ANTERIOR FONTANELLE PRESSURE RESPONSES TO TRACHEAL INTUBATION IN THE AWAKE AND ANAESTHETIZED INFANT

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Rapid control of the airway is essential during the induction of anaesthesia in the small infant. Difficulty in ventilating by mask, and the discovery of unsuspected upper airway anomalies, may result in difficulty with tracheal intubation. As a result, some authorities [1,2] recommend that intubation be performed before the induction of anaesthesia.

Raju and colleagues [3] reported that intracranial pressure (ICP) increases during oro-tracheal intubation in the awake infant. Kelly and Finer [4] reported similar findings during nasotracheal intubation in the sick neonate. Hatch and Sumner [2] suggested that these increases in ICP in the premature neonate may result in intracranial haemorrhage. However, as yet, there has been no report of a study which compares ICP responses to tracheal intubation in the awake and anaesthetized infant.

The Ladd transducer is an accurate non-invasive monitor of neonatal anterior fontanelle pressure (AFP) [5] which correlates well with actual ICP [6]. This study was designed to compare the effects of tracheal intubation on AFP, measured with the Ladd transducer, in awake and anaesthetized infants.

SUMMARY

In order to define the changes in intracranial pressure which occur during tracheal intubation in young infants, a Ladd transducer was used to monitor anterior fontanelle pressure (AFP) non-invasively in awake (group 1, n = 14) and anaesthetized (group 2, n = 10) infants during intubation of the trachea. Heart rate and systolic arterial pressure were also recorded. In quiet, undisturbed infants, AFP (mean ± SEM) was similar in groups 1 (9.6 ± 0.5 mm Hg) and 2 (8.7 ± 0.8 mm Hg); with crying, AFP increased significantly in both groups. During laryngoscopy in group 1, AFP increased to 33.5 ± 3.6 mm Hg, which was significantly greater than in the quiet infant, but did not differ significantly from measurements in the crying infant. In group 2, AFP increased significantly to 15.8 ± 18 mm Hg during laryngoscopy. This increase was significantly less than the group 1 response. Neither heart rate nor systolic arterial pressure changed significantly in either group during laryngoscopy—when compared with measurements in the quiet state. It was concluded that AFP increases significantly during intubation and during crying in the infant. The response to intubation is only partially attenuated by the prior administration of general anaesthesia.

PATIENTS AND METHODS

Following institutional approval, 24 infants were studied. Informed verbal consent was obtained from the parents. All infants were less than 8 weeks post-natal age, ASA I or II, and were scheduled to undergo a variety of surgical procedures. Those with central nervous system defects or neurological disease were excluded.


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Infants were fasted for 4 h and did not receive preoperative medication. On arrival in the operating room all infants were placed supine with the head in a neutral position supported by a head ring. The hair over the anterior fontanelle was shaved and the scalp painted with tincture of benzoin. A calibrated Ladd transducer was placed over the anterior fontanelle and secured by adhesive foam to minimize the effects of application pressure [6]. A single-channel chart recording of AFP was obtained throughout the procedure. Arterial pressure (by cuff), Doppler flow and the electrocardiograph were monitored. All patients were preoxygenated for 2 min. Infants who were awake during intubation of the trachea (group 1) were given atropine 0.02 mg kg⁻¹ i.v. 2 min before intubation. In those patients anaesthetized before intubation (group 2), anaesthesia was induced with i.v. sodium thiopentone 5 mg kg⁻¹, atropine 0.02 mg kg⁻¹ and suxamethonium 2 mg kg⁻¹. After a brief period of manual ventilation with 100% oxygen, the trachea was intubated. In both groups, care was taken during intubation to ensure that no hyperextension or hyperflexion of the neck occurred. Extraneous pressure on the Ladd transducer was avoided. Anaesthesia was maintained with halothane or isoflurane supplemented with nitrous oxide in oxygen. Intermittent positive pressure ventilation was used in all infants with zero end-expiratory pressure. All infants were supine and horizontal throughout the study.

The AFP was recorded five times in each case; pre-induction in the quiet, undisturbed infant; pre-induction during crying; peak pressure during laryngoscopy; immediately after tracheal intubation; and during steady state general anaesthesia. Heart rate and arterial pressure were measured before induction, at intubation, and during steady state general anaesthesia. All measurements were made by the investigator who was not involved in anaesthetizing the infant.

The demographic data were compared using Student’s unpaired t test. Within groups, statistical significance was determined by comparing values with quiet measurements using two-way analysis of variance and the Student–Newman–Keuls multiple range test. Values at each of the five recordings in both groups were compared using Student’s unpaired t test. Statistical significance was accepted as \( P < 0.05 \). Data are presented as mean ± standard error of the mean.

### RESULTS

Demographic data for groups 1 and 2 are shown in table I. Group 1 patients weighed less than group 2 patients, but they did not differ significantly in post-conceptual age.

Indications for surgery were: inguinal herniotomy (\( n = 5 \)), pyloromyotomy (\( n = 5 \)), creation of colostomy (\( n = 3 \)), pyeloplasty (\( n = 2 \)),...
insertion of central venous line (n = 1), gastrostomy (n = 1), vesicostomy (n = 1), removal of corneal sutures (n = 1), excision of sacral teratoma (n = 1), dilatation of choanal atresia (n = 1), cystoscopy (n = 1), laparotomy (n = 1) and ligation of patent ductus arteriosus (n = 1).

In both groups AFP increased significantly during crying compared with the quiet state. A significant increase also occurred during laryngoscopy in both anaesthetized and awake patients. In group 1, the increase in AFP associated with laryngoscopy did not differ significantly from that observed during crying. In group 2, however, the increase was significantly less than that seen with crying and was also significantly less than in group 1 infants (fig. 1). Changes in heart rate (HR) and systolic arterial pressure (SAP) are shown in table II. There was no significant difference in SAP or HR before induction. Values obtained during laryngoscopy, and during general anaesthesia, were also not significantly different except in group 2, in which SAP decreased significantly during anaesthesia. This was not felt to be clinically significant.

**DISCUSSION**

Significant increases in AFP during awake intubation in infants were demonstrated. The increases were significantly attenuated in infants anaesthetized before intubation. Although group 2 infants were slightly older, this difference was not statistically significant. Group 2 infants who were intubated asleep were significantly larger. As larger infants are generally more vigorous, they would be expected to have greater increases in AFP than smaller, weaker infants during airway manipulation. We feel, therefore, that the difference in weight does not invalidate our conclusions.

In another published study of AFP responses during tracheal intubation and anaesthesia, Raju and colleagues [3] found a significantly greater increase in AFP in infants undergoing awake intubation compared with those paralysed before intubation. However, the infants in their report ranged in age from 7 days to 10 months and were not anaesthetized. Furthermore, two of the nine patients studied had hydrocephalus, which may have accounted for the high mean preoperative AFP (16.5 cm H₂O) and the significant increase in AFP to 27.1 cm H₂O during anaesthesia. Their report did not specify the method of fixation of the Ladd transducer. A previous investigation [6] reported variability in AFP measurement, depending on the method of application of the transducer. Subsequent work from Raju, Doshi and Vidyasager [7] has demonstrated mean ICP values of 7.1 mm Hg, 7.5 mm Hg and 8.2 mm Hg in infants of gestational ages 28–32 weeks, 33–37 weeks and 38–40 weeks, respectively. Using a method of fixation known to minimize the effects of application pressure [6], we have been able to demonstrate similar AFP values in quiet, undisturbed infants (fig. 1).

The pathogenesis of intraventricular intracranial haemorrhage suggests that increases in arterial and central venous pressures may be more important than increases in ICP [8]. In the present study, high systolic arterial pressures were observed in infants during intubation, with higher values during awake intubation than in those anaesthetized before intubation. However, these changes were not statistically significant—possibly because of the small numbers of infants studied. Kelly and Finer [4] observed increases in systolic arterial pressure of 24.7 mm Hg in infants pretreated with atropine compared with 17.8 mm Hg in those paralysed with pancuronium. In contrast, Hinkle [9] demonstrated decreases in systolic arterial pressure during 30-s periods of laryngoscopy in infants pretreated with atropine. Further studies are required to define these changes more clearly.
Awake intubation may result in periods of apnoea sufficient to cause hypercarbia or hypoxaemia, with subsequent increases in ICP [3]. However, Fisher, Frewen and Swedlow [10] were unable to demonstrate increases in ICP in children during periods of apnoea lasting 30 s. Similar periods of apnoea during laryngoscopy caused a decrease in mean transcutaneous oxygen tension (\(PtcO_2\)) to 44.2 mm Hg in neonates who had breathed 100% oxygen for 2 min [9]. Neither arterial oxygen saturation nor \(PtcO_2\) was measured in the present study. Although there was no clinical evidence of cyanosis in any of the patients, it remains speculative as to whether short periods of hypoxaemia contributed to the increase in AFP during awake intubation.

The attenuated AFP response to intubation in the anaesthetized infant may result in part from neuromuscular blockades. Raju and colleagues [3] reported attenuated responses to intubation in infants paralysed with tubocurarine. Kelly and Finer [4] found an increase in AFP of 19.8 cm H_2O in awake neonates compared with 11.6 cm H_2O in those paralysed with pancuronium. Although suxamethonium may cause increases in ICP, White and co-workers [11] reported that the increase in ICP during tracheal suctioning in adults was attenuated to a greater extent with suxamethonium 1 mg kg\(^{-1}\) than with fentanyl, thiopentone or i.v. or intratracheal lignocaine. We postulate that paralysis attenuates the increase in AFP by preventing the increase in intrathoracic pressure which is associated with coughing and straining.

Recent concern over the increase in AFP during awake intubation may not be justified. We have demonstrated significant increases in AFP during crying in healthy infants. The paroxysmal increase in AFP during awake intubation may well be of less importance than the prolonged increases observed during crying in the healthy infant.

Awake intubation remains an important part of paediatric anaesthetic practice and further studies should be directed at identifying and attenuating the cardiovascular and ICP responses to this manoeuvre.

REFERENCES