



## Original Article

# The effects of dummy/pacifier use on infant blood pressure and autonomic activity during sleep



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## ABSTRACT

**Background:** Dummy/pacifier use is protective for sudden infant death syndrome (SIDS); however, the mechanism/s for this are unknown. As impaired cardiovascular control may be the underlying cause of SIDS, we assessed the effects of dummy/pacifier use on cardiovascular control during sleep within the first 6 months of life.

**Methods:** Term infants, divided into dummy/pacifier users and non-dummy/pacifier users, were studied at 2–4 weeks ( $n = 27$ ), 2–3 months ( $n = 35$ ) and 5–6 months ( $n = 31$ ) using daytime polysomnography. Heart rate, blood pressure (BP), heart rate variability (HRV), blood pressure variability (BPV), and baroreflex sensitivity (BRS) were measured in triplicate 1–2-min epochs during quiet and active sleep in the supine and prone positions.

**Results:** Overall, during the non-sucking periods, in the prone position, the BP was higher (10–22 mmHg) in dummy/pacifier users compared to non-users at 2–4 weeks and 5–6 months ( $p < 0.05$  for both). HRV and BRS were higher in dummy/pacifier users compared to non-users at 2–4 weeks ( $p < 0.05$ ). Active sucking increased HRV and BPV, consistent with increased sympathetic activity in dummy/pacifier users.

**Conclusions:** Higher BP and HRV in dummy/pacifier users indicate increased sympathetic tone, which may serve as a protective mechanism against possible hypotension leading to SIDS; however, these effects were not apparent at 2–3 months, when the risk of SIDS is highest.

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## 1. Introduction

Despite the dramatic decline in the incidence of the Sudden Infant Death Syndrome (SIDS), >2500 infants die in the USA each year and SIDS remains the leading cause of perinatal death in Western countries [1]. Evidence from cardiorespiratory recordings prior to death suggests that SIDS occurs during sleep and may be due to a failure of autonomic cardiovascular control to compensate for a profound hypotension in conjunction with a failure to arouse from sleep [2]. In support of this hypothesis, prone sleeping, the major risk factor

for SIDS, is associated with an impaired control of both heart rate (HR) and blood pressure (BP), and this is most marked at 2–3 months of age when the risk of SIDS is greatest [3,4]. While there has been much effort to understand how major risk factors, such as prone sleeping, contribute to the fatal event of SIDS, there has been little research directed at factors known to reduce the risk of SIDS. In 1979, it was first suggested that dummy/pacifier use might decrease SIDS risk [5], and this suggestion was later supported by further studies [6–13]. A meta-analysis in 2005 reported a 50% reduction in SIDS among dummy/pacifier users in the last sleep compared to a control group [14] and subsequent studies have confirmed these findings [15,16]. Two more recently published case–control studies have also found dummies/pacifiers to be protective [17,18]. Only one small study has failed to identify a significant protective effect [19].

The American Academy of Pediatrics recommended the use of a dummy/pacifier in 2005 [20]. This recommendation has been quite controversial, and not all countries have adopted this approach. In part, the hesitation to recommend a dummy/pacifier has been due to the lack of understanding of the mechanisms through which they provide protection [21] and the concern that dummy/pacifier use may adversely impact breastfeeding [22].

**Abbreviations:** SIDS, Sudden Infant Death Syndrome; HRV, Heart rate variability; BPV, Blood pressure variability; BRS, Baroreflex sensitivity; QS, Quiet sleep; AS, Active sleep; ECG, Electrocardiogram; LF, Low frequency; HF, High frequency; LF/HF, Low-frequency/high-frequency ratio.

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To date, there has only been one study on the effects of dummy/pacifier use on cardiovascular control of HR, and this suggested that sucking on a dummy/pacifier was associated with increased sympathetic activation during sleep [23]. No studies have investigated the impact of dummy/pacifier use on BP or autonomic BP control. The most recent epidemiological study identified that dummy/pacifier use provided the greatest protection to infants in the prone sleeping position [18]; however, no physiological studies have examined dummy/pacifier use in this position to date.

We aimed to assess HR and BP and their control during prone and supine sleep in infants who regularly used a dummy/pacifier and those who did not, and to assess the effect of active sucking in dummy/pacifier users across the first 6 months after birth when most SIDS occurs. We hypothesized that BP and HR would be elevated in dummy/pacifier users and cardiovascular control improved in the prone sleeping position.

## 2. Methods

Ethical approval was granted by the Southern Health and Monash University Human Research Ethics Committees. No monetary incentive was provided for participation, and written parental consent was obtained.

### 2.1. Subjects

Thirty-seven healthy term infants (17 males and 20 females) born at 38–42 weeks of gestational age were studied. Infants were recruited prior to birth via advertisements in staff newsletters at the Monash University and the Monash Medical Centre, or were recruited after birth in person from both private (Jesse Macpherson Private Hospital) and public wards at the Monash Medical Centre. Birth weights ranged from 2900 to 4615 g (mean  $3623 \pm 60$  g, mean  $\pm$  standard error of mean (SEM)), and the Apgar scores ranged from 9 to 10 (median 9) at 5 min. All infants were born to mothers who did not smoke during or after pregnancy and all routinely slept supine at home. Dummy/pacifier use was self-selected by the parent and the infant; thus, there was no control over which infants were provided with a dummy/pacifier or for how long the infant used the dummy/pacifier over the first 6 months after birth.

### 2.2. Polysomnography and BP measurement

Daytime polysomnographic studies (0900–1600) were performed at 2–4 weeks, 2–3 months, and 5–6 months as described previously [24,25]. Prior to each recording, the infant's date of birth, gender, age, birth weight, weight at study, and postal code were recorded.

Each infant was made to sleep in both the prone and supine sleeping positions. The initial starting position was randomized and changed between morning and afternoon sleep periods that were interrupted by a midday feed. At the time of the study, the infants were constantly monitored by the bedside staff to ensure that, when prone, the infant's head was never face down or buried in the bedding and always open to the air. Skin and room temperature were constantly monitored along with HR, oxygen saturation, and breathing respiratory movements by a second staff member outside of the monitoring room.

Electroencephalogram, electro-oculogram, sub-mental electromyogram, electrocardiogram, thoracic and abdominal breathing movements, arterial blood oxygen saturation, and abdominal skin temperature were attached to the infants during the routine morning feed. The BP was recorded using a photoplethysmographic cuff (Finometer™, FMS, Finapres Medical Systems, Amsterdam, the Netherlands) referenced to heart level placed around the infant's wrist as described previously [26]. Beat-beat BP measurements were made

in 1–2-min epochs for the duration of each sleep cycle, with 5–8 min being recorded in each infant for each sleep state in each position. The sleep state was defined as quiet sleep (QS) or active sleep (AS) [27,28].

Data were recorded at 512 Hz (Compumedics E-Series Sleep Recording system, Compumedics Limited, Abbotsford, VIC, Australia) and exported via European Data Format to analysis software (Chart 7.2, ADInstruments, Sydney, NSW, Australia).

### 2.3. Data analysis

The infants' age and weight were averaged at each age studied. The socio-economic status was determined using the Australian Bureau of Statistics Socioeconomic Indexes for Areas 2011 based on the postal code [29]. This index provides a score that is based on key variables, including household income, education, occupation and ethnicity. The lower the index value, the greater the disadvantage for the residential area.

In dummy/pacifier users, sucking was confirmed via video recordings and defined if (1) the dummy/pacifier was in the infant's mouth and (2) there was a clear increase in the electromyograph (EMG) during each sucking event. Data were averaged over 2-min epochs in each sleep state, sleeping position and postnatal age (PNA). In dummy/pacifier users, data were grouped for non-sucking epochs and sucking epochs at each PNA.

#### 2.3.1. Assessment of autonomic control

Spectral analysis techniques were used to assess the autonomic control of HR, BP and baroreflex function. The autonomic nervous system mediates oscillations in HR and BP predominantly in the low-frequency (LF) and high-frequency (HF) spectral ranges. LF changes in HR and BP reflect both sympathetic and parasympathetic activation for HR variability (HRV) [30,31] and sympathetic vasomotor modulation for blood pressure variability (BPV) [32–34]. HF HRV is attributed to respiratory-related changes and reflects the parasympathetic modulation of the heart [31]. The HF component of BPV, while influenced by respiration, is also influenced by parasympathetic activity [32,33]. The ratio between the LF and HF spectral power (LF/HF) provides a measure of sympatho-vagal balance [31,35].

#### 2.3.2. Assessment of baroreflex function

The baroreflex is the principal regulatory mechanism for the short-term control of BP and is non-invasively quantified by the assessment of baroreflex sensitivity (BRS) using spontaneous changes in HR and BP [36]. To estimate BRS, a cross-spectral analysis of systolic blood pressure (SBP) and R–R interval data was performed using the methods described previously [37]. Briefly, in infants, baroreflex-related changes on HR and BP occur within the LF band (defined as 0.04–0.15 Hz). Using transfer function analysis, the gain between SBP and R–R interval was computed within the LF band. The gain represents the ratio of the amplitude of fluctuations in the R–R interval over those in SBP and provides an estimate of BRS.

All artefact-free epochs of 1–2 min duration were analysed. The mean arterial pressure (MAP) and HR were calculated via peak detection using Labchart 7.2 (ADInstruments, Sydney, NSW, Australia). For HRV, BPV and BRS, SBP and R–R interval were determined via peak detection and Fast Fourier transformation performed to compute the spectral power for SBP and R–R series using MATLAB (Mathworks, Natick, MA, USA) [3,4,37]. For HRV and BPV, the LF (0.04–0.15 Hz) power reflecting baroreflex activity, the HF power (individualized for each infant depending on respiratory frequency), and total power were calculated [35,38]. BRS was calculated using transfer function analysis between SBP and R–R interval changes within the LF range [37].

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