Title : **Improved Automated Noninvasive Blood Pressure Monitoring in Infants**

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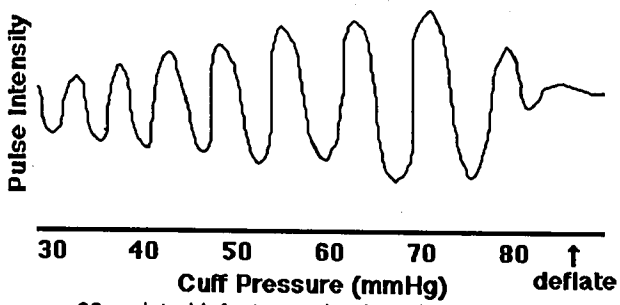
Conventional measurement of blood pressure (BP) using a mercury sphygmomanometer is not widely useful in infants because of the lack of Korotkoff sounds. Currently used instruments that measure blood pressure in infants have evolved rapidly using Doppler, Infrasonde, or oscillometric techniques. Currently the most widely used device is the oscillometer, a technique which incorporates a measurement of pulse intensity. As the pressure in a suprasystolic cuff is deflated, the signal intensity changes. Maximal amplitude of the arterial pulsation is coincident with mean arterial BP.

Infant movements and wide swings in BP inhibit the ability of these devices to yield consistent BP readings. More importantly, when blood pressure decreases rapidly, such as during induction of anesthesia, the machine response time is significantly prolonged. A new software modification incorporating the measurement of oscillations during inflation (Criticare Systems Inc #506) rather than the currently used suprasystemic BP inflation followed by a slow deflation (Criticon Inc. #1846) was compared. Inflations from 30 mmHg slowly increasing to just above systolic occlusion pressure yields the waveform seen in the figure.




60 sedated infants ranging in weight from 5 - 15 kg were studied using a conventional infant cuff. In each case the cuff bladder length was at least 2/3 of the extremity circumference. Mean arterial BP within and between groups was not different from either device. Percentage of recorded BPs was also not different between the two instruments. However, a blinded observer found significant response time and actual response differences between the instruments. The inflation mode recorded blood pressure more often and within 15 seconds opposed to a mean of 30 seconds for the deflation mode. The deflation mode caused more children to cry. When the deflation mode did not upset the child, measurements of lower BP were significantly prolonged or were absent compared to the inflation mode.



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Title: **Association of Airway Obstruction, Hypoxemia, and Postoperative Apnea in Preterm Infants**

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Recent studies of premature infants have indicated that airway obstruction and hypoxemia play an important role in the pathogenesis of apnea of prematurity. Because of the effects of anesthetics on $0_2$ chemoreceptors and pharyngeal muscle tone, airway obstruction and hypoxemia could also be key elements in the pathophysiology of postoperative apnea in former premature infants. The aim of the present study, therefore, was to determine arterial hemoglobin-$0_2$ saturation ($Sp0_2$) and frequency of airway obstruction with postoperative apnea in former premature infants after general anesthesia

**Methods.** We studied 40 infants (<37 weeks gestation, <50 wks postconception), ASA physical status 1 or 2, who had inguinal hernia repairs under general endotracheal inhalational anesthesia. No narcotic or hypnotic drugs were given. Postoperative apnea, airway obstruction, and hypoxemia were dectected with an Eden-tec 2400 monitor, which integrates pneumocardiography with nasal airflow and pulse oximetry. Data was recorded on tape and played back onto 4 channel paper for detailed analysis. Recording began on arrival in the recovery room and ended 12 hours later. We defined apnea as follows: brief apnea is no breathing >6sec; prolonged apnea is no breathing for >15sec. Apnea & bradycardia is apnea with a drop in heart rate to <80bpm for >5sec. We also defined apnea according to whether airway obstruction occurred. Central apnea consisted of no chestwall motion or nasal airflow. Obstructive apnea consisted of chestwall motion but no nasal airflow

**Results.** Fifteen infants had postoperative apnea; their gestational and postconceptional ages and weight at operation were 29±1wks, 43±1 wks, and 4±0.4kg respectively. Among these infants, airway obstruction occurred in over a quarter of the apneic episodes. (table 1) Both obstructive and central apnea was observed in each infant. The frequency and severity of hypoxemia depended on the type of apnea (table 2): 4% of the brief apnea had a Sp02 <80%, whereas 22% and 31% of the prolonged apnea and apnea & bradycardia had $Sp0_2$ <80%, respectively. $Sp0_2$ remained >90% in about half of the prolonged apnea and apnea & bradycardia.

**Conclusion.** Although hypoxemia has been considered a cause of postoperative apnea, our results suggest that hypoxemia actually results from apnea. Brief apnea in the postoperative period previously has been regarded as insignificant. However, this study indicates that even brief apnea in the postoperative setting may not be inconsequential because of the profound desaturation that may follow. Finally, the ratio of central and obstruction apnea that we observed is similar to that of premature infants who have not undergone anesthesia, suggesting that postoperative apnea may be the result of anesthetics unmasking pathways for apnea that had become quiescent with maturation of the respiratory control system.

TABLE 1 Episodes of Airway Obstruction with Postoperative Apnea in Infants Following Inguinal Hernia Repairs

| | N | % |
|---|---|---|
| Central Apnea | 195 | 73 |
| Obstructive Apnea | 73 | 27 |
| Total | 268 | 100 |

Values are numbered of apneic episodes >6seconds duration.

TABLE 2 Pulse Oximetry Saturation Ranges with Posteroperative Apnea in Infants Following Inguinal Hernia Repairs

| Sp02 (%) | Brief Apnea N (%) | Prolonged Apnea N (%) | Apnea & Bardycardia N (%) |
|---|---|---|---|
| 100-90 | 460 (85) | 43 (64) | 7 (37) |
| 89-80 | 61 (11) | 9 (14) | 6 (32) |
| 79-70 | 13 (3) | 9 (14) | 5 (26) |
| <70 | 5 (1) | 5 (8) | 1 (5) |
| | 539 (100) | 66 (100) | 19 (100) |

Values are number of apneic episodes.