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Case Report

Tidal-Breathing Analysis: A Potential Non-Invasive, Bedside Method for Objective Assessment of Respiratory Mechanics and Bronchodilator Responsiveness in Acute Bronchiolitis—Case Report and Review of the Literature

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Abstract

Background: Acute bronchiolitis is among the most common causes of lower-respiratory-tract infection in infancy and remains a clinically heterogeneous syndrome. Current international guidelines recommend supportive management and generally discourage routine bronchodilator administration; however, a subset of patients may be physiologically responsive. Tidal-breathing analysis (TBA) is a non-invasive, bedside method for objectively assessing respiratory mechanics and may objectively quantify physiological changes following bronchodilator administration in older children. **Case Presentation**: We report the case of a 23-month-old boy presenting with a severe RSV bronchiolitis-like wheezing illness complicated by acute respiratory failure. Bronchodilator responsiveness was evaluated using predefined TBA parameters before and after administration of an inhaled bronchodilator. Following treatment, the patient's time to peak tidal expiratory flow (tPTEF) increased from 0.17 to 0.33 s (+94%), while his tPTEF/TE ratio increased from 0.12 to 0.27 (+125%). Expiratory time (TE) and total respiratory cycle duration (Ttot) decreased by 12.6% and 5.1%, respectively, whereas tidal volume (VT) and minute ventilation (MV) increased by 12.1% and 18.3%. Peak expiratory flow remained unchanged (0.14 L/s before and after bronchodilator administration). **Discussion**: To the best of our knowledge, this is among the first reports of using bedside TBA to characterize acute bronchodilator responsiveness in a severe bronchiolitis-like illness. The improvement in expiratory timing indices, along with the shorter expiratory time without changes in peak expiratory flow, is consistent with improved expiratory flow dynamics following bronchodilator administration. These findings could support the hypothesis that a bronchodilator-responsive physiological phenotype may exist. Previous studies have demonstrated an association between severe bronchiolitis during infancy and subsequent recurrent wheezing or asthma; however, whether acute bronchodilator responsiveness, measured objectively via TBA, is an indication of this phenotype represents a testable hypothesis for future longitudinal studies. **Conclusions**: This case illustrates the feasibility of bedside TBA for identifying physiological bronchodilator responsiveness in severe bronchiolitis-like illness. Prospective longitudinal studies should be conducted to determine whether TBA-defined responsiveness represents an early physiological marker requiring prospective validation and whether such physiological stratification may support individualized follow-up strategies.

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Keywords: bronchiolitis; salbutamol response; tidal breathing analysis; tPTEF/TE; asthma risk

1. Introduction

Bronchiolitis, characterized by inflammation of the small airways that results in tachypnoea, wheezing, increased work of breathing, and sometimes hospitalization, is the most frequent lower-respiratory-tract infection in infants. Respiratory syncytial virus (RSV) was the leading cause of bronchiolitis before prophylaxis protocols were implemented (namely, maternal immunization during pregnancy and administration of monoclonal antibodies for infants), with rhinovirus (RV) and other viruses contributing substantially [1,2]. Airway interactions between viruses, especially rhinoviruses, and potentially pathogenic bacteria (Streptococcus pneumoniae, Hemophilus influenzae, and Moraxella catarrhalis) in early infancy may increase the risk of subsequent wheezing and asthma [3]. While most cases resolve, a certain subset of children develop recurrent wheezing or asthma, which, in some prospective-followed cohorts, occurred up to 4 years after the initial infection, with the proportion affected being as high as 12% [4]. Hospitalization for severe bronchiolitis is associated with an increased risk of subsequent asthma (with a two- to three-fold increase commonly reported across cohorts) [5–9]. Severe bronchiolitis in infancy may trigger development of asthma in genetically susceptible individuals, and it could be a marker of predisposition for future asthma, because studies have found that infants with lower lung function shortly after birth have an increased risk of developing asthma by 10 years of age [10]. Genetic variants in CDHR3 and GSDMB modulate susceptibility to bronchiolitis, especially when it is caused by viruses other than RSV [11]. Host and environment early-life risk factors are associated with progression of wheezing symptoms over time, and several groups of researchers have speculated that dynamic interactions between these factors and an infant's developing respiratory system constitute the dominant factor for subsequent wheezing and asthma [12–14].

A key unresolved question is whether bronchiolitis leads to the development of asthma or unmasks pre-existing susceptibility [15–17] in certain children.

Current guidelines emphasize supportive care and discourage routine bronchodilator use in typical bronchiolitis cases because of an inconsistent clinical benefit at the general-population level [18–20]. Nevertheless, certain subgroups appear bronchodilator- or steroid-responsive, potentially signaling airway hyperreactivity akin to early asthma [21,22]. In adults, bronchodilator responsiveness, even in the presence of lung function within normal limits, might be a risk factor for incident chronic airflow obstruction [23]. In young pediatric asthma patients, small-airway dysfunction (SAD) could be present despite normal FEV_1 values [24], so exploring SAD, via tidal breathing analysis (TBA), could unmask future asthma trajectories in infants with severe bronchiolitis. Evidence of SAD was found in almost one-third of subjects who were ventilated for life-threatening RSV disease, although it was not always accompanied by respiratory symptoms [25].

TBA is a non-invasive pulmonary function technique that quantifies timing and flow indices from tidal breathing loops (e.g., tPTEF, tPTEF/TE, VT, and MV), and it has been validated in infants from various ethnic groups [26,27]. Changes in timing indices after bronchodilator therapy can objectively demonstrate responsiveness, although mild bronchiolitis cases are associated with rather poor responsiveness in sedated infants [28].

Here, we present a wheezing bronchiolitis-like LRTI case with documented salbutamol responsiveness determined via TBA, synthesize evidence on bronchiolitis–asthma links

and treatment response heterogeneity, and propose a testable hypothesis for research on physiology-defined responsiveness trajectories in these young children.

2. Case Presentation

A 23-month-old boy presented to the emergency department with a 2-day history of seromucous rhinorrhea, a productive cough, and dyspnea. His family history was notable for paternal asthma and a sibling who had severe bronchiolitis during infancy. The child, who was term-born (39 weeks gestation; birthweight: 2800 g; Apgar score: 9), was breastfed in early infancy, and his immunizations were up to date. His medical history included a febrile seizure at 9 months, measles at 12 months complicated by pneumonia, and one severe episode of bronchiolitis—with respiratory failure—at 19 months of age, requiring hospitalization. Environmental risk factors included exposure to household tobacco smoke and wood-stove heating and contact with domestic animals (dogs and backyard chicken), which could have changed his lung-function trajectory into a recurrent-wheezing phenotype.

On admission, the boy weighed 10.3 kg, was afebrile, and appeared normal. Our respiratory findings were as follows: nasal obstruction with seromucous rhinorrhea and a productive cough, tachypnea (54 breaths/min) with intercostal and subcostal retractions, and an SpO_2 of 92% in room air conditions, which improved to 99% with a low-flow nasal cannula. Chest auscultation revealed diminished vesicular breath sounds with bilateral wheezes and fine crackles. Cardiac examination revealed a regular rhythm with tachycardia (150 bpm). The abdomen was non-tender, and oral intake was reduced. But bowel movements were normal, and his urine output was preserved. No meningeal signs were present.

Laboratory evaluation revealed leukocytosis with neutrophilia and a normal eosinophil count (0.03×10^3/microliter or 0.1%). The patient's C-reactive protein concentration was 2 mg/dL. Elevated total IgE levels were documented (294 kUI/L—reference values for age in our lab = 0–40). Capillary blood gas analysis revealed respiratory acidosis (pH 7.20; pCO_2: 63 mmHg). The working diagnosis was acute RSV bronchiolitis (RADT documented in emergency department) complicated by acute respiratory failure and mild acute dehydration secondary to reduced intake. The patient was provided with non-invasive ventilatory support for a total of 7 days.

Tidal-breathing analysis (TBA) was performed with a SpiroScout (Ganshorn Medizin Electronic GmbH, Schiller Group, Germany) pneumotachograph-linked spirometer (see Figure 1) immediately before and ~20 min after administration of nebulized salbutamol (0.15 mg/kg). Recordings were acquired in a non-sedated, quiet–awake state using a well-fitted facemask with an optimized seal to minimize leakage and upper-airway artifacts. At each timepoint, tidal breathing was recorded for a stable period, and $\geq$3 technically acceptable segments were obtained, in accordance with ERS/ATS infant PFT standards (see Figure 2). Primary indices were derived from a prespecified, artifact-free segment using predefined quality criteria (a stable baseline, minimal leakage, and no cough/swallow/sigh). In cases of uncertainty, segment eligibility was adjudicated by two independent reviewers blinded with respect to test order.

The pre-salbutamol administration values were as follows: VT—107 mL; RR—28/min; MV—3.0 L/min; TI—0.72 s; TE—1.43 s; Ttot—2.15 s; PEF—0.14 L/s; tPTEF—0.17 s; and tPTEF/TE—0.12.

Post-salbutamol administration, tidal-breathing-index values improved markedly: tPTEF and tPTEF/TE increased from 0.17 to 0.33 s (+94%) and 0.12 to 0.27 (+125%), respectively, while TE and Ttot fell from 1.43 to 1.25 s (−12.6%) and 2.15 to 2.04 s (−5.1%), respectively. Minute ventilation increased from 3.0 to 3.55 L/min (+18.3%), mainly through

a higher VT (107→120 mL, +12.1%) with a small RR increase (28→30/min, +7.1%). PEF remained unchanged (0.14 L/s). This overall pattern supports bronchodilator responsiveness and is consistent with an improved tidal expiratory profile. Key parameters are summarized in Table 1 and Figure 3.

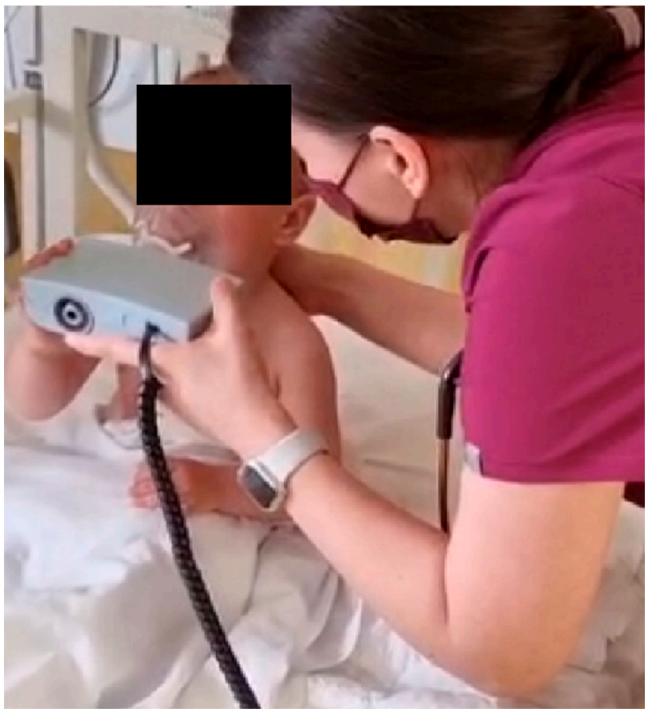


Figure 1. Bedside tidal-breathing analysis used in a case of bronchiolitis. The non-sedated patient was subjected to tidal-breathing flow–volume loop (TBVL) acquisition using a pediatric facemask connected to a pneumotachograph (Ganshorn). The operator supported the patient's head/neck and maintained a gentle mask seal to minimize leakage and upper-airway artifacts.

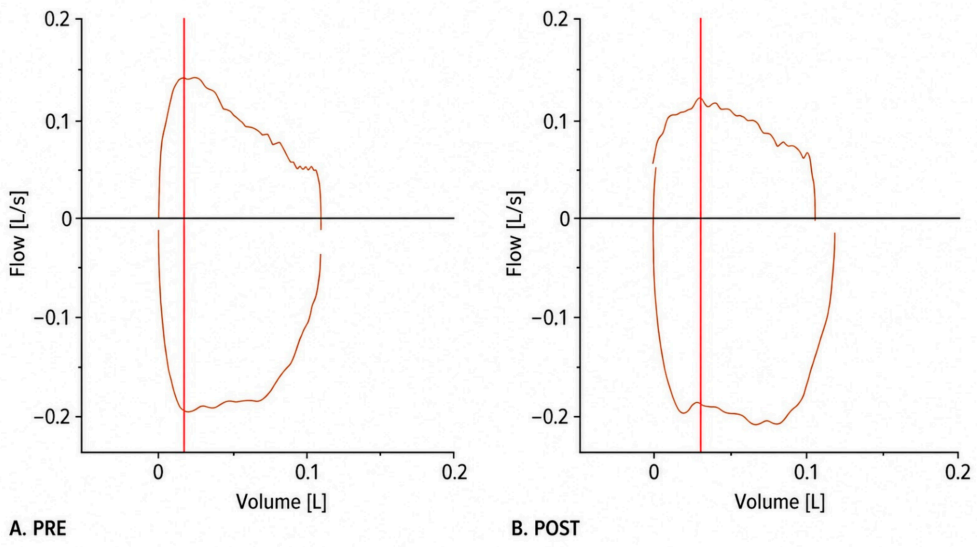


Figure 2. Tidal-breathing flow–volume loops pre- and post-salbutamol administration. (**A**) Pre: A scooped expiratory limb with delayed time-to-peak expiratory flow (tPTEF) and prolonged expiration, a finding consistent with expiratory flow limitation. (**B**) Post: A larger loop with later peak timing indices (tPTEF 0.17 → 0.33 s; tPTEF/TE 0.12 → 0.27) and shorter expiration (TE 1.43 → 1.25 s; Ttot 2.15 → 2.04 s); PEF remains 0.14 L/s. Axes: flow (L/s) vs. volume (L). Morphology indicates clear bronchodilator responsiveness.

Table 1. Tidal-breathing parameters before and after the SABA challenge.

Parameter	Unit	Pre	Post	Absolute Change	Percentage Change (%)
PEF	L/s	0.14	0.14	0	0
tPTEF	s	0.17	0.33	0.16	94.1
Ttot	s	2.15	2.04	−0.11	−5.1
TI	s	0.72	0.79	0.07	9.7
TE	s	1.43	1.25	−0.18	−12.6
tPTEF/TE	ratio	0.12	0.27	0.15	125
MV	L/min	3	3.55	0.55	18.3
RR	1/min	28	30	2	7.1
VT	mL	107	120	13	12.1

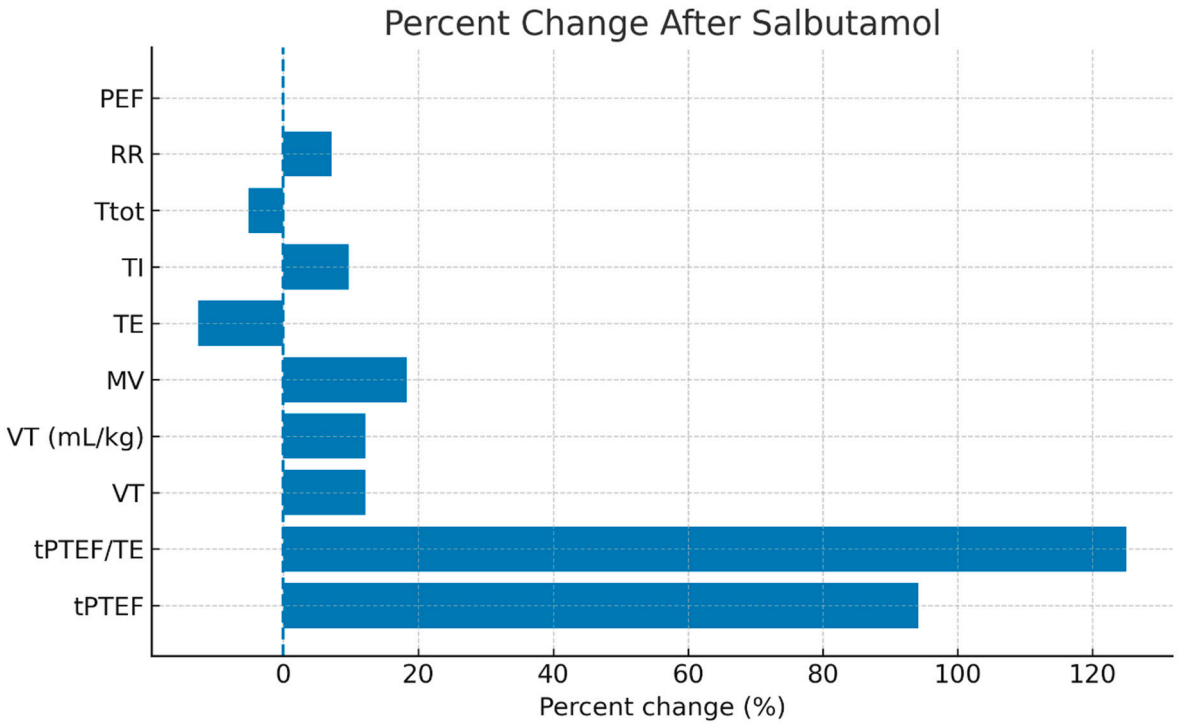


Figure 3. Percent change in tidal-breathing parameters after the salbutamol challenge.

Management comprised intravenous rehydration for 2 days when respiratory distress was impeding normal fluid intake; a high-flow nasal cannula (HFNC) for two days and low-flow nasal cannula (LFNC) support for up to 7 days; systemic corticosteroid therapy with methylprednisolone administered at 2 mg/kg/day for 2 days followed by tapering; inhaled bronchodilator therapy with nebulized salbutamol for a total of 9 days, with adjunctive ipratropium bromide for administered for the first 5 days; and inhaled fluticasone propionate. The clinical course was favorable, with a rapid decrease in distress and normalization of blood gas measurements in 2 days and constant improvement in respiratory status over one week.

3. Data from the Literature

3.1. Etiology and Epidemiology

Bronchiolitis primarily affects children under 2 years of age, with RSV being responsible for the majority of cases; rhinovirus, parainfluenza, human metapneumovirus, and adenovirus are also involved, and co-infections are common [1,2]. The global burden is substantial, with RSV being a leading cause of lower-respiratory-tract infection in young children [1]. New prevention tools, like long-acting RSV monoclonal antibodies (nirsevimab and clesrovimab) and the RSV-prefusion-F-protein-based maternal vaccine during pregnancy, have had an impact on the real-life burden regarding hospitalizations and emergency department visits among infants [29–33], and they will probably generate a more tailored approach to RSV-afflicted wheezy infants in the foreseeable future because there is still a heterogenous approach to these patients [34].

3.2. Association with Asthma

Multiple studies link severe bronchiolitis to an increased risk of developing asthma, and rhinovirus-associated bronchiolitis generally carries a higher subsequent risk than RSV [35–39]. One study [35] including 408 children hospitalized for bronchiolitis in a prospective, three-center, 4-year follow-up study conducted in Finland documented a median age of 7.5 months upon inclusion in the study. In total, 42% had RSV, 29% had RV, 2% had both RSV and RV, and 27% had a non-RSV/-RV etiology. The children with RV-A (adjusted hazard ratio, 2.3; $p = 0.01$), RV-C (adjusted hazard ratio, 3.5; $p < 0.001$), and non-RSV/-RV (adjusted hazard ratio, 2.0; $p = 0.004$) bronchiolitis started taking the asthma control medication earlier than the children with RSV bronchiolitis.

INSPIRE is a large, population-based birth cohort of healthy infants with non-low birthweights born at term in Tennessee, USA. These children were followed up prospectively for the primary outcome of 5-year current asthma. Not being infected with RSV during infancy was associated with a 26% lower risk of 5-year current asthma than being infected with RSV during infancy (adjusted RR, 0.74; 95% CI, 0.58–0.94; $p = 0.014$). The estimated proportion of 5-year current asthma cases that could be prevented by avoiding RSV infection during infancy was 15% [40].

Several studies on non-invasive biomarkers useful for assessing asthma in preschool children have been published [41–43]. The specimens that can be easily obtained using non-invasive methods are blood, exhaled breath, and urine. Eosinophils, eosinophil cationic protein, and eosinophil-derived neurotoxin (EDN) in the blood are helpful for evaluating eosinophilic inflammation of the airways. Exhaled breath contains nitric oxide, volatile organic compounds, and various cytokines and mediators, all of which serve as analytical components. The fraction of exhaled nitric oxide has been used to assess the degree of eosinophilic inflammation. It has been standardized for school-age children and adults but not yet for preschool children. Exhaled breath condensate (EBC) pH and various cytokines/mediators that are detected in EBC seem to be promising biomarkers for assessing asthma, but further standardization and validation are required.

Dysregulated interferon responses (changes in gene transcription during cold-associated asthma exacerbations) are important contributors to this risk in children. Low baseline interferon expression, followed by greater upregulation of interferon pathways, in the airway and blood during respiratory illnesses increases the risk of exacerbation [44].

Bronchiolitis cohort studies have identified early-life environmental-exposure-related [45], genetic [46–49], and immune risk factors [50] of childhood asthma development by carrying out analysis at a single level (e.g., analyzing associations with respiratory virus types, host immune response, or the composition of the host's microbiome) [51]. While multi-omics technologies have elucidated certain mechanisms underlying the gut–lung axis,

numerous pathways remain to be fully delineated [52]. Severe bronchiolitis pathogenesis involves interaction of factors at multiple levels (e.g., the genome, epigenome, transcriptome, metabolome, and microbiome levels) [53–55]. Proposed mechanisms through which early-life RSV infection might contribute to asthma include epithelial injury, immune dysregulation with Th2-skewed responses, and airway/microbiome remodeling, in conjunction with host susceptibility [56].

3.3. Bronchodilator Response in Bronchiolitis

Guidelines discourage using routine β_2-agonists in typical bronchiolitis because of nonexistent or inconsistent clinical benefits [18,19]. Nonetheless, subgroup patients may respond, potentially indicating asthma-like airway hyperreactivity [21]. An old meta-analysis [57] found no overall clinical benefits among infants treated with bronchodilators: they do not improve oxygen saturation, reduce hospital admission after outpatient treatment, shorten the duration of hospitalization, or reduce the time until the illness is resolved at home. Some trials suggest that epinephrine may transiently reduce early admission in emergency settings, especially in combination with dexamethasone, as noted in the BIPED study [58].

3.4. Tidal-Breathing Analysis

TBA can be used to identify potential responders via meaningful improvements in timing indices (e.g., tPTEF/TE), supporting physiology-based phenotyping. TBA is feasible for non-sedated children and quantifies airflow obstruction through timing and flow indices [59]. A greater tPTEF/TE and a higher respiratory rate but similar V_T while awake relative to sleeping suggest that separate normative TFV loop values according to arousal state may be required in early infancy [60]. In wheezing infants, lower tPTEF/TE values have been reported in those with more persistent symptoms, although variability necessitates standardized acquisition and loop selection [61].

3.5. Treatment Reviews for Acute Bronchiolitis

Supportive care is a cornerstone, comprising hydration, oxygen, and suctioning [62].

Hypertonic saline may modestly reduce hospital length of stay and slightly improve clinical severity scores (CSSs). Treatment with nebulized hypertonic saline may also reduce the risk of hospitalization amongst outpatients and ED patients, although in a randomized clinical trial of 777 infants, the hospital admission rate in the hypertonic saline group was 48.1%, while it was 52.2% in the normal saline group [63]. Nebulized hypertonic saline seems to be a safe treatment for infants with bronchiolitis, with only minor and spontaneously resolved adverse events, especially when administered in conjunction with a bronchodilator, as documented in a recent review including a total of 34 trials, involving 5205 infants with acute bronchiolitis, 2727 of whom received hypertonic saline [64].

In studies that explored a combination of medications, low-quality evidence suggested that nebulized epinephrine plus hypertonic saline may be considered a safe and efficient therapy for decreasing length of stay and CSSs among infants with acute bronchiolitis, especially among those who require hospitalization for more than 48 h [65]. A total of 69 infants were enrolled in a prospective, double-blind, placebo-controlled trial and randomly assigned to receive nebulized salbutamol, ipratropium bromide, or a placebo. The authors concluded that clinical scores and oxygen saturation levels improved more rapidly in the bronchodilator groups than in the placebo group for up to 24 h, but the effect of these drugs was not sufficient to change the natural course of the disease [66].

Corticosteroids generally show no consistent benefits in typical bronchiolitis; evidence of benefits in combination regimens (e.g., with epinephrine) has been mixed across trials [67], but among children with severe bronchiolitis, the duration of clinician-managed

pressure support in intensive care (defined as high-flow nasal-prong oxygen, nasopharyngeal continuous positive airway pressure, or mechanical ventilation) was reduced (relative to standard care) by regular treatment with systemic corticosteroids and inhaled epinephrine [68].

Antibiotics are ineffective in the absence of bacterial co-infection [69], except for azithromycin, which may reduce hospitalization time regarding acute bronchiolitis and wheezing episodes among children aged less than two years. Azithromycin administered during the acute wheezing period does not have a preventive effect on wheezing recurrence [70].

DNase has been explored for mucus clearance, but evidence is limited [71].

4. Future Research

4.1. Study Question

Does acute bronchodilator responsiveness during an acute bronchiolitis episode, defined by prespecified TBA criteria, predict higher 5-year asthma risk compared with non-responsiveness?

4.2. Objectives

Primary: Compare physician-diagnosed asthma at 5 years (as per GINA 2026 criteria for asthma in children 5 years and younger) [72] among salbutamol-responders and non-responders (TBA-defined).

Secondary: (i) acute changes in TBA after salbutamol; (ii) proportion classified as responders; (iii) predictors of response (family history, viral etiology, tobacco smoke exposure, biomarkers); (iv) associations between response and acute severity (PRESS, oxygen/HFNC, length of stay, PICU) [71,73].

5. Discussion

Most infants afflicted with wheezing have transient conditions associated with diminished airway function at birth and do not have an increased risk of asthma later in life. Overall, 30–40% of children with severe bronchiolitis develop childhood asthma, but accurate prediction remains challenging. In a severe bronchiolitis cohort consisting of 875 children, the authors of one study applied the Asthma Predictive Index (API), the modified Asthma Predictive Index (mAPI), and the Pediatric Asthma Risk Score (PARS) to predict asthma at age 5. A total of 294 (34%) asthma patients were diagnosed, but all three predictive tools yielded sub-optimal performance (AUC < 0.8) [74]. To diagnose asthma in young children, family physicians have to rely on history taking and physical examination, as spirometry is difficult to perform in primary-care settings for this age group. The additional diagnostic value of specific immunoglobulin E (IgE) for inhalant allergens was explored in a cohort of 752 children aged 1–4, introducing a potentially useful diagnostic indicator of the presence of asthma at the age of 6 [75].

Although recent guidelines recommend taking a minimalist approach to bronchiolitis, there are some issues with this approach. First, there are concerns about the precise definition of the disease, the quality and uniformity of the guidelines, the method of administering bronchodilators, and the availability of tools for evaluating the response to therapies [76–78]. Second, for decades, it has been assumed that all cases of viral bronchiolitis are similar, but recent evidence has shown that this is false. Distinct bronchiolitis phenotypes have been described, with heterogeneity in clinical presentation, molecular immune signatures, and clinically relevant outcomes such as respiratory failure and recurrent wheezing [79]. All these limitations are more stringent in primary-care settings, even in developed countries [80].

The physiological changes observed after salbutamol administration were compatible with bronchodilator responsiveness, objectively assessed via TBA, in a child presenting with a second episode of recurrent wheezing triggered by a respiratory-syncytial-virus-induced lower-respiratory-tract infection. During the initial episode three months earlier, which required hospitalization in our unit for severe bronchiolitis (pediatric respiratory severity score [PRESS] = 5), the patient exhibited a clinically significant response to inhaled salbutamol. However, TBA was not performed at that time to objectively quantify changes in lung function parameters.

During this second major broncho-obstructive episode, which required hospitalization, nebulized salbutamol administration resulted in a marked increase in respiratory timing indices (tPTEF and tPTEF/TE), a shortening of expiratory duration, and increased minute ventilation, while peak expiratory flow (PEF) remained unchanged. The observed alterations in TBA waveform morphology were consistent with an improved tidal expiratory pattern in the absence of an increase in peak flow, indicating the presence of pharmacologically mediated bronchodilation. In this patient, who appeared to follow a recurrent wheezing trajectory compatible with a viral-induced asthma phenotype, responsiveness to short-acting β_2-agonists was objectively documented. This interpretation accounts for potential state-dependent physiological variability and measurement-related fluctuations that may influence respiratory timing indices in young children.

This pattern seems similar to that reported by a group of researchers who monitored 76 children presenting with their first wheezing episode at the ages of 3 to 23 months. Upon study entry, viral etiology, rhinovirus genome load, atopic and clinical characteristics, and answers to a standardized questionnaire were analyzed. At the 4-year follow-up visit, impulse oscillometry with an exercise challenge was performed. Bronchial reactivity (a $\geq$35% change in mean crude values of resistance) after the exercise challenge or bronchodilation was present in nine (12%) children [81].

This paper has several limitations: It presents only one case in which TBA was used to describe bronchodilator responsiveness in a 23-month-old child who had suffered a previous wheezing episode. This scenario can be considered hypothesis-generating, and it may provoke future research. As a strength of this case, to the best of our knowledge, it is among the first reports of using bedside TBA to characterize acute bronchodilator responsiveness in a severe bronchiolitis-like illness. Another limitation of our findings is that the results are very sensitive to operator skills, and the procedure employed could be difficult to duplicate in a setting lacking access to dedicated pediatric pulmonologists working with infants and young children on a daily basis.

We must also highlight that our patient was older than average acute bronchiolitis patients and fully cooperative with the TBA procedure, as shown in Figure 1. Non-cooperative infants can be evaluated while they sleep, with the main limitation being that some TBA parameters could be influenced during wakefulness [25,60].

Future precision medicine strategies integrating validated asthma prediction models and pulmonary function tests with artificial intelligence-based platforms and machine learning models that incorporate electronic health records, genomic data, and molecular biomarkers may further optimize individualized management and improve long-term quality of life in some high-risk populations [82].

6. Conclusions

Bedside physiological assessment can reveal clinically relevant heterogeneity within phenotypically typical bronchiolitis presentations.

Although routine bronchodilator therapy is not recommended at the population level, physiology-guided bedside trials may provide a pragmatic and evidence-based approach,

complementing the clinical and medical history patterns used for identifying selected potential responders without relying on empirical trial-and-error management strategies.

In future research, TBA-based phenotyping could refine acute therapeutic decision-making, enable risk-stratified surveillance during early childhood, and be incorporated into artificial intelligence-driven personalized treatment algorithms.

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Informed Consent Statement: Written informed consent was obtained from the patient's legal guardian for participation in this study and for the publication of the patient's clinical information and anonymized image.

Data Availability Statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request. The data are not available due to privacy and ethical restrictions.

Conflicts of Interest: The authors declare no conflicts of interest.

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