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Marc Tebruegge [ab]; Anastasia Pantazidou [a]; Kent Thorburn [c]; Andrew Riordan [d]; Jonathan Round [e]; Claudine De Munter [cf]; Sam Walters [bf]; Nigel Curtis [a]
[a] From the Department of Paediatrics, The University of Melbourne & Infectious Diseases Unit, Department of General Medicine & Murdoch Children's Research Institute, Royal Children's Hospital Melbourne, Parkville, Australia [b] Paediatric Infectious Diseases, St. Mary's Hospital, London, UK [c] Departments of Paediatric Intensive Care, [d] Paediatric Infectious Diseases, Royal Liverpool Children's Hospital (Alder Hey), Liverpool, UK [e] Department of Paediatric Intensive Care, St. George's University Hospital, London, UK [f] Imperial College Medical School, London, UK

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ORIGINAL ARTICLE

Bacterial tracheitis: a multi-centre perspective

MARC TEBRUEGGE[1,6], ANASTASIA PANTAZIDOU[1], KENT THORBURN[2], ANDREW RIORDAN[3], JONATHAN ROUND[4], CLAUDINE DE MUNTER[5,7], SAM WALTERS[6,7] & NIGEL CURTIS[1]

From the [1]Department of Paediatrics, The University of Melbourne & Infectious Diseases Unit, Department of General Medicine & Murdoch Children's Research Institute, Royal Children's Hospital Melbourne, Parkville, Australia, Departments of [2]Paediatric Intensive Care & [3]Paediatric Infectious Diseases, Royal Liverpool Children's Hospital (Alder Hey), Liverpool, UK, [4]Department of Paediatric Intensive Care, St. George's University Hospital, London, UK, [5]Departments of Paediatric Intensive Care & [6]Paediatric Infectious Diseases, St. Mary's Hospital, London, UK, and [7]Imperial College Medical School, London, UK

Abstract
The published literature on bacterial tracheitis is limited. We report the first multi-centre study of bacterial tracheitis together with a concise review of the literature. We conducted a retrospective study of cases admitted during the period 1993–2007 to 3 tertiary paediatric centres in the United Kingdom and 1 in Australia. A total of 34 cases were identified. 31 patients (91%) required intubation. Complications included cardiorespiratory arrest in 1, ARDS in 1, hypotension in 10, toxic shock syndrome in 1 and renal failure in 1 patient(s). Staphylococcus aureus was the most commonly implicated bacterial organism, isolated from the respiratory tract in 55.8% of the cases overall. Other pathogens commonly isolated from the respiratory tract included Streptococcus pyogenes (5.9%), Streptococcus pneumoniae (11.8%) and Haemophilus influenzae (11.8%). Viral coinfection was identified in 9 (31%) of the 29 cases in whom immunofluorescence testing was performed (influenza A in 4 cases; parainfluenza 1 in 2 cases; parainfluenza 3 in 2 cases; adenovirus in 1 case). The combined experience from 4 major paediatric intensive care units suggests that bacterial tracheitis remains a rare condition with an estimated incidence of approximately 0.1/100,000 children per year. Short-term complications were common but long-term sequelae were rare. There were no fatal outcomes, which contrasts with the high historical mortality rates and likely reflects improvements in intensive care management.

Introduction

Upper respiratory tract infections (URTI) are among the most common infections in childhood. A variety of URTI are associated with airway inflammation resulting in oedema and potentially in upper airway obstruction. The clinical hallmark of these infections is stridor, which typically occurs in viral croup, epiglottitis and bacterial tracheitis.

The vast majority of children presenting to the emergency department with stridor suffer from viral croup, a generally benign condition [1]. In contrast, epiglottitis is now an uncommon condition in industrialized countries, as a result of the introduction of Haemophilus influenza type b vaccines into routine childhood vaccination programmes [1,2]. Bacterial tracheitis, also called bacterial croup or laryngotracheobronchitis, is a term that was first coined by Jones et al. in 1979 [3]. Bacterial tracheitis is a potentially fatal condition that primarily affects children [1,4]. The condition is frequently associated with rapidly worsening respiratory distress, potentially leading to complete airway obstruction and respiratory arrest. In contrast to viral croup, patients with bacterial tracheitis show little response to inhaled steroids or adrenaline [3,5,6]. Visualization of the airways reveals subglottic inflammation, oedema of the tracheal mucosa and copious purulent endotracheal secretions. The epiglottis generally appears unremarkable or only mildly inflamed, contrasting with the findings in bacterial epiglottitis.

Correspondence: N. Curtis, Department of Paediatrics, The University of Melbourne, Royal Children's Hospital Melbourne, Flemington Road, Parkville, Victoria 3052, Australia. Tel: +61 3 9345 4545. Fax: +61 3 9345 6667. E-mail: nigel.curtis@rch.org.au

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Bacterial tracheitis has traditionally been considered a rare condition, and to date only an estimated 400 cases have been described in the literature. However, no previous study has provided an estimate of its incidence. Most publications are case reports or very small case series. Only 9 reports described a series larger than 10 patients [6–14]. The 2 largest series originated from the same institution, with a considerable overlap between the cohorts described [8,11]. Notably, all previous studies on bacterial tracheitis were limited to a single study site.

A recent study from the US suggests that the epidemiology of life-threatening airway infections may be changing [10]. In this report bacterial tracheitis was the most common cause of acute infectious upper airway obstruction in children admitted to the intensive care unit. Alarmingly, a paediatric intensive care unit (PICU) in Northern Ireland has also recently reported a considerable rise in the number of cases of bacterial tracheitis [12].

We present the results of the first multi-centre study on bacterial tracheitis. By analysing the collective experience from 4 large PICUs in 2 countries spanning more than a decade, we provide a considerable addition to the existing data on this condition. In addition, we present a concise review of the previously published literature on bacterial tracheitis.

Methods and material

Setting

Four large paediatric regional referral centres, each with its own PICU, participated in this retrospective study. Three of the centres are located in England (UK) – St. Mary's Hospital (SMH: now Imperial College Healthcare Trust), St. George's Hospital (SGH) and the Royal Liverpool Children's Hospital Alder Hey (AHH). SMH and SGH are located in the urban area of London. AHH jointly provides intensive care with another unit of a similar size for the paediatric population in the North-West of the country (estimated population 1.475 million children aged 0–16 years in 2001 [15]). The fourth centre, the Royal Children's Hospital Melbourne (RCH), incorporates one of the busiest PICUs in Australia, providing intensive care for the majority of children living in the state of Victoria (estimated population 1.190 million children aged 0–18 years in 2007 [16]).

Case definition

In the absence of universally accepted diagnostic criteria for bacterial tracheitis, we used a modified version of the criteria proposed by Eckel et al. [14] as the case definition: 1) presence of stridor; 2) presence of respiratory distress; 3) lack of or minimal response to inhaled corticosteroid or adrenaline treatment if administered; and 4) presence of purulent secretions, inflammation of the trachea and subglottic narrowing with normal appearance of the epiglottis on direct laryngoscopy or bronchoscopy. Exclusion criteria comprised pre-existing, chronic airway problems, tracheostomy, and suspected or confirmed foreign body inhalation.

Data collection

In the UK, potential cases of bacterial tracheitis were identified from prospectively maintained PICU databases, which covered variable time periods as follows: SMH: 1993–2007; SGH: 2000–2007; AHH: 1997–2007. In Australia (RCH), cases were identified from the hospital diagnostic coding system covering the years 1993–2007. The hospital records of potential cases were systematically evaluated by the authors. Information about the clinical features, investigations and treatment were collected by use of a standardized data collection sheet.

Microbiological analysis

Material for microbiological analysis was obtained via endotracheal suction (in intubated patients) or via throat swabs (non-intubated patients). Nasopharyngeal aspirates were used for virological testing. Samples from the respiratory tract were subjected to bacterial culture and direct immunofluorescence tests (IFT) for influenza A and B, parainfluenza 1, 2 and 3, adenovirus and respiratory syncytial virus.

Statistical analysis

Data were analysed with SPSS, v16.0 (SPSS Inc., Chicago, IL) and Prism, v5.0 (GraphPad, San Diego, CA). Groups were compared using an unpaired, two-tailed *t*-test statistic with Welch's correction. A two-sided Fisher's exact test was used to compare microbiological results from the UK with those from the Australian centre. A two-way ANOVA was used to analyse the trend of microbiological results over time. Statistical significance was defined as $p < 0.05$ at the 95% confidence level.

Results

A total of 34 patients (RCH, 14; SMH, 10; AHH, 7; SGH, 3) fulfilled the inclusion criteria. The mean age was 5.2 y (range 0.6 – 15.2 y). Potentially predisposing conditions were identified in 3 patients: 1 case

each of human immunodeficiency virus (HIV) infection, hypogammaglobulinaemia and diabetes mellitus type 1. In both countries the majority of cases (UK, 80.0%; Australia, 78.6%) occurred during their autumn and winter months, respectively (Figure 1).

Clinical features and laboratory parameters at the time of presentation to hospital are summarized in Table I. All patients had prodromal symptoms suggestive of an URTI (i.e. coryza, sore throat, cough, pyrexia) for more than 24 h prior to the onset of an acute deterioration. In 30 patients (88.2%) the stridor started less than 24 h prior to presentation; only in 1 case did the stridor start more than 2 d prior to presentation. All but 1 patient (97.1%) had acute onset of dyspnoea less than 24 h prior to presentation.

The majority of patients had only low- to medium-grade pyrexia at presentation. Notably, 4 patients were apyrexial (defined as temperature $\leq$ 37.5°C); only 3 had high-grade pyrexia (defined as temperature $\geq$39.0°C). In 14 cases (41.2%) the initial CRP was less than 40 mg/l. In 11 patients the initial chest X-ray was reported as unremarkable. Tracheal narrowing was noted only in 3 (8.8%) cases (Table I).

A total of 31 patients (91.2%) required intubation: 28 were mechanically ventilated, and the other 3 remained self-ventilated while receiving supplemental oxygen. In all but 4 of these patients (87.1%) intubation became necessary within the first 24 h after presentation to the hospital. None of the patients underwent tracheostomy.

Two patients had to be commenced on high-frequency oscillation (HFO), as conventional ventilation failed to achieve adequate oxygenation. One of these patients had chest X-ray changes suggestive of coexisting bilateral upper lobe consolidation; the X-ray of the other patient was unremarkable prior to intubation. In both patients Staphylococcus aureus was isolated from the endotracheal secretions; one of these cases was coinfected with adenovirus. The latter developed acute respiratory distress syndrome (ARDS), with bilateral ground-glass appearance on chest X-ray, after 24 h. In both patients no predisposing conditions were identified.

10 patients were hypotensive on admission or developed hypotension within the first 24 h of admission. Five responded to fluid boluses alone (normal saline and/or albumin), and 5 required treatment with inotropes. None of these patients required inotropic support for more than 48 h; this included 1 patient with S. aureus infection who developed toxic shock syndrome and acute renal failure.

A total of 30 patients received systemic corticosteroids: 26 dexamethasone, 3 prednisolone, 3 hydrocortisone and 2 methylprednisolone (4 patients sequentially received 2 different corticosteroids). The mean duration of corticosteroid treatment was 3.3 d (range 1–7 d). There was no statistically significant difference between patients who received systemic corticosteroids and those who did not, regarding the duration of intubation ($p = 0.49$) and the duration of hospital admission ($p = 0.10$).

The majority of patients required only a relatively short period of assisted ventilation. The mean duration of ventilation was 3.5 d (range 1–12 d); 17 cases (55%) were extubated within 72 h.

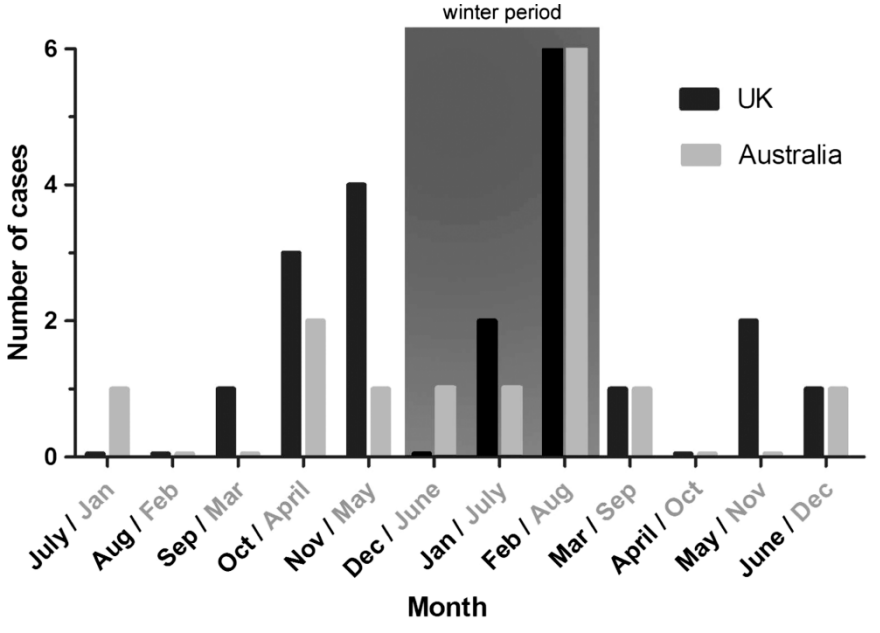

Figure 1. Seasonal incidence of bacterial tracheitis in comparison between the UK centres and the Australian centre.

Table I. Clinical, laboratory and radiological features of patients with bacterial tracheitis ($n=34$) at presentation.

Clinical features		
Pyrexia[a] (no. (percentage))	30 (88.2%)	
Temperature (mean (range))	38.3 (37.4–39.6)°C	
	Number (%) of patients with clinical feature	Mean (range) duration of clinical feature
Cough	28 (82.4%)	2.4 (1–5) d(s)
Recessions	30 (88.2%)	<1 (<1–3) d(s)
Hoarseness	13 (38.2%)	<1 (<1–5) d(s)
Aphonia	8 (23.5%)	<1 (<1–5) d(s)
Laboratory parameters	Mean	Range
C-reactive protein	82 mg/l	6–245 mg/l
White blood cell count	10.5×10^9/l	$1.8–23.9 \times 10^9$/l
Neutrophil count	7.9×10^9/l	$1.5–23.3 \times 10^9$/l
Chest X-ray findings[b]	Number (%) of patients with radiological feature	
No abnormalities reported	11 (32.4%)	
Pulmonary consolidation/ collapse	19 (55.9%)	
Tracheal narrowing	3 (8.8%)	
Pneumomediastinum	1 (2.9%)	

[a]Defined as temperature >37.5°C.
[b]Intubation prior to radiological examination may have obscured tracheal narrowing.

The mean duration of hospital admission was 7.1 d (range 3–13 d). This excludes 1 patient who spent more than 3 months in the hospital while undergoing rehabilitation. This patient had a cardiorespiratory arrest after presentation to a local hospital with subsequent hypoxic brain injury. The long-term sequelae in this case included epileptic seizures and neurodevelopmental delay. None of the remaining 33 patients had any long-term complications related to bacterial tracheitis and there were no fatalities.

There were no significant differences between the UK centres and the Australian centre regarding the pattern of organisms isolated. S. aureus was the most common causative organism in both the UK (61.9% of positive bacterial cultures) and Australia (54.5%) ($p=0.72$) (Table II). There was no significant variation in the proportion of cases caused by S. aureus over the study period ($p=0.18$) (Figure 2).

Only 1 of the staphylococcal isolates was found to be a methicillin-resistant S. aureus (MRSA). The organism was isolated from a 4-year-old HIV-infected boy who presented with stridor and severe respiratory distress requiring intubation. He was started on empirical treatment with flucloxacillin, cefotaxime and gentamicin, which was changed to vancomycin once susceptibility results became available. He required ventilatory support for 4 d and made an uneventful recovery leading to discharge after a total of 13 d.

The 2 patients in whom Haemophilus influenzae type b was isolated had been immunized against this organism, and therefore represent vaccination failures. In 3 patients, all of whom had received more than 24 h of antibiotic treatment before the endotracheal samples were obtained, both blood and respiratory cultures were sterile.

Of the 29 patients (85.3%) who had IFT for viruses on respiratory tract samples, 9 (31.0%) had positive results, comprising influenza A virus in 4 (13.8%), parainfluenza virus 1 in 2 (6.9%), parainfluenza virus 3 in 2 (6.9%) and adenovirus in 1 case(s) (3.5%).

The majority of patients received a combination of antibiotics; only 8 patients received monotherapy, most commonly a third generation cephalosporin. The most common choices were third generation cephalosporins ($n=19$) and flucloxacillin ($n=18$). The mean (range) duration of antibiotic treatment was 11.0 (7.0–19.0) d. None of the patients experienced a relapse or re-presented to hospital during the first month after discharge.

Discussion

This is the first multi-centre study on bacterial tracheitis and one of the largest series published to

Table II. Microbiological results of samples obtained in the first 48 h after admission.

	Isolates from endotracheal aspirates, number (percentage)[a,b]	Isolates from blood cultures, number (percentage)[c]
Staphylococcus aureus	19 (55.9%)[d]	1 (3.3%)[e]
Streptococcus pneumoniae	4 (11.8%)	1 (3.3%)[e]
Streptococcus pyogenes	2 (5.9%)	0
Haemophilus influenzae (non-typeable)	3 (8.8%)	0
Haemophilus influenzae type B	1 (2.9%)[f]	1 (3.3%)[f]
Pseudomonas aeruginosa	1 (2.9%)	0
Moraxella catarrhalis	1 (2.9%)	0

Upper respiratory tract flora (likely contaminants) cultured from endotracheal aspirate in 4 patients; in 3 patients, who had received antibiotics prior to specimen collection, cultures were sterile.
[a]Percentage calculated in relation to the total number of patients in whom aspirates were cultured ($n=34$).
[b]In 4 patients 2 different bacteria were simultaneously isolated from the ET secretions.
[c]Percentage calculated in relation to the total number of patients in whom blood cultures were performed ($n=30$).
[d]One methicillin-resistant strain (MRSA).
[e]Both bacteria grown simultaneously from the blood cultures of the same patient.
[f]H. influenzae type b was isolated from 2 different patients.

date. The small number of cases recorded over a period of more than a decade in 4 major paediatric intensive care units suggests that bacterial tracheitis remains uncommon. The incidence for the centres in London cannot be reliably calculated, as there is considerable overlap in the populations served by the 2 participating centres and other hospitals in the region. In contrast, the relatively well-defined population served by the other 2 participating centres enables an approximate estimate of the incidence of bacterial tracheitis. In the North-West of England the estimated incidence was 0.09/100,000 children per year. In Victoria (Australia) the incidence was remarkably similar with an estimate of 0.08/100,000 children per year.

Table III summarizes all publications that have reported more than 10 cases of bacterial tracheitis. It is worth noting that the largest study, reported by Salamone et al. [11], is unusual in a number of respects. First, the large number of cases identified during a 10-year period contrasts with the experience in other major paediatric centres. Secondly, only a relatively small proportion of the cases had symptoms typical of bacterial tracheitis, such as stridor (54%) and recessions (30%), while only 6% were described as having severe respiratory distress. Finally, a causative organism was identified in only

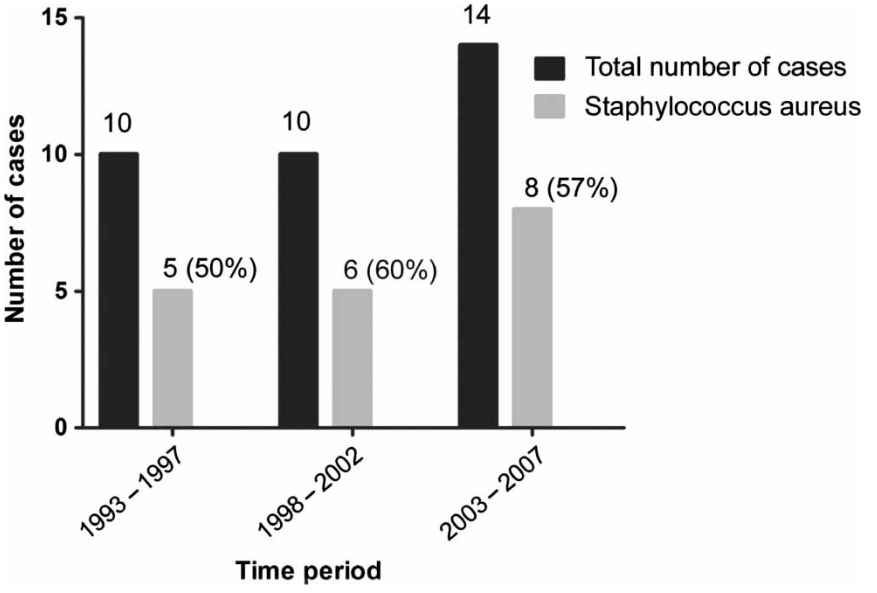

Figure 2. Total number of cases of bacterial tracheitis and proportions caused by Staphylococcus aureus over the study period.

Table III. Characteristics of cases with bacterial tracheitis in major previous studies and the current report.

	Number of cases	Mean age	Number intubated (percentage)	Mean duration of intubation	Mean duration of admission	Complications, number (percentage)	Number of deaths (percentage)	Bacteria isolated[b], number (percentage)	Viruses identified[c], number
Liston et al., 1983	17	30 mo (11–108 mo)	14 (82.4%) 9 tracheostomy	n.d.	n.d.	Cardiorespiratory arrest (4) Pneumothorax (2)	1 (5.9%)	S. aureus: 6 S. pneumoniae: 1 S. pyogenes: 1 H. influenzae: 2 E. coli: 4	Parainfluenza virus: 6
Kasian et al., 1989	14	39 mo (7–123 mo)	13 (92.8%)	182 h	12.0 days	Toxic shock syndrome (1) Pneumothorax (1) Subglottic stenosis (1)	3 (21.4%)	S. aureus: 7 S. pneumoniae: 1 H. influenzae: 4 M. catarrhalis: 2	Parainfluenza virus: 1 RSV: 1
Eckel et al., 1993	11	61 mo (9 – 138 mo)	10 (90.9%)	n.d.	n.d.	ARDS (1)	0	S. aureus: 6 S. pyogenes: 1 E. coli: 1 P. aeruginosa: 2	n. d.
Danis et al., 2004	17	n.d. (7–96 mo)	17 (100%)	n.d.	n.d.	n.d.	0	S. aureus: 1 S. pneumoniae: 2 S. pyogenes: 1 H. influenzae: 1 M. catarrhalis: 2	Influenza virus: 1 Parainfluenza virus: 2 Rhinovirus: 1 Picornavirus: 1
Salamone et al., 2004[a]	94	95 mo (11–186 mo)	50 (53.2%)	n.d.	5.3 days	None	0	S. aureus: 29 S. pneumoniae: 15 S. pyogenes: 8 H. influenzae: 15 M. catarrhalis: 13 P. aeruginosa: 1	Influenza virus: 31 RSV: 3
Marcos-Alonso et al., 2005	12	24 mo (1–156 mo)	12 (100%)	48 h	n.d.	Hypotension (2) Congestive heart failure (1)	0	S. aureus: 3 S. pneumoniae: 1 S. pyogenes:1 H. influenzae: 2	n. d.
Hopkins et al., 2006	18	67 mo (8–168 mo)	15 (83.3%)	180 h	11.0 days	ARDS (4) Pulmonary oedema (1) Subglottic stenosis (1)	1 (5.6%)	S. aureus: 6 S. pneumoniae: 3 S. pyogenes: 1 H. influenzae: 1 M. catarrhalis: 1	Influenza virus: 5 RSV: 1
Present study	34	62 mo (7–182 mo)	31 (91.2%)	84 h	7.1 days	Cardiorespiratory arrest (1) ARDS (1) Hypotension (10) Toxic shock syndrome (1)	0	S. aureus: 19 S. pneumoniae: 4 S. pyogenes: 2 H. influenzae: 4 M. catarrhalis: 1 P. aeruginosa: 1	Influenza virus: 4 Parainfluenza virus: 4 Adenovirus: 1

Table III (*Continued*)

	Number of cases	Mean age	Number intubated (percentage)	Mean duration of intubation	Mean duration of admission	Complications, number (percentage)	Number of deaths (percentage)	Bacteria isolated[b], number (percentage)	Viruses identified[c], number
Total (percentage)	217	Range: 1–186 mo	162 (74.7%)	Range: 48–182 hrs	Range: 5–12 d	Hypotension: 12 (5.5%) Cardiorespiratory arrest: 5 (2.3%) ARDS: 6 (2.8%) Pneumothorax: 3 (1.4%) TSS: 2 (0.9%) Subglottic stenosis: 2 (0.9%) Congestive heart failure: 1 (0.5%) Pulmonary oedema: 1 (0.5%)	5 (2.3%)	S. aureus: 77 (43.8%) S. pneumoniae: 27 (15.3%) S. pyogenes: 15 (8.5%) H. influenzae: 29 (16.5%) M. catarrhalis: 19 (10.8%) E. coli: 5 (2.8%) P. aeruginosa: 4 (2.3%)	Influenza virus: 41 Parainfluenza virus: 13 RSV: 5 Adenovirus: 1 Picornavirus: 1 Rhinovirus: 1

All reports describing more than 10 patients with bacterial tracheitis were included, with the exception of the report by Devlin et al. [12], which contained insufficient detail.h: hours; n.d.: not done or not described; mo: months; RSV: respiratory syncytial virus; TSS: toxic shock syndrome.

[a]The experience described by Bernstein et al. [8] falls into the longer time period described by Salamone et al. [11], reporting the data from the same institution. The former report was consequently excluded.

[b]Bacterial organisms likely to be non-pathogenic that were described in the original reports are not included (e.g. coagulase-negative staphylococci, viridans streptococci).

[c]Viral studies were not carried out universally in all patients in any of the studies. The actual numbers of viral coinfections may be considerably higher. Therefore, percentages were not calculated.

about two-thirds of the patients. As many patients were uncharacteristic of 'classic' bacterial tracheitis, the authors proposed the term 'exudative tracheitis', which may represent a milder form of the disease.

Consistent with previous studies, the majority of our patients had no known pre-existing conditions that would have predisposed to severe bacterial infections [5] and the incidence peaked during the colder winter months [6,11,17]. This is the first report to document this pattern in the Southern hemisphere.

In our series S. aureus was the most common causative organism, in accordance with the majority of previous reports in which this organism accounted for 36% to 75% of cases [3,5,6,9–11,17–21]. As in our study, group A streptococci, pneumococci and H. influenzae played a significant role in almost all previous series [3,5–11,17–22]. Other publications have stressed the importance of Moraxella catarrhalis as a causative organism in bacterial tracheitis [11,23–26], but we only observed a single case associated with this organism. Other bacteria implicated in bacterial tracheitis from case reports include group B streptococci [27], corynebacteria [28,29], Escherichia coli [7], Bacillus cereus [30] and Pseudomonas aeruginosa [11,31].

Based on the microbiological data from our study and previous reports (Table III), a combination of an anti-staphylococcal agent (e.g. flucloxacillin) and a third generation cephalosporin (e.g. ceftriaxone or cefotaxime) is a reasonable empirical choice of treatment for suspected bacterial tracheitis. However, in areas where community acquired methicillin-resistant S. aureus (MRSA) are common, flucloxacillin might have to be replaced by vancomycin as a first-line agent for empirical therapy.

It is noteworthy that all of our patients had prodromal symptoms suggestive of a minor URTI for at least 24 h, an observation frequently reported in bacterial tracheitis [6,11,17,32]. This was generally followed by a period of rapid deterioration, with almost all cases having experienced dyspnoea and stridor for less than 1 d. The rapid progression of bacterial tracheitis is also evident from the fact that almost all patients who required intubation did so within a few hours after presentation.

In this report, as well as in most previous publications, the peak incidence of bacterial tracheitis coincided with the seasonal peak incidence for viral URTIs. Interestingly, Bernstein et al. reported a large number of cases ($n = 46$) of bacterial tracheitis that occurred during an influenza epidemic over a single year [8]. In their cohort, influenza A virus was detected in 72% of the patients who had viral cultures.

Only about one-third of the patients in our study were found to be coinfected with a viral pathogen. However, the IFT panels used were relatively limited and did not include a variety of other viruses that commonly cause respiratory infections in children and might play a contributory role, such as the more recently discovered metapneumoviruses and bocaviruses [33,34].

Based on these observations it is probable that a large proportion of patients initially suffer from a viral URTI, which is followed by secondary bacterial superinfection. We hypothesize that viruses initially disrupt the mucosal integrity and local defence mechanisms, allowing bacteria to invade the mucosa and rapidly spread in local tissues. Experimental data from animal models support this theory further [35–37].

In a substantial proportion (41%) of our patients, the CRP at presentation was less than 40 mg/dl, suggesting that this test is not useful for ruling out bacterial tracheitis. Similar observations were reported by Eckel et al., who described that 7 out of 11 patients in their series had an unremarkable CRP at the time of presentation [14]. In addition, we found that chest X-rays at presentation were of relatively poor diagnostic value. This is supported by a number of previous studies, which also described that tracheal narrowing is seen in only a relatively small proportion of patients, unless specific airway films are obtained [3,5,14]. Lateral neck X-rays may produce better yields [11,38], but can potentially delay appropriate management. As in previous reports, a significant number of patients in our cohort had coexisting pulmonary infiltrates [3,5,14,32].

Traditionally, the management of bacterial tracheitis has included treatment with corticosteroids, based on the rationale that this reduces airway inflammation and oedema. We did not find a significant difference in the duration of intubation or hospital stay between the group of patients that received corticosteroids and the group that did not. However, statistically significant differences were unlikely to be detected given the low number of patients in the latter group. In the absence of definitive data, we continue to use systemic corticosteroids routinely in the initial phase of the illness, based on the belief that this is more likely to be of benefit than harm.

As illustrated in our study, most patients with bacterial tracheitis require intubation and a relatively short period of mechanical ventilation, commonly between 2 and 4 d [5,9,21]. Following initial intubation, which overcomes airway obstruction, patients often improve dramatically. This situation may, however, change when the lung parenchyma becomes involved in the infectious process or

ARDS develops, as was observed in one of our patients who required HFO.

The potential limitations of this study include its retrospective nature. However, it is unlikely that a prospective study of bacterial tracheitis will be conducted in the future, given the low incidence of this condition. In the participating centres in this study, which are among the largest PICUs in their respective countries, an average of only 1–2 cases were encountered per year. Also, as cases in the UK were identified from PICU databases, less severe cases may not have been identified, potentially leading to an underestimate of the true incidence. We also did not actively search for cases who died prior to presentation to the hospital. However, the Office for National Statistics recorded only a single paediatric death due to acute tracheitis in England and Wales during the most recent available 5-year-period (2003–2007) [39].

Active intervention and improvements in PICU care have undoubtedly contributed to lowering the mortality rate in patients with bacterial tracheitis, which historically has been reported to be as high as 6% to 40% [4,6,7,40]. Our study shows that in the modern PICU setting, bacterial tracheitis is now generally associated with a good prognosis and low rates of long-term morbidity.

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