Clinical Application of Neonatal Instantaneous Heart Rate Monitoring (Part One: Neonatal Instantaneous Heart Rate Response to Apnea)

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Introduction: Fetal monitoring of instantaneous heart rate (IHR) is one of major advances in recent perinatal medicine. It is widely accepted that changing pattern of fetal IHR associated with uterine contraction will reflect fetal well or ill being. Uterine contraction produces temporary hypoxia to the fetus and apnea does the same to the neonate. Therefore we analysed changing patterns of neonatal IHR associated with apnea to investigate if there are similar clinical meanings as does the fetal monitoring.

Method: Subjects for the study were 22 frequently apnic neonates who were admitted to Kitasato University Hospital during 1977-8. They are subdivided into three groups. Group A is consisted with 10 sleepy babies due to anesthetic delivery by diazepam and pentazocine, group B with otherwise normal preterm infants and group C with 6 seriously ill neonates. IHR and respiration were recorded on strip chart during their apnic episodes. IHR was automatically calculated from R-R interval and respiratory movements were recorded by impedance method. Neonatal IHR response to apnea was classified as follow; Type I deceleration (reflex bradycardia) is dropping of IHR synchronized with apnic episodes, Type II deceleration (hypoxic bradycardia) is gradual decrease of IHR during apnic episodes, Type III deceleration (primary bradycardia) is decrease of IHR with no consistent relation with apnic episodes, Non-reactive type means no appreciable change of IHR during apnic episodes, and Acceleration is sudden increase of IHR associated with apnic episodes.

Result: Neonatal IHR response to apnea of each group were analysed whether above mentioned patterns were seen or not (Table 1). None of group A infants had type II or III deceleration except one who had secondary apnea requiring IPPB. All of them became apnea free since the second day of life and remained well thereafter. Among group B, only one extremely preterm infants (26 weeks, 707 gm) had Type II and III deceleration and he also did not show acceleration pattern. In group C, all of them had type II and/or type III deceleration and half of them did not have acceleration pattern. Though 26 weeks preterm baby in group B survived without much trouble except apnea, that degree of prematurity could not be regarded as normal. Therefore if he was subtracted from group B and added to group C, the difference of group A-B and group C is more contrasted. Namely the former groups rarely have type II and III decelerations and almost always has acceleration. On the contrary, group C have type II and III deceleration very frequently and often did not have acceleration.

Comment: Considering apnea might have similar pathophysiological effect to the neonate as does uterine contraction to the

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fetus, we classified neonatal IHR response to apnea. Type I deceleration is sharp and sudden decrease of IHR, suggesting reflexic response to apnea. This is seen most frequently in both well and ill neonates. Type II deceleration is somewhat similar to late deceleration of the fetus, suggesting hypoxic response of heart rate. This is seen mostly during prolonged apnea, mixed with reflexic component. Type III deceleration is probably primary vagal reflex because it was avolished by atropine in three such cases. Latter two types of heart rate change were seldom seen in normal well neonates but frequently seen in sick neonates. Non-reactive type could be the expression of loss of variability or inadequate autonomic stimuli by apnea. Therefore it is seen in very ill neonates and also in relatively well neonates with mild degree of apnea. Acceleration represents good autonomic response and is seen most frequently in well neonates. Its absence will indicate compromised reflex system of the neonate. Though autonomic nurve system of the fetus and of the neonate seems to be well established, neonates may not react the same way as does the fetus and our interpretations of neonatal IHR response to apnea are grossly speculation. But our clinical analysis of cardiorespirogram of apnic infants was so far very encouraging.

Conclusion: Neonatal IHR response to apnea was devided in 5 patterns. Type II and III decelerations were thought to be ominous signs and presence of acceleration represented well-being of the neonate.

Table 1.

	G.A. (w)	B.W. (g)	Type I	Type II	Type III	Non-reac.	Accel.
Group A	37-38	2405-3571	9/10	1/10	0/10	4/10	10/10
Group B	26-32	707-1890	6/6	1/6	1/6	3/6	5/6
Group C	27-38	1310-2603	6/6	3/6	5/6	5/6	3/6

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