14(46.6) 3 (13.3) 6 (23.3) 5 (20)	0.002
Right ventricular hypokinesia	0 (0)	6 (20)	11(36.7)	0.001
Pulmonary Hypertension Mild Moderate Severe	 3 (10) 0 (0) 0 (0)	 1 (3.3) 3 (10) 1 (3.3)	 1 (3.3) 3 (10) 4 (13.3)	0.112
Left ventricular hypokinesia	0 (0)	5 (16.7)	5 (16.7)	0.06
Rightatrial/rightventricular dilatation	0 (0)	2 (6.7)	6 (20)	0.021
Mitral regurgitation with global hypokinesia	0 (0)	1 (3.3)	3 (10)	0.16

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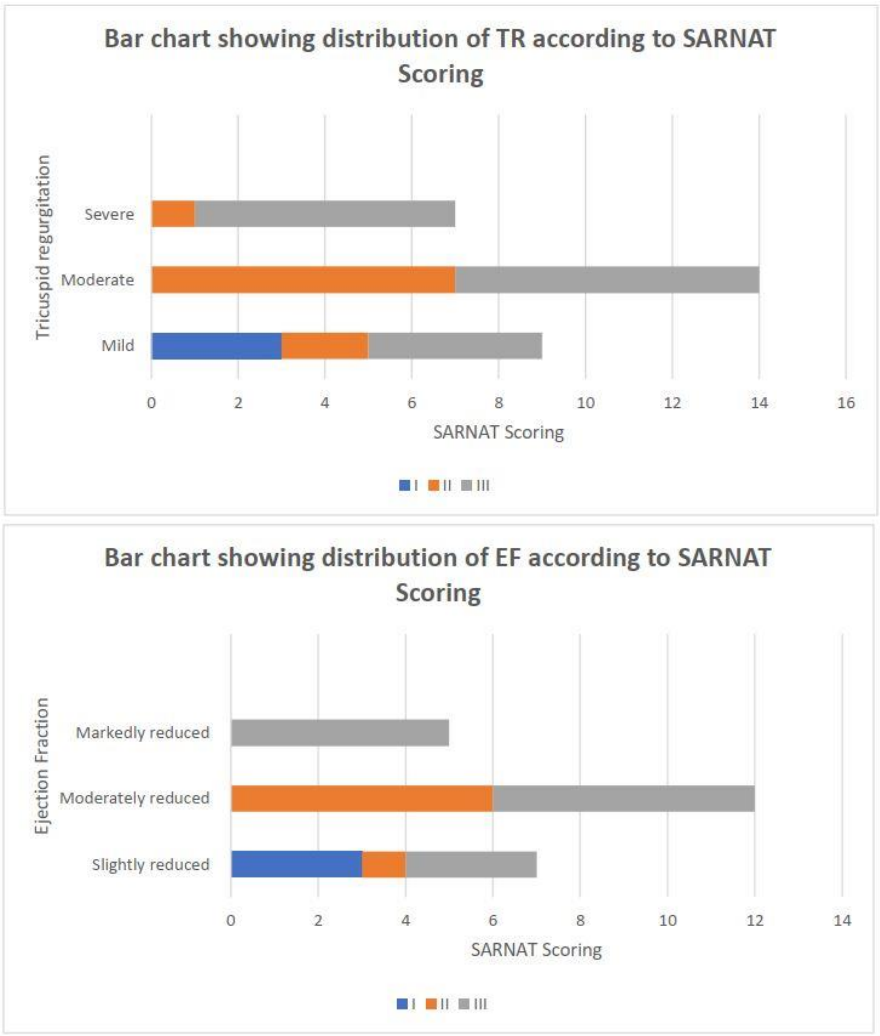

Figure 2: Variation in Cardiac Dysfunction as Compared to Sarnat Staging(10)

Table 4: OUTCOME OF STUDY PARTICIPANTS

Outcome	With cardiacinvolvement	Withoutcardiacinvolvement	Total	p Value
Discharged	40	29	69	0.007
Expired	19	2	21	

Table 4 describes the outcome of neonates with birth asphyxia. Of ninety neonates included in the study, 21(23.3%) expired. Most of the neonates (90.4%) who expired were found to haveechocardiographic abnormalities. Out of 59 neonates with cardiac involvement, 19 failed tosurvive (case fatality rate: 32.2%). Mortality observed in neonates without cardiac involvementwas only 6.4%. The impact of cardiac dysfunction on the overall survival of babies wasstatistically significant ($p < 0.05$).

DISCUSSION

This hospital-based cross-sectional analytical study was conducted with 90 neonates at theNeonatal Intensive Care Unit of a tertiary care center. The babies were stratified based onthe severity of HIE into three stages using Sarnat&Sarnat staging (10). The results andinterpretation of findings were based on severity of HIE stagings and extent of myocardialdysfunction.

Table 1 describes the different variables of study participants showing 64.4 % were male, 60%weighing between 2.5-3 kg with mean birth weight 2.798 ± 0.2665 kg and most babies (86.7%)born term. Shadique AM et al (11) also found out in their study of 70 cases that 57.1% weremale, 41.4% weighing between 2.5-3 kg.

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Birth history of the babies in the current studyrevealed that 46.7% of the babies delivered by LSCS and 13.3% had a history of meconium-stained liquor. Shadique AM et al(11) also found in their study that 34.3% of babies weredelivered by LSCS and 27.1% had a history of meconium-stained liquor. Their findings wereconsistent with our present study. Higher than usual rate of LSCS could be explained by thefact that these babies were most probably suffering from foetal distress from antenatal periodwhich was an indication for LSCS delivery. In utero foetal compromise can result in earlypassage of meconium which explains higher than usual proportion of meconium-stained liquorbabies.

Out of 90 babies enrolled in this study, 59 (65.5%) were found to have abnormalechocardiography findings. Shadique AM et al (11) found out in their study that 65.7% hadabnormal echo findings. Rajakumar PS et al (12) also reported cardiac involvement in 73.3%of their study participants. Ischemic myocardial injury caused secondary to birth asphyxiacould be a reason for abnormal echo findings.

Table 2 shows echocardiography findings of neonates with birth asphyxia. In our study themost common echocardiographic abnormality observed was tricuspid regurgitation (TR) seenin 30 (33.3%) neonates followed by reduced ejection fraction in 24(26.6%) and RVhypokinesia in 17(18.9%). Other findings were pulmonary artery hypertension (17.7%), LVhypokinesia (11.1%), RA/RV dilatation (8.9%) and MR with global hypokinesia (4.4%).Similar findings were observed by Rajakumar PS et al (12) who reported TR was the mostcommon finding seen in 23.3% neonates followed by RV hypokinesia (20%), LV hypokinesia(13.3%). Papillary muscle ischemia or persistent pulmonary hypertension could be theunderlying cause for TR in perinatal asphyxia. Sub endocardial region being closest to highpressure of ventricular cavity and most distant from coronary blood supply is most severelyaffected in asphyxiated neonates. Elevated pulmonary pressure in neonatal period along withlarger RV muscle mass make the RV more susceptible to ischemic damage as compared to LV.Reasons for pulmonary artery hypertension in these babies could be persistent hypoxia resultin persistence of constricted pulmonary vascular bed with consequent right to left shunt.

Table 3 describes comparative assessment of HIE severity with degree of cardiac dysfunction.The study participants were divided into three groups according to HIE severity andechocardiographic findings of different groups were compared. Of 30 neonates with TR, 56.7% were from HIE III group whereas 33.3% from HIE II and 10% from HIE I. Most of theneonates (85.7%) with severe TR belonged to HIE III group. None of the neonates from HIE Igroup showed severe TR. The inverse relationship of HIE severity with TR was statisticallysignificant (p 0.002). The more severe the birth asphyxia, the worse chances of ischemic injuryto papillary muscles and persistent pulmonary hypertension (PPHN) which could lead tovalvular regurgitation explaining more severe grades of TR. Jain D et al (13) have also documentedan increasing trend in TR, MR, RV dilatation and LV dilatation with increasing HIE severitywere also reported by them.

Out of 90 babies, 24 (26.7%) were found to have reduced ejection fraction. Seventeen (70.8%)belong to HIE III group whereas only 3(12.5%) babies were from HIE I group. Also 85.7% ofthe babies with markedly reduced ejection fraction (<30%) were from HIE 3 subgroup. Astatistically significant decline in ejection fraction was noted with increasing severity of HIE.Jain D et al (13) have also documented of significant decline in ejection fraction with increasingHIE severity. Goel M et al (9) also found that ejection fraction was lower in asphyxiatedneonates, with values of 64.5%, 70.5% and 81.5% for ejection fraction in their study groupscontrol, moderate and severe asphyxiated newborns. Bradycardia and acidosis associated withbirth asphyxia might eventually lead to myocardial dysfunction, hence resulting in a declineejection fraction.

In the current study, 23.3% of the neonates expired. Most of these babies (90.4%) hadechocardiographic abnormalities. Simultaneously, case fatality was much higher in babies withcardiac involvement as compared to those without cardiac involvement. Rajakumar PS et al(12) have also reported 30% mortality in their study of neonates with birth asphyxia and all theseneonates had abnormal echocardiographic changes. Their findings were similar to our study.Higher mortality in babies with abnormal echocardiographic findings could be explained bythe fact that cardiovascular instabilities like arrhythmia, hypotension and myocardial ischemiamight be more common in these babies. These changes might be adversely affecting theoutcome of the neonates.

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CONCLUSION

Myocardial dysfunction secondary to birth asphyxia is often underestimated, though it is quitecommon. It can also be concluded that the increasing severity of HIE is related to more severecardiac involvement. Hence a comprehensive routine echocardiographic assessment inneonates with birth asphyxia shall be included as a part of routine care. This will lead to earlydetection of ischemic cardiac damage hence enabling timely management of these patients.

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Ethical clearance- Approved

REFERENCES

1. World Health Organization. New estimates of perinatal mortality. Weekly Epidemiological Record Releve Epidemiologique Hebdomadaire. 1996;71(40):297- 303.
2. Neonatal Morbidity and Mortality. Report of the National Neonatal Perinatal Database. Report; 2002-2003. p. 1-58.
3. World Health Organization. Neonatal and Perinatal Mortality 2004; Country, Regional and Global Estimates. Geneva: WHO; 2006.
4. Committee on Fetus and Newborn, American Academy of Pediatrics. Use and abuse of the Apgar score. Pediatrics. 1996;98: 141-2.
5. Tanzil WL, Wilar R, Mantik MF, Umboh A, Tatura SN Comparison of urine neutrophil gelatinase-associated lipocalin to serum creatinine to assess kidney function in neonatal asphyxia. PaediatrIndones. 2016; 56:356-359. doi: 10.14238/pi56.4.2016.356-9
6. Eric CE, Anne R, Camilia R, Ann R. Cloherty and Stark's manual of neonatal care 9th Ed. Wolters Kluwer 2022. Chapter 55; 825-833
7. Doroszko, A., Polewicz, D., Sawicka, J. *et al.* Cardiac dysfunction in an animal model of neonatal asphyxia is associated with increased degradation of MLC1 by MMP-2. *Basic Res Cardiol*104, 669–679 (2009). https://doi.org/10.1007/s00395-009-0035-1
8. Simovic AM, Prijic SM, Knezevic JB, Igrutinovic ZR, Vujic AJ, Kosutic JL. Predictive value of biochemical, echocardiographic and electrocardiographic markers in nonsurviving and surviving asphyxiated full-term newborns. Turkish Journal of Pediatrics. 2014;56(3).
9. Goel M, Poorva G, Yadav B. Assessment of myocardial function in birth asphyxia. Int J Med Res Rev. 2013;1(5):228-32.
10. Sarnat HB, Sarnat MS. Neonatal encephalopathy followingfetal distress. A clinical and electroencephalographic study. Arch Neurol. 1976;33(10):696–705
11. Shadique AM, Sailavasan M. A prospective study on cardiac changes (electrocardiographic, enzymatic and echocardiographic) in birth asphyxiated neonates admitted in the tertiary care centre. Int J ContempPediatr. 2019;6(2):269-74
12. Rajakumar PS, Vishnu Bhat B, Sridhar MG, Balachander J, Konar BC, Narayanan P et al. Electrocardiographic and echocardiographic changes in perinatal asphyxia. Indian J. Pediatr. 2009; 76:261-4.
13. Jain D, Pandey AK, Das BK, Prasad R. Cardiac function in perinatal asphyxia. Sch J Appl Med Sci. 2016;4(7F):2718-28