insufficient in assessing patients with CF for lower airway infection. Even when BALs are taken from the RML and lingual, a significant number of infections are missed.

Is it asthma or not?

S145 HIGH CONCENTRATION OXYGEN CAUSES CARBON DIOXIDE RETENTION IN SEVERE ASTHMA: A RANDOMISED CONTROLLED TRIAL

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Introduction and Objectives The use of high concentration oxygen in acute exacerbations of chronic obstructive pulmonary disease (COPD) is well known to result in an increase in $PaCO_2$ in some patients. High concentration oxygen is often used routinely in acute severe asthma in the belief that it is safe and indicated in most patients; however, there is some evidence to suggest that this causes an increase in $PaCO_2$. In this randomised controlled trial we compared the effects of high flow vs titrated oxygen therapy on $PaCO_2$ levels in acute severe asthma.

Methods 80 patients with severe exacerbations of asthma (forced expiratory volume in 1 s (FEV_1) $\leqslant$50% predicted) presenting to the Emergency Department of Wellington Hospital, New Zealand were recruited. Participants were randomised to receive either high flow oxygen (8 l/min via a medium concentration mask) or titrated oxygen (via nasal prongs or a medium concentration mask) adjusted to achieve oxygen saturations of 93–95% for 1 h along with routine asthma treatment. Transcutaneous carbon dioxide measurements ($PtCO_2$) were made at 0 and 60 min. The primary outcome variable was the proportion of patients with a rise in $PtCO_2$ $\geqslant$4 mm Hg at 60 min. The secondary outcome variables were: the proportion of patients with a rise in $PtCO_2$ $\geqslant$4 mm Hg and a $PtCO_2$ $\geqslant$40 mm Hg at 60 min, the proportion of patients with a rise in $PtCO_2$ $\geqslant$8 mm Hg and the mean rise in CO_2.

Results Three subjects withdrew from the high flow group leaving 36 for analysis in the high flow group and 41 in the titrated group. The mean (SD) FEV_1 % predicted was 33.4% (10.5) in the high flow group and 35.4% (9.7) in the titrated group ($p = 0.35$). Results for the primary and secondary outcome measures are presented in table 1.

Conclusion These results show that uncontrolled high concentration oxygen therapy results in an increase in $PtCO_2$ when administered to patients with severe exacerbations of asthma. We propose that in severe asthma oxygen should only be used if hypoxaemia is present, and delivery should be titrated to achieve oxygen saturations within the normal range.

S146 AIRWAYS DYSFUNCTION AND EOSINOPHILIC INFLAMMATION IN ELITE ATHLETES WITH SYMPTOMS SUGGESTING EXERCISE-INDUCED ASTHMA

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Introduction and Objectives Symptoms suggesting exercise-induced asthma are common in athletes, particularly those participating in endurance sports. Increased use of asthma medications at elite level has led governing bodies to introduce legislation that requires proof of airways dysfunction prior to use of medication. The current gold standard is a drop in forced expiratory volume in 1 s (FEV_1) of 10% following the eucapnic voluntary hyperventilation (EVH) test. However, there remains controversy as to what the optimum criteria for a positive test should be and whether this test captures all domains of asthma. In order to explore the relationship between airway dysfunction and potentially steroid-responsive disease, we compared the response to EVH with markers of airway inflammation in a group of 30 international athletes who reported symptoms suggesting exercise-induced asthma.

Methods Inhaled steroids and long-acting β-agonists were withheld for at least 2 weeks prior to assessment, and short-acting β-agonists, caffeine and exercise for >8 h. Exhaled nitric oxide (FE_{NO}) was assessed using the NIOX Mino prior to spirometry and EVH challenge. Sputum induction was done after recovery and following pretreatment with inhaled salbutamol.

Results There was a significant correlation between the percentage fall in FEV_1 after EVH challenge and the sputum eosinophil count ($r = 0.46$, $p = 0.01$). A fall of 10% was a sensitive (100%) but not specific (45.5%) indicator of eosinophilic airway disease (defined as a sputum eosinophil count of >3%); a drop of 24% was a more valid marker (sensitivity 88%, specificity 91%). There was close correlation between FE_{NO} and the sputum eosinophil count ($r = 0.901$, $p<0.0001$), suggesting that FE_{NO} might be a simpler and more valid marker of eosinophilic, corticosteroid-responsive airway inflammation in elite athletes.

Conclusions We conclude that the current criteria for a positive EVH test identify significant numbers of athletes who do not have corticosteroid-responsive airway pathology. Either much greater falls in FEV_1 or alternative means are required to identify this dimension of the disease.

S147 HYPERTONIC SALINE CHALLENGE IN THE DIAGNOSIS OF VOCAL CORD DYSFUNCTION IN PATIENTS WITH ASTHMA ATTENDING A SECONDARY CARE CLINIC

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Introduction Symptoms of vocal cord dysfunction (VCD) are caused predominantly by vocal cord adduction during inspiration. It is

Abstract S145 Table 1

	High flow O_2	Titrated O_2	Relative risk	p Value
Subjects with a rise in $PtCO_2$ $\geqslant$4 mm Hg	15/36 (41.7%)	6/41 (14.6%)	2.8 (CI 1.2 to 6.6)	0.008
Subjects with a rise in $PtCO_2$ $\geqslant$4 mm Hg and $PtCO_2$ $\geqslant$40 mm Hg at 60 min	7/36 (19.4%)	1/41 (2.4%)	8.0 (CI 1.0 to 61.7)	0.022
Subjects with a rise in $PtCO_2$ $\geqslant$8 mm Hg	5/36 (13.9%)	3/41 (7.3%)	1.9 (CI 0.5 to 7.4)	0.35
	High flow O_2	Titrated O_2	$PtCO_2$ difference	p Value
Mean rise in $PtCO_2$	2.6 mm Hg (SD 4.2)	0.5 mm Hg (SD 4.4)	2.0 mm Hg (95% CI 0.08 to 4.0)	0.042

$PtCO_2$, transcutaneous CO_2.

Abstract S147 Table 1 VAS before and after HS challenge (score scale 0–100)

		Median (IQR)							
		Healthy		Asthma		VCD		ANOVA p value	
VAS question		Baseline	8 min	Baseline	8 min	Baseline	8 min	Baseline	8 min (Δ)
1	I feel I can't get enough breath in	0 (0)	0 (2)	1 (3)	1 (18)	29 (53)	53 (39)	0.001	0.019
2	I feel like I am being strangled	0 (0)	0 (0)	0 (1)	0 (6)	22 (52)	49 (59)	0.001	0.020
3	My chest feels tight	0 (0)	0 (2)	0 (1)	0 (51)	39 (62)	45 (47)	<0.001	0.355
4	My throat feels tight	0 (0)	0 (0)	0 (0)	0 (16)	49 (68)	64 (46)	<0.001	0.149
5	I have a wheeze	0 (0)	0 (0)	0 (0)	14 (26)	5 (28)	36 (62)	0.002	<0.001

ANOVA, analysis of variance; HS, hypertonic saline; IQR, interquartile range; VAS, visual analogue scale; VCD, vocal cord dysfunction

commonly seen in the asthma clinic, both where asthma has been misdiagnosed and as an additional diagnosis. The current "gold standard" for diagnosis of VCD is direct visualisation of the vocal cords during laryngoscopy. A flow volume loop recorded whilst symptomatic may also demonstrate an abnormal pattern, and VCD may be induced in susceptible subjects—for example, by hypertonic saline (HS) challenge. Taramarcaz and colleagues (J Allergy Clin Immunol. 2004) have reported a decrease in forced inspiratory flow (FIF_{50}) after HS challenge in a case series of three patients with postviral VCD.

Methods We designed a prospective, controlled study to determine whether HS challenge testing provokes VCD, as evidenced by changes in FIF_{50} and visual analogue scores (VAS). Baseline questionnaires (Hospital Anxiety Depression Score (HAD), adapted John Hunter Cough Questionnaire (aJHCQ) and a VCD VAS) were completed, then HS challenge performed, with a shortened VAS and spirometry recorded after each dose interval.

Results Twenty-seven subjects (mean (range) age 38 (21–62) years, 38% male) completed the study, seven subjects with VCD (6 with a previous diagnosis of asthma), six physician-confirmed asthma alone and 14 healthy controls (HCs). Subjects with VCD were older, with higher HAD, baseline VAS and aJHCQ scores than the other groups. As expected, HS induced a greater fall in forced expiratory volume in 1 s (FEV_1) in the group with asthma than in the VCD or HC groups. However, change in FIF_{50} after the final dose of HS did not significantly differ between groups. The change in mean VAS score, and particularly items 1 and 2, discriminated between VCD and the other groups during HS challenge (table 1).

Conclusion FIF_{50} may not be a useful dynamic marker of VCD during HS challenge, but our modified VCD VAS did show symptom changes in subjects with VCD compared with those with asthma and healthy controls. This tool could potentially be used in the diagnosis and treatment of VCD after further evaluation and validation.

S148 DIAGNOSIS OF VOCAL CORD DYSFUNCTION IN ASTHMA WITH HIGH RESOLUTION DYNAMIC VOLUME CT OF THE LARYNX

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Background Vocal cord dysfunction (VCD) often masquerades as asthma, and previous studies have suggested that up to 30% of patients with asthma have upper airway dysfunction consistent with a diagnosis of VCD. Diagnosis of VCD is difficult, in part because it involves laryngoscopy which has practical constraints, and there is need for rapid non-invasive diagnosis.

Methods We used high resolution 320-slice dynamic volume CT to examine laryngeal function in patients with asthma with ongoing symptoms and suspected VCD. This new technology uses a 320-slice detector to scan a 16 cm Z-axis "volume" over time. It permits visualisation of the physical movement of an anatomical structure and provides imaging not achieved with traditional 64-slice helical scanners. Laryngeal luminal area was measured, and reductions >40% were judged to be consistent with a diagnosis of VCD.

Results A case series of 14 patients with a history of severe, symptomatic asthma and suspected VCD is presented. High speed 320-slice CT generated dynamic volume scans and provided accurate images of laryngeal function during inspiration and expiration. VCD could be assessed using static and real-time cine images combining volume-rendering reconstruction and virtual bronchoscopy techniques. Eight patients had clear evidence of VCD and the median reduction of luminal area during expiration was 75.2% compared with 12.7% in the six patients without VCD. Patients with VCD had no distinguishing clinical characteristics but tended to be obese females. Radiation doses were modest, in the range 1–3 mSv.

Conclusions Dynamic volume CT provided explicit images of the larynx, distinguished function of the vocal cords during the respiratory cycle and could identify VCD in a selected group of patients with asthma. With further refinement the technique will potentially provide a simple, non-invasive investigation to identify laryngeal dysfunction/VCD, permitting improved management of asthma.

S149 EXPOSURE TO OCCUPATIONAL AGENTS AND RISK OF ASTHMA IN THE 1958 BIRTH COHORT FROM AGE 16 TO AGE 33 YEARS

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Introduction Occupational exposures may cause adult-onset asthma. In this analysis we present the prevalence of exposure to asthmagenic agents and the associated risk of asthma by age 33 in participants in the 1958 birth cohort.

Methods All persons born in the first week of March in Britain in 1958 were recruited into the cohort. At age 33, 11 469 cohort members responded and of these 10 043 participants provided a full occupational history. Job descriptions were initially coded into Standard Occupational Classification 1990 using a text-based computer program. Blind to asthma status, we have recoded these jobs into the International Standard Classification of Occupations 1988 codes and applied an Asthma Specific Job Exposure Matrix,[1] including an expert judgement step. Exposure to 22 agents was

Abstract S149 Table 1

Type of workplace exposure	% ever exposed	OR (crude)	OR (adjusted)
HMW	21.1	1.02 (0.80 to 1.30)	0.95 (0.74 to 1.21)
LMW	24.2	0.99 (0.79 to 1.25)	1.12 (0.89 to 1.43)
Mixed environments	10.4	0.81 (0.57 to 1.15)	0.90 (0.63 to 1.28)
Any high risk	37.7	1.06 (0.87 to 1.30)	1.09 (0.88 to 1.33)
Peak irritant	4.1	0.53 (0.27 to 1.04)	0.77 (0.39 to 1.53)
ETS	11.7	1.59 (1.23 to 2.07)	1.44 (1.10 to 1.88)

ETS, environmental tobacco smoke; HMW, high molecular weight; LMW, low molecular weight.

assigned. Major groups were high (HMW) and low molecular weight (LMW) antigens, mixed environments, irritants and other exposures, including environmental tobacco smoke (ETS). The prevalence of ever being exposed to one of these agents was determined. Using logistic regression adjusted for sex, smoking, father's social class at birth and region. and after excluding those with ''asthma/wheezy bronchitis'' by age 16, we assessed the association of ''ever asthma'' at age 33 with these 22 exposures.

Results 38% of the cohort by age 33 had worked in a job identified as having a high risk for asthma. Excluding those with asthma/wheezy bronchitis by age 16, the prevalence of ''ever asthma'' at 33 was 4.8%. The prevalence of occupational exposure and the associated risk of asthma at 33 are shown in table 1. Reporting having worked in a job considered highly exposed to ETS was significantly associated with asthma. Nearly all jobs ascribed to this exposure were within the hospitality trade.

Conclusion In the period 1974–1991 over a third of those taking part in the 1958 cohort had been exposed to potentially high risk asthmagenic agents in the workplace. Working in jobs associated with high risk of exposure to ETS was associated with an increased risk of reporting asthma at age 33.

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