clinically reduced following treatment with continuous positive airway pressure (CPAP). In those patients with good adherence to CPAP therapy, a significant percentage recovered their normal nocturnal dipper pattern and/or reversed their riser pattern. Miguel Ángel Martínez-García, MD, PhD, University and Polytechnic La Fe Hospital, Valencia, Spain, presented the results of the HIPARCO study (Hipertensión Arterial Resistente Control con CPAP).

The objective of this multicenter study was to evaluate the effect of CPAP treatment on the BP levels and nocturnal BP pattern in patients with OSA and RH. Patients with RH and confirmed OSA were randomly assigned to usual medical therapy (n=96) or medical therapy plus tritated fixed CPAP pressure (n=98) for 12 weeks. Patients with 24-hour BP >130/85 mm Hg (determined by ambulatory blood pressure monitoring [ABPM]), an apnea-hypopnea index >15, and with at least an 80% adherence to antihypertensive drug treatment were included in the study. The prevalence of OSA was 82.7% in this population. Patients were monitored at 2, 4, 8, and 12 weeks for CPAP and antihypertensive drug adherence, changes in body mass index (BMI), and new cardiovascular (CV) events. At the 12-week visit, patients underwent a second 24-hour ABPM. All randomized patients independent of their use or not of CPAP were included in the intention-to-treat (ITT) analysis and those patients with a good adherence to CPAP (≥4 hours of CPAP; 68% of randomized patients) use were included in the per protocol (PP) analysis.

Demographics and baseline characteristics were similar in both treatment groups. Mean BMI was 34.1±5.4 kg/m^2; mean number of antihypertensive drug used was 3.8±0.9; 21.4% of participants had past CV events; mean 24-hour BP was 144.2±12.5/83.0±10.5 mm Hg. More than 70% of patients had a nondipper (42.8%) or a riser nocturnal (31.4%) BP pattern. Almost 95% of the patients were on diuretics.

In the ITT analysis, the use of CPAP significantly reduced diastolic (p=0.005) and mean BP (p=0.016) compared with the control group and was associated with a near-significant reduction in 24-hour systolic BP (p=0.09). The reductions (about 3 mm Hg), were clinically relevant. In the PP analysis (only those patients with good CPAP compliance), CPAP use significantly decreased systolic BP, diastolic BP, and mean BP by 4 to 5 mm Hg (p=0.01, p=0.001, and p=0.001, respectively).

The decreases in BP levels were more pronounced during the night especially in those patients with better tolerance to CPAP.

The probability of recovering the dipper pattern and reversing the riser pattern was significantly greater (p≤0.03) in both the ITT and PP analysis in those patients allocated to the CPAP arm. Moreover there was a positive and linear relationship between the number of hours of CPAP use and the decrease in 24-hour BP values, both BP diurnal and nocturnal values. The presence or absence of daytime hypersomnolence, sex, age, years from RH diagnosis and BMI had no impact on treatment effectiveness.

Prof. Martínez-García would like to see future long-term studies that analyze the effect of CPAP on the incidence of CV events or death and BP treatment in these patients with RH and OSA.

Effects of Obesity on COPD

Written by Lori Alexander

The impact of obesity on many chronic diseases is well known, but the effect of obesity on clinical outcomes for people with chronic obstructive pulmonary disease (COPD) is less clear. The number of people with COPD who are obese is expected to increase in line with the obesity pandemic, making it essential to gain a better understanding of the effects of obesity on COPD.

Although classically considered a wasting disease, a link between COPD and obesity is becoming increasingly recognized, and it may influence clinical diversity in COPD, said Frits M. E. Franssen, MD, PhD, CIRO+, Center of Expertise for Chronic Organ Failure, Horn, The Netherlands. Data on the prevalence of obesity in COPD have conflicted, with some studies showing a higher prevalence among patients with COPD and others showing a lower prevalence [Vozoris NT, O'Donnell DE. *Can Respir J* 2012; Montes de Oca M et al. *Respir Med* 2008; Steuten LM et al. *Prim Care Respir J* 2006; Eisner MD et al. *Respir Res* 2007]. Low levels of physical activity have been consistently reported for patients with COPD, and this may contribute to weight gain.

> Increasing evidence suggests a relationship between visceral adipose tissue dysfunction and the pathophysiology of COPD.

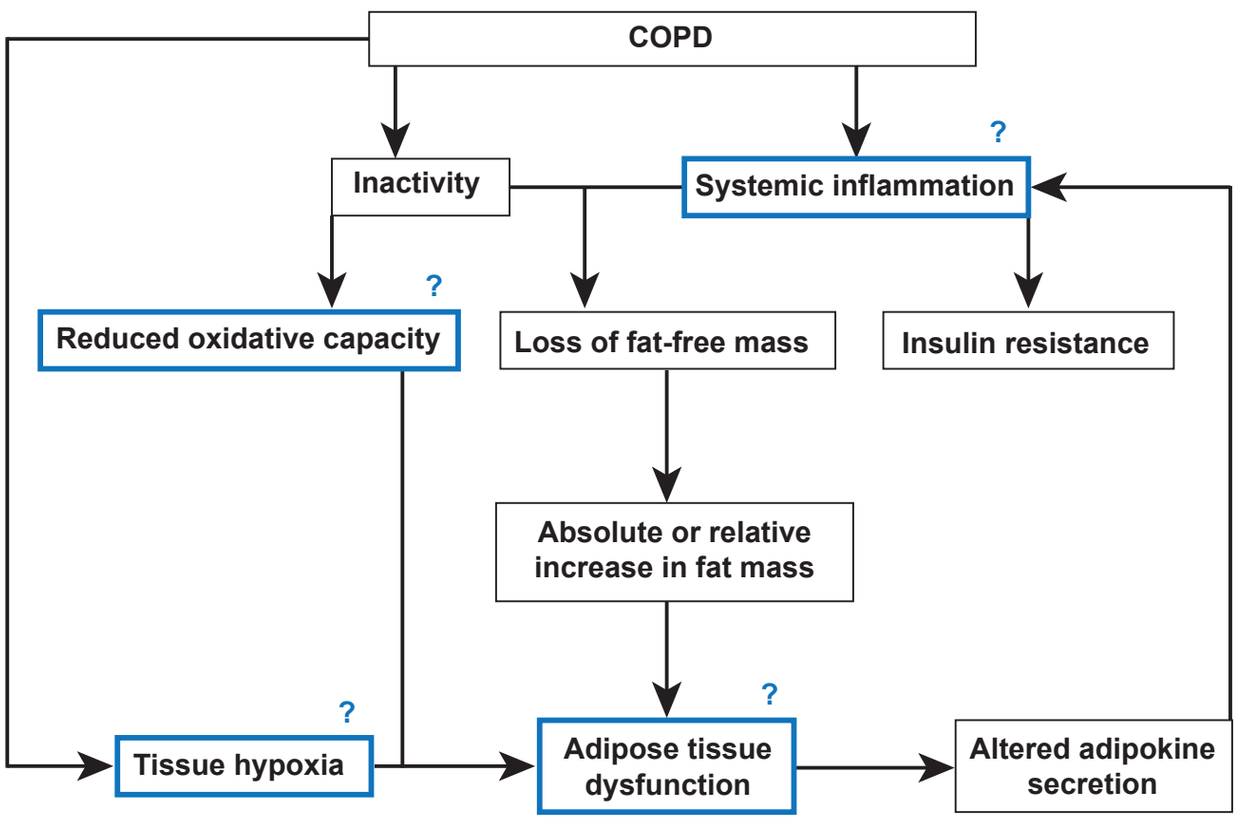
Contrary to expectations, obesity is not necessarily associated with worse patient-related outcomes in COPD, said Prof. Franssen. He pointed to an early study in which the risk of mortality was evaluated in obese and normal-weight patients with COPD [Landbo C et al. *Am J Respir Crit Care Med* 1999]. The relative risk of all-cause mortality was increased for obese patients with mild or moderate COPD compared with normal-weight patients. However, in contrast to patients with mild and moderate COPD, among patients with severe disease, the risks of all-cause and COPD-related mortality were lowest for obese patients. These findings are referred to as the obesity paradox.

Studies have also found different effects of obesity on dyspnea. In one study, obese patients with COPD reported increased dyspnea and poorer health-related quality of life

than normal-weight patients, even though lung function was better for obese and overweight patients [Cecere LM et al. *COPD* 2011]. In another study, researchers found that absolute peak workload increased in obese patients during cycling (compared with normal-weight patients matched for age and degree of airway obstruction), but these patients reported less dyspnea during all levels of ventilation during cycling [Ora J et al. *Am J Respir Crit Care Med* 2009].

Obesity appears to be a substantial contributor to systemic inflammation in COPD. Adipose tissue is a source of inflammation in obesity, and obesity (high body mass index [BMI]) has been associated with highly elevated levels of C-reactive protein in patients with COPD; the highest levels have been found in patients with abdominal obesity [Breyer MK et al. *Clin Nutr* 2009]. In addition, persistent inflammation in COPD has been associated with a high BMI but not with the fat-free mass index, which suggests a role for adipose tissue in the inflammatory process [Agusti A et al. *PloS One* 2012; van den Borst B et al. *Am J Respir Crit Care Med* 2013]. Prof. Franssen said that increasing evidence suggests a relationship between visceral adipose tissue dysfunction and the pathophysiology of COPD, and he proposed several links between COPD, adipose tissue dysfunction, and systemic inflammation, noting that several questions remain unanswered (Figure 1) [Franssen FME et al. *Thorax* 2008].

Figure 1. Obesity and Chronic Obstructive Pulmonary Disease: Unanswered Questions



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