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Environmental temperature and relative humidity influence exhaled breath condensate pH

To the Editors:

Exhaled breath condensate (EBC) is used to obtain information about the composition of airway surface lining fluid [1]. Acidification of EBC has been reported in different inflammatory airway diseases [2]. Interpretation of data is compromised by the fact that condensate pH shows day-to-day variability, even in healthy subjects [3]. Coating of condensing surfaces [4] and salivary contamination of the condensate [5] have been proposed to influence exhaled biomarker levels. It was our aim to investigate whether environmental temperature and relative humidity could influence condensate pH and whether they could be responsible for the observed variability.

EBC was collected from 12 healthy subjects with an R-Tube condenser (Respiratory Research Inc., Charlottesville, VA, USA) before and after the forecasted arrival of three cold, humid and two warm, dry meteorological fronts. In summer, on three occasions the room temperature and relative humidity paralleled the outside conditions; in winter, on two occasions room temperature and relative humidity were maintained at 26°C and between 47–52%.

EBC pH was determined using the carbon dioxide (CO₂) standardisation method, as described previously [3]. Briefly, pH and CO₂ were measured using a blood gas analyser (ABL 520; Radiometer, Copenhagen, Denmark) five times consecutively after 1-s CO₂ load to the sample between each measurement. A pH–CO₂ plot was created from the results. The pH at 5.33 kPa CO₂ partial pressure was calculated using the logarithmic regression equation obtained from the plot.

The arrival of forecasted meteorological fronts caused a pronounced change both in temperature and relative humidity of ambient air (table 1). In summer, when the study room conditions paralleled the weather conditions outside, EBC pH decreased significantly with the humid, cold fronts and inversely increased with the dry, warm front. In winter, when room conditions were controlled, EBC pH was not affected significantly by either the humid, cold or the dry, warm fronts. The coefficient of variation of EBC pH was 3.0±1.3 under uncontrolled room conditions in summer and 1.8±0.9 under controlled temperature and relative humidity of the room in winter (p<0.02). There was no correlation between pH and volume of condensates.

The results have two possible readings, one clinical and the other pathophysiological. From a clinical point of view the results

TABLE 1 Effect of meteorological fronts on exhaled breath condensate (EBC) pH

Season	Front	Day	Temp. °C	RH	EBC pH	p-value
Summer	Humid	Before	29	53	6.11±0.18	0.008
		After	18	74	5.92±0.19	
	Humid	Before	30	38	5.96±0.20	0.030
		After	23	53	5.83±0.13	
	Dry	Before	23	40	5.88±0.20	0.015
		After	31	36	6.02±0.16	
Winter	Humid	Before	16	53	6.00±0.15	NS
		After	5	83	5.97±0.16	
	Dry	Before	1	55	5.98±0.24	NS
		After	12	42	5.95±0.14	

Temperature (temp.) and relative humidity (RH) were registered at the time of collections. Study room temperature and RH paralleled the outside conditions in summer and were kept at 26°C and between 47–52% in winter. EBC pH values are presented as mean±SD. NS: nonsignificant.

suggest that controlling room temperature and relative humidity should be part of standardisation of EBC collection. From a pathophysiological point of view the results are interesting as they may contribute to the explanation of catching a common cold. It is not clear why the microbes that are present continuously in the air and on the mucous membranes cause respiratory tract infections that are more likely in cold weather. Compared with the stability of blood pH, changes ≥0.5 may occur in the pH of EBC and probably, therefore, the airway surface lining fluid. A relative important change in pH may result in a decrease in the ciliary beat [6] or in the activity of immune cells. The deterioration in the function of the nonspecific immune barrier may allow the viruses to invade the tissues.

It is widely accepted that meteorological factors can trigger several respiratory diseases. Asthma attacks have been related to: the rupture of pollens during thunderstorms [7]; higher concentrations of sulphur dioxide, nitric oxide and carbon monoxide in the air; and lower temperature and sunshine hours [8]. Exacerbations of chronic obstructive pulmonary disease have been demonstrated to be more frequent in cold weather [9].

In summary, the results suggest that environmental temperature and relative humidity contribute to the variability of condensate pH.

T. Kullmann*, **I. Barta**, **B. Antus**, **M. Valyon[#]** and **I. Horváth[†]**

*Dept of Pathophysiology and [#]Clinical Laboratory, National Korányi Institute for TB and Pulmonology [†]Institute of Human Physiology and Clinical Experimental Research, Semmelweis University, Budapest, Hungary.

STATEMENT OF INTEREST

None declared.

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Isolated diastolic dysfunction of right ventricle: stress-induced pulmonary hypertension

To the Editors:

We read with great interest the article of HUEZ *et al.* [1]. Their results, which were mostly noninvasive, suggest that isolated longitudinal diastolic dysfunction of the right ventricle may be a sign of stress-induced (or latent) pulmonary hypertension. The aim of our letter is to confirm this observation with the help of our results based on invasive measurements.

In total, 58 patients (mean age 54±8 yrs, 48 female) were examined. These comprised 15 healthy subjects who had no signs or symptoms of heart disease and 43 consecutive patients suffering from connective tissue disease (CTD), of whom 38 had systemic sclerosis, two had systemic lupus erythematosus, two had mixed CTD and one had polymyositis. Patients in the latter group were referred to the University of Pécs (Pécs, Hungary) on suspicion of pulmonary artery hypertension (PAH). Patients with atrial fibrillation and severe mitral or tricuspid insufficiency were excluded from the study.

The local ethics committee approved the study. All subjects had given written informed consent prior to undergoing echocardiography and right heart catheterisation.

Echocardiography was performed using an Aloka ProSound 5500 ultrasound system (Aloka Co. Ltd, Tokyo, Japan). Left ventricular ejection fraction was measured by Simpson's method. Right ventricular end-diastolic diameter was obtained from standard parasternal long axis view using M-mode measurements. Transmitral flow was recorded from the apical four-chamber view. The peak of the early (E) diastolic

velocities was measured. Systolic pulmonary artery pressure (PAP) was estimated as a sum of the pressure difference across the tricuspid valve calculated using the modified Bernoulli equation and an estimate of mean right atrial pressure. Myocardial systolic, early (E') and late diastolic velocities were measured from apical four-chamber view at the lateral border of the mitral and tricuspid annulus using tissue Doppler imaging. The mitral E/E' ratio was calculated. Doppler measurements were obtained from at least three consecutive beats during end-expiratory apnoea.

All patients with CTD underwent right heart catheterisation. A Swan-Ganz catheter was introduced to a main pulmonary artery branch. If the resting mean PAP (P_{pa}) was <30 mmHg, a 3 min bench-fly physical stress test was performed using two 1-kg dumbbells. P_{pa} was measured at rest and at peak exercise.

Differences between groups were tested for significance using ANOVA. *Post hoc* tests were performed by Scheffe's method. Comparisons of nonparametric data were performed using Chi-squared tests. A p-value of <0.05 was considered significant.

Thirteen patients belonging to the CTD group had resting (P_{pa} : 37.9±10.9 mmHg) PAH diagnosed by right heart catheterisation. In six patients, normal PAP values were measured at rest (22.8±1.7 mmHg) while elevated values were measured at peak exertion (39.8±6.3 mmHg).

Considering these results, the CTD patients were divided into the following three groups: 24 patients without PAH, six patients with stress-induced PAH and 13 patients with resting