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Left heart function in COPD

Impact of lung deflation

COPD and cardiovascular dysfunction

Chronic obstructive pulmonary disease (COPD) plays a major part in morbidity and mortality worldwide. The burden of disease is high with 384 million cases, corresponding to a prevalence of 11.7% [1]. Moreover, COPD is the third leading cause of death globally [2]. Although COPD primarily affects the lungs, cardiovascular disease is the most common extrapulmonary comorbidity [3]. The mortality of COPD patients due to cardiovascular factors is higher than the mortality related to respiratory insufficiency and failure [4]. These patients have an increased risk for developing ischemic heart disease, heart failure, arrhythmias, peripheral vascular disease, and arterial hypertension, of which the latter is the most common cardiovascular comorbidity [5]. In part, this co-occurrence can be explained by shared risk factors such as cigarette smoking. Synergistically, the pathophysiologic consequences of inflammatory small-airway damage directly affect the cardiovascular system. Among them, exacerbations increase the risk of myocardial infarction in COPD patients with coronary artery disease. Also, they can contribute to the development of atrial fibrillation.

Pulmonary hypertension impairs right ventricular function, leading to dilation, hypertrophy, and the risk of atrial fibrillation. Another factor that adversely modulates the cardiovascular system is the striking neurohumoral activation that has been reported in COPD [6]. The resulting increased sympathetic activity is associated with more frequent hospitalization and premature death [7]. The pathogenesis of this increased sympathetic activity is multifactorial; hypoxia [8], impaired baroreflexes [9], and hyperinflation [10] have been described as triggers. Hyperinflation is thought to increase the wall tension of the left and right ventricle via a more negative pleural pressure [10, 11], thus leading to an increased sympathetic tone. Detrimental consequences include increased breathing frequency, dead-space ventilation and exertional dyspnea [12, 13], impaired endothelial function with decreased exercise-induced vasodilatation in skeletal muscles, a decrease in the number of type 1 slow muscle fibers, cardiomyocyte injury, apoptosis, muscle wasting, and reduced exercise capacity [6, 14, 15].

Hyperinflation is a major lung function abnormality characterized by an increased residual volume and intrathoracic gas volume as well as a decreased vital capacity. The resistance and compliance of the lung deteriorate, and gas exchange is compromised. Hyperinflation has been identified as an important factor contributing to the impairment of cardiac function in COPD (**Fig. 1**) [16]. For example, Barr et al. observed an inverse relationship between emphysema with airflow obstruction and left ventricular filling, stroke volume, and cardiac output [17]. More recently, Alter et al. demonstrated reduced diastolic filling in COPD subjects with emphysema and suggested a decrease in preload to be the underlying mechanism [18]. Substantial benefits are to be gained from investigating the connection between hyperinflation and impaired cardiac function in COPD, and how pharmacotherapy can positively intervene in this devastating relationship. The following section presents previous contributions and the latest insights in this field of COPD research.

Hyperinflation and impaired left heart function

The link between lung hyperinflation and reduced cardiac function has received much attention in recent years. Basically, it has long been acknowledged that in severe COPD with its high pulmonary vascular resistance and intrathoracic pressure exceeding venous pressure, reduced cardiac filling is a logical consequence. Yet Barr et al. demonstrated that airflow obstruction, as measured by spirometry, and extent of emphysema, as measured by computed tomography, are inversely related to left ventricular end-diastolic volumes, stroke volume, and cardiac output. Interestingly, these phenomena are also evident in patients with mild hyperinflation and no cardiac comorbidity [17]. The underlying mechanism is apparently more complex and still not fully understood.

Smith et al. observed that the pulmonary veins were compressed in patients with emphysema and proposed that left ventricular filling is lowered by reduced preload due to pulmonary—not cardiac—causes [19]. Confirming this assumption, Watz et al. described an

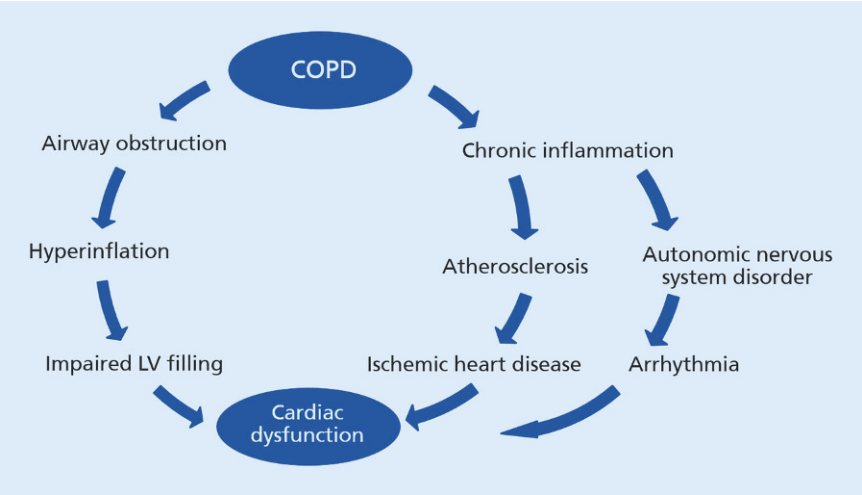

Fig. 1 ▲ Pathological mechanism of cardiac dysfunction in chronic obstructive pulmonary disease (*COPD*). *LV* left ventricular

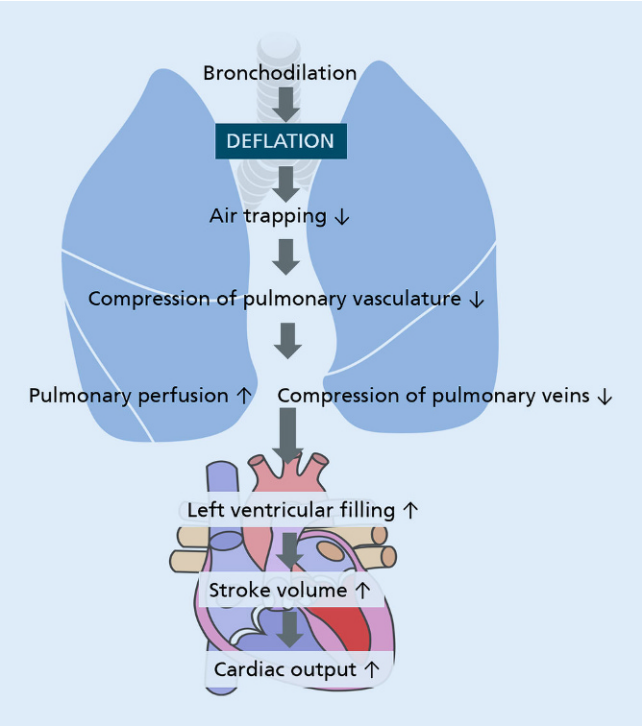

Fig. 2 ◀ Effect of lung deflation on left ventricular filling and cardiac output in patients with chronic obstructive pulmonary disease and hyperinflation. (© "Lung" from emcurious.com by Michael Macias/CC BY 4.0; © "Heart" from en.wikipedia.org by Drake/CC BY 2.0; Both pictures made transparent from original)

impaired left ventricular diastolic filling pattern and an impaired global right ventricular function in hyperinflated patients [16]. This supports the concept of reduced preload in patients with emphysema, since left ventricular isovolumetric relaxation time was unaffected by hyperinflation, indicating no connection with left ventricular distensibility. Kohli et al. found that hyperinflated patients have a lower cardiac output, left and right ventricular end-diastolic volume, although the ejection fraction remained unchanged [20]. Another effect of COPD on the heart is decreased chamber sizes. Watz et al. observed that with increasing GOLD (Global Initiative for Chronic Obstructive Lung Disease) stage, all cardiac chamber sizes decrease [16]. The strongest association was seen between parameters of static hyperinflation (inspiratory-to-total lung capacity ratio) and chamber sizes. Another relevant aspect of lung dysfunction in COPD is the reduced pulmonary microvascular blood flow (PMBF). Aron et al. suggested that smoking-related pulmonary vascular changes such as hyperinflation might lead to compression of the pulmonary capillary bed [21]. They found an association between reduced total pulmonary vascular volume (TPVV) and decreased left ventricular end-diastolic volume (LVEDV), stroke volume, and cardiac output. Ventricular relaxation and ejection fraction were not impaired, again hinting at pulmonary causes. Besides hyperinflation-induced pressure increase, further pulmonary causes could be regional hypoxic vasoconstriction resulting in reduced blood flow to the left heart [21].

In summary, the association between COPD and reduced left ventricular function might be due to reduced blood flow caused by pulmonary vasculature damage as well as increased intrathoracic pressure compressing the pulmonary veins that supply the left heart with oxygenated blood from the lungs. Hyperinflation likely plays a major role in this pathophysiological process. These observations lead to the question of whether lung deflation might positively affect left ventricular function.

Impact of deflation

Long-acting bronchodilators, which are recommended as the pharmacological treatment of choice in COPD [5], effectively reduce gas trapping and thus antagonize hyperinflation [22, 23]. The parasympatholytic or sympathomimetic stimulation of muscarinic and beta-adrenergic receptors in the bronchial muscles facilitate relaxation, which stabilizes the bronchioles and prevents collapsing. This results in an improved air flow in the small airways und temporarily reduces hyperinflation for the duration of the drugs' action. The deflation decreases the compression of the pulmonary microvasculature and consecutively pulmonary perfusion increases. As a consequence, cardiac filling also improves (◘ **Fig. 2**).

Santus et al. were the first group to examine the effects of single-bronchodilator-mediated deflation on cardiac

function [24]. They observed improved right ventricular compliance indexes and a reduced heart rate in association with a decreased residual volume under treatment. Stone et al. added an inhaled corticosteroid (ICS) to the treatment regimen with a single long-acting beta2-agonist [25]. They reported an improved biventricular stroke volume, left atrial function, and pulsatility within the pulmonary circulation. Deflation led to a decompression of the pulmonary vasculature and heart, thus end-diastolic volume and consecutively stroke volume increased. However, Beeh et al. demonstrated, that a combination of a long-acting beta2-agonist (LABA) with a long-acting muscarinic receptor antagonist (LAMA) is superior to a LABA combined with an ICS in improving lung functioning [26]. Hence, Hohlfeld et al. recently investigated the effect of a combined LABA/LAMA treatment on cardiac functioning and hyperinflation in the CLAIM study [27]. Since bronchodilation is more pronounced under a dual treatment, it was assumed that the effects on deflation and consequently cardiac functioning would be more distinct. The study results confirmed this: The once-daily dual bronchodilator treatment for 14 days with indacaterol and glycopyrronium significantly reduced hyperinflation and airflow obstruction in COPD patients. Left ventricular end-diastolic volume increased by approximately 10%. The positive cardiac effects were stronger than previously shown in the aforementioned study by Stone et al.

Complementary to the assessment of lung and heart parameters, Vogel-Claussen et al. evaluated data on pulmonary microvascular blood flow obtained during the CLAIM study [28]. Besides deflation of the lung, the LABA/LAMA combination was able to remarkably improve pulmonary microvascular blood flow (PMBF). This improvement in pulmonary vasculature is significantly linked to the increased left ventricular end-diastolic volume. The authors presumed the positive effects to be mediated by an increased regional ventilation leading to reduced parenchymal hypoxia, improved endothelial function and vasodilation of the pulmonary vas-

Abstract · Zusammenfassung

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Left heart function in COPD. Impact of lung deflation

Abstract

Chronic obstructive pulmonary disease (COPD) primarily affects the lungs; however, cardiovascular conditions are among the most common extrapulmonary comorbidities. Besides shared risk factors such as cigarette smoking, pathophysiological connections between the lung and the heart have been identified as mediators of reduced cardiac output. Recent research has focused on hyperinflation of the lung as a pulmonary cause for heart dysfunction. Hyperinflation is a typical lung abnormality seen in COPD; it is characterized by increased residual volume, intrathoracic gas volume, and total lung capacity while vital capacity is decreased. The degree of hyperinflation with airway obstruction is inversely related to left ventricular filling, stroke volume, and cardiac output. The underlying mechanisms are assumed to be compression of the pulmonary veins and thus reduced preload of the left heart as well as decreased pulmonary microvascular blood flow due to compression of the pulmonary vasculature. Treatment with a dual bronchodilator antagonizes this detrimental lung–heart unbalance effectively: Pulmonary blood flow, left ventricular end-diastolic volume, and stroke volume increase in COPD patients without cardiac abnormalities. Similar effects, yet less pronounced, were reported with single bronchodilator therapy. Future work needs to investigate whether these promising findings can be reproduced in COPD patients with cardiovascular diseases.

Keywords

Pulmonary disease, chronic obstructive · Hyperinflation · Bronchodilator effect · Left ventricular function · Cardiovascular disease

Linksherzfunktion bei COPD. Einfluss der Lungenentblähung

Zusammenfassung

Die chronisch obstruktive Lungenkrankheit (COPD) betrifft in erster Linie die Atemwege, kardiovaskuläre Erkrankungen zählen jedoch zu den häufigsten Komorbiditäten. Neben gemeinsamen Risikofaktoren wie dem Rauchen tragen zusätzlich pathophysiologische Wechselwirkungen zwischen Lunge und Herz zu einer verringerten kardialen Auswurfleistung bei. Die aktuelle Forschung konzentriert sich v. a. auf die Lungenüberblähung als Ursache für eine reduzierte Herzleistung bei Patienten mit COPD. Die Überblähung ist eine für die COPD typische pulmonale Funktionsabweichung, die durch ein erhöhtes Residualvolumen, intrathorakales Gasvolumen und eine vergrößerte totale Lungenkapazität bei erniedrigter Vitalkapazität charakterisiert ist. Das Verhältnis zwischen dem Grad der Überblähung und linksventrikulärem enddiastolischem Volumen, Schlagvolumen und Auswurfleistung ist umgekehrt proportional. Dem zugrunde liegen vermutlich erstens eine Kompression der Pulmonalvenen, die zu einer verringerten linksventrikulären Vorlast führt, und zweitens eine Kompression des pulmonalen Kapillarbetts, die einen verminderten pulmonalen mikrovaskulären Blutfluss zur Folge hat. Die Behandlung mit einem dualen Bronchodilatator korrigiert dieses Ungleichgewicht zwischen Lunge und Herz in eine günstige Richtung: Pulmonaler Blutfluss, linksventrikuläres enddiastolisches Volumen und Schlagvolumen erhöhen sich bei überblähten COPD-Patienten ohne kardiale Vorerkrankungen. Ähnliche Effekte, jedoch schwächer ausgeprägt, lassen sich mit einer einfachen bronchienerweiternden Therapie erreichen. Eine zukünftige Forschungsaufgabe liegt darin zu untersuchen, ob sich ähnlich positive Ergebnisse bei COPD-Patienten mit kardiovaskulären Erkrankungen erzielen lassen.

Schlüsselwörter

Lungenerkrankung, chronisch obstruktive · Überblähung · Bronchodilatatorwirkung · Linksherzfunktion · Kardiovaskuläre Erkrankung

culature. Moreover, the enhanced organ functioning under LABA/LAMA treatment translated into improved patient-reported outcomes as indicated by increases in health status and dyspnea during the CLAIM study. The magnitude of effect on symptoms and quality of life was greater compared with previous studies on indacaterol/glycopyrronium [29, 30]. While all participants suffered mainly from moderate-to-severe COPD, patients in the CLAIM study differed in their degree of hyperinflation because they were selected accordingly and they did not suffer from relevant cardiac abnormalities. Nonetheless, left ventricular end-diastolic volume obtained at baseline was near the lower limit of normal and cardiac function parameters significantly improved after lung deflation. The rise that was achieved with the LABA/LAMA treatment is likely to be beneficial in the long term even for patients with hyperinflation and COPD without distinct cardiovascular dysfunction.

Practical conclusion

Prior work has documented the connection between lung hyperinflation and impaired cardiac function; Watz et al., for example, reported reduced heart chamber sizes, impaired left ventricular diastolic filling pattern, and an impaired global right ventricular function in patients with hyperinflation and COPD . Pharmacological deflation with single-bronchodilator treatment increased left ventricular end-diastolic volume and decompressed the pulmonary vasculature. However, treatment with a dual bronchodilator surpassed the positive effects of a mono-therapy, confirming the substantial benefits of a strong bronchodilation in reducing air trapping and thus reversing hyperinflation. Furthermore, there is compelling evidence that dual bronchodilation improved pulmonary microvascular blood flow, which enhances organ functioning of the heart and lung. Although patient-reported outcomes regarding health status and dyspnea also improved under this therapy regimen, it still remains unclear whether the positive vascular changes are sustained over extended periods of time. Also, future research needs to explore whether these promising results can be extended to COPD patients with comorbid cardiac diseases.

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Compliance with ethical guidelines

Conflict of interest J. Bauersachs's, T. Welte's and J.M. Hohlfeld's institutions received grant support for conducting clinical studies on the impact of dual bronchodilation in COPD patients by Novartis Pharma GmbH. Moreover, they received personal honoraria for advisory boards and lectures from Novartis Pharma GmbH. N. Struß declares that she has no competing interests.

For this article no studies with human participants or animals were performed by any of the authors. All studies performed were in accordance with the ethical standards indicated in each case.

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Schonender Blick ins Herz

Die nicht-invasive Messung der Herzdurchblutung mit Magnetresonanztomographie (MRT) ist dem Herzkatheter ebenbürtig. Das zeigt eine internationale Studie unter Federführung der Goethe-Universität, die im New England Journal of Medicine erschienen ist.

Bei Patienten mit Brustschmerzen und vermuteter stabiler koronarer Herzkrankheit (KHK) hängt die Therapie in erster Linie davon ab, wie stark die Koronararterien verengt sind. Dies wird oft durch das Einführen eines Herzkatheters untersucht. Im Zweifel wird zusätzlich der Druck in den Koronararterien gemessen. Die Kombination beider Methoden ist der derzeit anerkannte Standard für Therapieentscheidungen.
Eine vielversprechende Alternative, die Durchblutung des Herzmuskels nichtinvasiv und direkt zu erfassen, ist die kardiovaskuläre MRT.
Im Gegensatz zur Computertomographie (CT) kommt das MRT ohne ionisierende Strahlung aus und liefert obendrein präzisere Messungen des Blutdurchflusses als herkömmliche Techniken. Das konnte das Team um Prof. Eike Nagel, Direktor des Instituts für Experimentelle und Translationale Kardiovaskuläre Bildgebung an der Goethe-Universität, jetzt zeigen. Im Rahmen der MR-INFORM-Studie untersuchte es an 918 Patienten mit einer Indikation zur Herzkatheteruntersuchung, ob die Untersuchung mit dem MRT zu gleichen Ergebnissen führt wie die derzeitige invasive Technik.
Hierfür wurden die Patienten zufällig in zwei Gruppen eingeteilt. Die eine Gruppe erhielt die Standarddiagnostik mit Herzkatheter mit einer zusätzlichen Druckmessung in den Koronararterien, während die andere Gruppe nichtinvasiv mit MRT untersucht wurde. Wenn im MRT eine beeinträchtigte Durchblutung des Herzens nachgewiesen wurde, planten die Forscher mit Hilfe einer Katheter-Untersuchung das weitere Vorgehen. In jedem Studienarm wurden verengte Herzkranzgefäße erweitert, wenn dies aufgrund der Untersuchung angezeigt war. Innerhalb des folgenden Jahres dokumentierten die Ärzte, wie viele Patienten starben, einen Herzinfarkt erlitten oder eine erneute Gefäßerweiterung benötigten. Außerdem erfassten sie, ob die Herzbeschwerden weiterhin bestanden.

Das Ergebnis: in der Gruppe der mit MRT untersuchten Patienten benötigten weniger als die Hälfte einen diagnostischen Herzkatheter und weniger Patienten bekamen eine Gefäßerweiterung (36% vs 45%). Das bedeutet: durch eine vorgeschaltete MRT-Untersuchung lassen sich Herzkatheter-Untersuchungen sowohl zu diagnostischen als auch zu therapeutischen Zwecken einsparen. Beide Gruppen unterschieden sich jedoch nicht bezüglich weiterbestehender Beschwerden oder des Auftretens erneuter Beschwerden, Komplikationen oder Todesfällen.

„Damit können Patienten mit stabilen Brustschmerzen, die bisher einen Herzkatheter bekommen, alternativ mit einer Durchblutungsmessung mit MRT untersucht werden", folgert Prof. Eike Nagel. „Die Ergebnisse für den Patienten sind genauso gut, die Untersuchung mit MRT hat jedoch viele Vorteile: Sie dauert weniger als eine Stunde, Patienten erhalten lediglich eine kleine Kanüle in den Arm und werden keiner Strahlung ausgesetzt."
Die Hoffnung des Mediziners ist, dass die schonende Untersuchung nun als Methode erster Wahl eingesetzt wird und so Herzkatheteruntersuchungen eingespart werden können.

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