SUBCLINICAL PULMONARY OBSTRUCTION AND VENTRICULAR ARRHYTHMOGENESIS

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PURPOSE: Impairments in forced expiratory volumes uniquely predispose patients with chronic obstructive pulmonary disease (COPD) to life-threatening cardiovascular events, including ventricular tachyarrhythmias and sudden cardiac death. Whether milder subclinical impairment in the expiratory pulmonary airflow correlates with increased propensity to ventricular arrhythmogenesis (premature ventricular contractions [PVC] and ventricular tachycardia [VT]) has not been tested. This line of investigation is important because it could open an entirely novel approach to identifying patients at increased risk of ventricular tachycardias treatable by widely available anti-arrhythmic therapies.

METHODS: In order to avoid possible selection bias of studying patients clinically indicated for spirometry, we conducted a prospective single-center study in which we enrolled adult patients who presented for a routine outpatient interrogation of their existing implantable arrhythmia-detecting device (implantable loop recorder, pacemaker, or defibrillator) who had no known history of COPD. We asked the subjects to undergo portable spirometry (best of 3 attempts was used for determining FEV1 and FVC), administered research questionnaires, and extracted relevant variables from their device interrogations and from the electronic medical record. Exclusion criteria: implanted device less than 3 months or lack of recording ability of PVC or VT.

RESULTS: The studied patients (N = 54, 44% women, mean age 64.7 years) recorded FEV1 2.2 ± 0.8L (84 ± 22% predicted), FVC 2.6 ± 0.97L (78% ± 18% predicted), and FEV1/FVC 83.4%±10.0%. Subjects with subclinical decrease in FEV1/FVC < 80% had greater frequency of PVCs (157 vs 1,115 PVC/day; p = 0.007) than subjects with FEV1/FVC > 80%. When testing cut offs for FEV1/FVC of 85% we found consistent results (34 vs 655 PVC/day; p = 0.016). The number of patients with recorded clinically relevant VT was low, and statistically similar in and across the pulmonary function cohorts.

CONCLUSIONS: Our study is the first to show increased ventricular automaticity in patients with even mild subclinical decreases in expiratory pulmonary parameters as recorded by long-term implanted arrhythmia monitoring devices.

CLINICAL IMPLICATIONS: The previously shown predisposition of COPD patients towards ventricular arrhythmias may extend even to patients with subclinical decrease in FEV1/FVC, and subsequent studies should explore whether there exists an additive value of using spirometry variables in assessing the risk of sudden cardiac death due to VT.

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