CPAP and Oral Feeds

CPAP has a **complex and multifaceted impact on oral feeding readiness** in preterm infants. It does not directly reduce pharyngeal or esophageal muscle tone, but it alters aerodigestive reflex coordination and swallowing mechanics in ways that increase aspiration risk during active oral feeding. The concern for oral aversion is primarily related to prolonged NPO status and negative perioral experiences rather than a direct physiological effect of CPAP itself.

Impact on Pharyngeal and Esophageal Function: CPAP does not decrease intrinsic pharyngeal or esophageal muscle tone per se, but it significantly alters the mechanics and coordination of swallowing:

Pharyngeal swallowing disruption: A video fluoroscopic swallow study (VFSS) by Ferrara et al. (2017) demonstrated that oral feeding while on nCPAP significantly increased the incidence of **deep laryngeal penetration** ($p = 0.03$) and **tracheal aspiration** ($p = 0.01$) compared with feeding off CPAP. Mild penetration and nasopharyngeal reflux were not affected. The pressurized airflow is suspected to interfere with laryngeal closure during swallowing.[1]

Aerodigestive reflex alterations: Jadcherla et al. (2016) performed pharyngoesophageal manometry in 38 infants (~28 weeks GA) and found that infants on nCPAP or nasal cannula (vs. room air) had **increased pharyngeal waveform recruitment and duration, greater UES nadir pressure, decreased esophageal contraction duration, decreased distal esophageal contraction amplitude, and decreased completely propagated esophageal peristalsis** (all $p < 0.05$). However, upper and lower esophageal sphincter protective reflexes remained intact, suggesting that GERD concerns are unfounded in this population.[2]

Esophageal motility during nutritive swallowing: A preclinical study (Djeddi et al., 2013) using esophageal multichannel intraluminal impedance-pH monitoring in newborn lambs found that nCPAP at 6 cmH_2O **did not alter nutritive esophagodeglutition** in any measured variable ($p > 0.1$ for all), suggesting the esophageal phase of swallowing is preserved.[3]

In summary, CPAP primarily disrupts the **pharyngeal phase** of swallowing (laryngeal closure and airway protection) rather than reducing esophageal muscle tone or peristalsis.

Impact on Suck-Swallow-Breathe Coordination: Coordination of sucking, swallowing, and breathing matures at approximately **32–34 weeks PMA**.[4] CPAP affects this coordination through several mechanisms:

Positive pressure may **inhibit laryngeal closure** and interfere with the swallowing reflex, as the continuous airflow competes with the normal respiratory pause during swallowing.[5]

Chronic lung disease (BPD), which often necessitates prolonged CPAP, independently delays the development of suck-swallow-breathe coordination, making it difficult to separate the effects of CPAP from those of the underlying respiratory disease.[5] Tachypnea and dyspnea from ongoing respiratory disease further challenge swallowing and feeding skill acquisition.[6]

Does CPAP Cause Oral Aversion?
There is no direct evidence that CPAP itself causes oral aversion, but the **prolonged withholding of oral feeding** while on CPAP, combined with negative perioral stimuli (nasal prongs, orogastric tubes, suctioning), is recognized as a contributor to oral aversion and feeding difficulties:[6][4]

Medical interventions including prolonged intubation, CPAP, nasal cannula, and regular oropharyngeal/nasal suctioning may result in **negative responses to oral feeding** and long-term oral sensitivity.[6]

A 2026 U.S. cross-sectional survey of 159 NICUs found that **83% of units never allow oral feeding on CPAP**, meaning infants on prolonged CPAP are kept NPO for extended periods. The concern is that these delays may **increase oral aversion and length of stay**.[4]

However, Dumpa et al. (2020) found that delaying oral feeding until after CPAP discontinuation did not contribute to feeding-related morbidities. Infants who started oral feeds later reached full oral feeding at a similar PMA as those who fed on CPAP. Notably, there was a positive correlation between the duration of oral feeding while on CPAP and time spent on respiratory support, suggesting that feeding on CPAP may not accelerate the transition.[7]

Does Delaying Oral Feeds Until Off CPAP Harm Outcomes?
The evidence suggests that timely CPAP weaning is more important than attempting oral feeds while on CPAP:

Implementing standardized CPAP weaning guidelines reduced the time to achieve full oral feeds by **7 days** (29 → 22 median days, $p < 0.001$), without increasing BPD or total respiratory support days.[8]

Oral stimulation programs (non-nutritive sucking, perioral stimulation) can be safely provided while on CPAP to maintain oromotor development and reduce the risk of oral aversion, even when nutritive oral feeding is withheld.[6]

Practical Implications

Concern	Evidence Summary	Clinical Implication	References
Aspiration risk on CPAP	VFSS shows increased deep penetration and aspiration on CPAP	Caution with oral feeds on CPAP; consider VFSS if feeding on CPAP	[1]
Esophageal motility	Esophageal phase of swallowing preserved on CPAP	GERD concern from CPAP is unfounded	[2], [3]
Pharyngeal reflexes	Altered pharyngeal waveform recruitment and decreased esophageal peristalsis propagation	Infants may benefit from targeted oromotor therapies	[3]
Oral aversion	Related to prolonged NPO + negative perioral stimuli, not CPAP per se	Non-nutritive sucking and oral stimulation programs while on CPAP	[4], [5]
Feeding timeline	Delaying oral feeds until off CPAP does not delay PMA at full oral feeds	Prioritize efficient CPAP weaning over feeding on CPAP	[6], [7]

The most effective strategy to promote oral feeding readiness is **efficient, standardized CPAP weaning** combined with **non-nutritive oral stimulation programs** (pacifier use, perioral stimulation) during the CPAP period to maintain oromotor development. When oral feeding on CPAP is considered, it should involve strict readiness criteria, SLP/OT involvement, and continuous monitoring, as practiced by the small minority (~9%) of U.S. NICUs that routinely permit it.[4]

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Prolonged CPAP and Delayed Oral Feeding Readiness

Preterm infants who have received **prolonged CPAP (or other noninvasive respiratory support) do experience oral feeding challenges and delays even after CPAP is discontinued**, though the evidence suggests this is primarily driven by the severity of underlying lung disease rather than a direct lasting effect of CPAP itself.

Duration of Noninvasive Ventilation Independently Predicts Delayed Full Oral Feeding
In a retrospective study of 122 infants born <32 weeks, **duration of noninvasive ventilation** was independently associated with an older PMA at full oral feeding (FOF) in multivariable regression analysis, alongside BPD, oxygen therapy duration, and patent ductus arteriosus. The median PMA at FOF was **36 6/7 weeks**, with BPD as the single most potent predictor of delayed FOF.[1] Similarly, a Cox regression analysis of 110 preterm infants with RDS found that **noninvasive ventilation >14 days** was independently associated with delayed achievement of full enteral feeding ($p < 0.05$).[2]

The PMA at which oral feeding is first introduced also correlates strongly with the duration of respiratory support. The longer the respiratory support, the later the first oral feed, and the later FOF is achieved. Importantly, PMA at first oral feed was the single most significant predictor of PMA at FOF ($r = 0.61$, $p = 0.0001$), suggesting that the delay in initiating oral feeds during prolonged CPAP has a compounding effect.[3]

Prolonged CPAP and Gastrostomy Tube Placement
Anderson et al. (2022) studied infants born <30 weeks and found that those who ultimately required a **gastrostomy tube (GT)** at discharge had CPAP to a significantly later PMA than those who did not (median PMA at last CPAP day: **39.9 weeks vs. 32.6 weeks**, $p < 0.0001$). Every respiratory variable, total ventilator days, total ventilator + CPAP days, PMA at last ventilator day, and PMA at last CPAP day, was significantly different between GT and non-GT groups. However, in the final multivariable predictive model, only exposure to high-frequency ventilation (HFV) remained as an independent respiratory predictor, suggesting that the need for prolonged CPAP is a **marker of disease severity** rather than an independent causal factor for GT placement.[4]

Mechanisms of Feeding Difficulty After Prolonged CPAP: Several interrelated mechanisms contribute to feeding

challenges after prolonged CPAP:

Missed critical window for oromotor development: Suck-swallow-breathe coordination matures between 32–34 weeks PMA. Infants on prolonged CPAP during this window are typically kept NPO (83% of U.S. NICUs never allow oral feeding on CPAP), depriving them of essential sensory and motor experiences during a critical period of brain development when central patterning of suck and feeding skill is refined.[5][6]

Negative perioral conditioning: Prolonged exposure to nasal prongs, orogastric/nasogastric tubes, and regular oropharyngeal suctioning may result in negative responses to oral feeding and long-term oral sensitivity, even after CPAP is discontinued.[6]

Residual respiratory compromise: Even after CPAP is weaned, many of these infants have BPD or CLD that causes tachypnea and dyspnea, which directly challenge swallowing coordination and feeding skill acquisition.[6][4]

Deconditioning of oromotor musculature: Prolonged tube feeding without oral stimulation leads to disuse of the oral musculature needed for nutritive sucking.[3]

Does Delaying Oral Feeds Until Off CPAP Worsen Outcomes?
Reassuringly, Dumpa et al. (2020) found that infants whose oral feeds were delayed until after CPAP discontinuation reached full oral feeding at a **similar PMA** as those who were fed on CPAP, and delayed feeding did not contribute to feeding-related morbidities.[7] This suggests that the delay in oral feeding per se does not cause lasting harm. The key determinant is the infant's underlying respiratory trajectory.

Longer-Term Feeding Difficulties
Extreme prematurity, which is closely linked to prolonged CPAP use, is a recognized risk factor for **longer-term oromotor feeding problems** at 12 months corrected age and even at school age.[6] Post-discharge, parents consistently report physiological difficulties including infant sleepiness, poor latch, fatigue during feeds, weak sucking, and impaired suck-swallow-breathe coordination.[8] Some infants remain partially or exclusively tube-fed for months or years after discharge.[6]

Mitigating Strategies - The most effective approaches to reduce feeding delays in infants who have been on prolonged CPAP include:

- **Oral stimulation programs during CPAP:** Non-nutritive sucking (NNS) and perioral stimulation can be safely provided while on CPAP to maintain oromotor development. Combining colostrum administration with NNS in VLBW infants on noninvasive ventilation significantly shortened time to initiate oral feeding, achieve complete oral feeding, and reduced feeding intolerance (all $p < 0.05$).[9]
- **Efficient, standardized CPAP weaning:** Implementing gradual pressure weaning guidelines reduced time to full oral feeds by **7 days** (29 → 22 median days, $p < 0.001$).[10]
- **Early SLP/OT involvement:** Units that do permit oral feeding on CPAP use strict readiness criteria with speech-language pathology/occupational therapy involvement, which may help identify and address feeding difficulties earlier.[5]

Summary
Prolonged CPAP is strongly associated with delayed oral feeding and, in the most severe cases, need for GT

placement at discharge. However, this relationship is largely mediated by the severity of underlying lung disease (particularly BPD) rather than a direct lasting effect of CPAP on oromotor function. The most impactful interventions are efficient CPAP weaning, oral stimulation programs during the CPAP period, and early involvement of feeding specialists once CPAP is discontinued.

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