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Respiratory movement and pain thresholds in airway environmental sensitivity, asthma and COPD

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Summary
Introduction: Patients with "sensory hyperreactivity" (SHR) have airway environmental sensitivity, chronic cough and dyspnoea. Cough, chest discomfort and sense of difficulties getting air are some of the symptoms these patients seek medical attendance for. The patients have increased cough sensitivity to inhaled capsaicin, mediated by ion channel receptors on sensory nerves also known to react to pain stimuli. Whether a link exists between capsaicin airway sensitivity and pain sensitivity has not yet been evaluated. The aim was to investigate chest mobility, respiratory movement and pain sensitivity in SHR patients compared with patients with asthma, chronic obstructive pulmonary disease (COPD) and alleged healthy control subjects.
Methods: Thirty-five patients diagnosed with SHR, 19 with COPD, 32 with asthma and 28 control subjects were included. Chest expansion was measured with a measuring tape and thoracic and abdominal movement with light sensors. Pain sensitivity was assessed using a pressure algometer.
Results: Groups differed significantly in lung function, respiratory rate and pain sensitivity but also in chest expansion and abdominal breathing movement. In comparison with the control and asthma groups but not the COPD patients, SHR patients had an increased respiratory rate and reduced abdominal movement during deep breathing. All patient groups showed lower pain thresholds than the controls.
Conclusion: Patients with SHR have evident signs of dysfunctional breathing and appeared to be most similar to the COPD group except for lung function. Lower pain thresholds among the patients indicate a general up-regulation of the sensory nerve system.

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Introduction

Patients with airway symptoms induced by environmental irritants report problems with persistent coughing, chest discomfort and dyspnoea.[1,2] These symptoms mimic asthma, but asthma-specific tests are negative and lung function tests are within normal range. The patients have an increased cough reaction to inhaled capsaicin, a tasteless and odorless substance that stimulates sensory nerves and reflects sensory nerve reactivity.[3] Such airway symptoms are interpreted as airway sensory hyperreactivity (SHR). Cigarette smoke, car exhaust and perfumed products are some of the triggers for SHR symptoms.[1,2] In most cases, the patients could also be diagnosed with chronic cough[4,5] or cough hypersensitivity syndrome.[6,7] SHR affects more than 6% of the adult population in Sweden, mainly women, according to a population-based epidemiologic study[8] where SHR diagnosis was based on a validated questionnaire, the chemical sensitivity scale for sensory hyperreactivity (CSS-SHR), in combination with a standardized positive capsaicin inhalation provocation test.[9,10] This inhalation test has a good repeatability and ability to distinguish patients with SHR from healthy control subjects.[1,11]

In recent years, there has been a growing interest in the family of transient receptor potential (TRP) ion channels in airway diseases.[12,13] They are an important means for multiple organ systems to interact with their environment.[14] Because the transient receptor potential vanilloid subtype 1 (TRPV1), which is responsible for the increased capsaicin cough response in SHR, is also known to react to pain stimuli and mirror pain sensitivity,[15] we hypothesize that SHR patients may have a corresponding augmented sensitivity to pain.

Complains about dyspnoea, though normal lung function and uncharacteristic chest pain are common in SHR.[1,2] Discomfort, pain, or sense of trouble in the body or parts of the body, in this case the chest, could, through the sensory feedback to the brain, lead to increased tonus of the musculature. Heavy breathing, sense of dyspnoea and decreased chest mobility may lead to dysfunctional breathing and follow this vicious circle of pain, increased tonus, and even more pain.[16–18] Dysfunctional breathing as an explanation for similar symptoms has been suggested by Hagman et al[19,20] but a difference in comparison to SHR is that environmental irritants as trigger factors are not described. Whether patients with SHR have dysfunctional respiratory movement and/or impaired chest mobility in relation to the reported symptoms of chest tightness and dyspnoea has not yet been evaluated.

Respiratory movement dysfunction as a consequence of impaired chest mobility is shown in different respiratory and chest diseases, such as after thoracic surgical procedures,[21,22] in ankylosing spondylitis[23] and in patients with persistent asthma, who had musculoskeletal dysfunction and chronic pain that was independent of the severity of their disease.[24] In severe COPD the breathing pattern seemed to be dependent of the inspiratory muscle activity[25] and when studying the breathing pattern in such patients, using a respiratory inductive plethysmography, Tobin et al found high respiratory rate, short inspiratory time and an asynchrony between ribcage and abdominal movements.[26]

The aim of this study was to investigate chest mobility, respiratory movement, and sensitivity to pain in SHR patients in comparison to patients with well-defined asthma and COPD and to alleged healthy control subjects.

Methods

Subjects

The patients were consecutively selected from the asthma and allergy outpatient clinic at Sahlgrenska University Hospital in Gothenburg, Sweden during 2009 and 2010. Thirty-five patients clinically diagnosed with SHR, 19 with COPD and 32 with asthma participated in the study. Twenty-eight control subjects were included; they considered themselves healthy, reported no airway symptoms and did not use airway-related drugs. No further physical examination was assessed. The control subjects were recruited from hospital workers, friends and relatives.

The SHR patients all had pronounced airway symptoms induced by environmental irritants and a positive capsaicin inhalation cough test, performed in accordance with several earlier studies.[8,10] The capsaicin test was performed with an interval of at least six weeks after any respiratory tract infection. All but one of the patients with SHR had also complained of daily or weekly problems with coughing for more than two months and could additionally have been diagnosed with chronic cough.[4,5] They further had a negative skin prick test (SPT) that tested ten of the most common respiratory allergens in Sweden, a negative methacholine test in accordance with international guidelines[27,28] and no signs of spirometric reversibility or variability in lung function.

The patients with asthma and COPD were diagnosed according to international guidelines.[29,30] Seventeen asthma patients used a combination of inhaled corticoid steroids and long acting β_2 agonists (ICS + LABA), 8 used ICS and LABA separately, 6 used ICS and all 32 asthma patients used short acting β_2 agonists, when needed. Twenty of the 32 asthma patients had previously shown a positive SPT to at least one allergen and all had a positive methacholine test.[27,28] Amongst the 19 COPD patients 16 regularly used ICS, 15 long acting anti cholinergic (LAAC) and 18 short acting β_2 agonists when needed. All COPD patients were former smoker.

Patients diagnosed with SHR, asthma or COPD considered themselves to have a habitual condition, medicating according to instructions from their physicians and with no ongoing airway infection or any change in the use of medication from four weeks prior to study participation. There were no changes in use of airway-related medications before the tests. The participants reported no symptoms of gastro-oesophageal reflux and were not allowed to take any medication for such symptoms. Use of angiotensin-converting enzyme inhibitors, diseases or injuries affecting the thoracic range of motion, pregnancy and breastfeeding were exclusion criteria. Active smokers were not included.

Informed consent was obtained from all participants at the start of the investigation. The Regional Ethical Review Board of Gothenburg approved the study.