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EDITORIAL

Concomitance of cardiovascular and pulmonary disorders: mutual bystanders or causal interactions?

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Article Polak et al., see p. 1055

In the present issue of the journal Polak et al. [1] report a study on the relationships between cardiovascular risk factors, a derived cardiovascular risk score, and spirometric parameters of lung function in 4104 participants of the Polish HAPIEE project [2]. As major risk factors, the presence of hypertension, diabetes, and hypercholesterolaemia was assessed, and the 10-year risk of fatal cardiovascular disease (SCORE risk) was calculated using an established algorithm for high-risk countries [3]. To circumvent potential problems caused by using predicted values, lung function was directly adjusted for age and height. After this adjustment, absolute values of forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) were higher in men than in women. The presence of obesity, hypertension, and diabetes was associated with a lower FVC in both sexes, whereas its relation to FEV_1 differed between men and women. The authors conclude that reduced spirometric lung function is associated with an increased risk of cardiovascular disease and that disorders such as obesity and diabetes play a role in mediating this association.

The study addresses relationships that are important for the estimation of cardiovascular risk, which is known to depend on comorbidities including metabolic disorders, as well as physical characteristics including lung function. These influencing factors are linked to each other within a complex network of mechanical and inflammatory pathways. To disentangle these pathways, large studies with comprehensive and reliable assessments are of great value. The strength of the discussed study is the fact that the data were obtained by trained personnel using standardised procedures and questionnaires in a large random urban population. Naturally, it shows the limitations of cross-sectional study designs, but the potential bias arising from the recruited population and the scoring system used is discussed thoroughly. Irrespective of the limitations, the authors' conclusions seem warranted and may even be clinically useful to identify an increased cardiovascular risk on the basis of measures that can be easily obtained, in particular from spirometry. Future analyses have to determine whether the use of such risk estimates significantly contributes to the individual health assessment. In any case, a markedly reduced lung function should increase the awareness of unrecognised or underestimated cardiovascular disorders.

Lung-heart interactions are a major topic of current research. Beyond associations with lung function impairment per se, the presence or absence of chronic obstructive pulmonary disease (COPD) appears to be a crucial determinant. Comorbidities are common in COPD; in particular, cardiovascular comorbidities are linked to a worse prognosis [4]. Correspondingly, both symptoms and exacerbations of COPD correlate with frequent comorbidities [5]. A reverse view is to consider COPD as a comorbidity deteriorating cardiovascular disease [6]. The question of which causal (especially inflammatory and mechanical) factors mediate the interactions between comorbidities, lung function, and COPD was not the topic of the present paper, but it naturally leads into this issue. Probably the association between COPD and atherosclerotic events is due to inflammatory processes [7]. This is supported by the finding of increased systemic levels of inflammatory cytokines [8], although the role of systemic inflammation in general seems questionable [9]. Regarding mechanical causes from lung hyperinflation and airway obstruction, the risk for right-heart disorders such as *Cor pulmonale* is well known,

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but recently also a reduced right-heart size (*Cor pulmonale parvus*) has been described [10]. In addition, patients with COPD show changes of the left-heart, including decreases in left ventricular volume and mass [11], which are clearly linked to lung mechanics [12]. The same is true for left ventricular wall stress [12] and diastolic filling [13], again underlining the role of mechanical factors.

Possibly, it is worthwhile considering whether the present study could be extended to explore also the role of COPD and to determine the associations with cardiovascular comorbidities in greater detail, such as in the multicentre cohorts COPDGene or COSYCONET. Recent interventional studies using bronchodilation [14] in COPD showed short-term benefits of improved lung function on cardiac filling and function; whether this applies to patients showing a reduction of lung function without COPD is unknown. Mechanical factors are also a major determinant of a rightward rotation of the electrical heart axes in the electrocardiogram, which can reach a clinically significant degree [15]. This again underlines that alterations in lung function are relevant for cardiac parameters and outcomes, as demonstrated in the present study.

To conclude, the topic of lung-heart interactions is of great interest in current research and possibly in the future treatment of both cardiac and lung diseases. Due to the common symptom, dyspnoea, assessment and distinction of pulmonary and cardiac causes are not always trivial, but it is important to keep in mind that lung function is linked to cardiovascular risk even in the absence of a diagnosis of COPD, as well as to alterations of cardiac parameters, even in the absence of manifest cardiac disease.

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