in the management of pyopneumothorax with intercostal tube drainage only. The outcome of treatment was poor in the tubercular pyopneumothorax.

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Effect of 3-month CPAP treatment on blood pressure and serum aldosterone concentration in patients with resistant hypertension

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Introduction: The role of the renin-angiotensin-aldosterone system (RAAS) on the association obstructive sleep apnea (OSA) and resistant hypertension (RH) is unclear. Aim: To analyze the effect of CPAP on 24-h blood pressure monitoring (ABPM) and on serum aldosterone in RH patients. Methods: 102 patients with an OSA15 and office-RH were randomized to CPAP (n=50) or to conventional treatment (n=52) for 3 months. 24-h ABPM and serum aldosterone were measured. 78 patients completed the follow-up (36 CPAP, 42 conventional treatment). Results: 66.7% were male, aged 58.27±9.3yrs and AHI 50.1±21.6, without significant differences between both groups. Serum aldosterone and AHI correlated significantly in patients with ABPM-confirmed RH (r= 0.25, p=0.02). CPAP achieved a significant decrease in serum aldosterone and 24-h BP decreased especially in those with ABPM-confirmed RH.

Effects of 3-month treatment on 24-h BP and serum aldosterone in patients with ABPM-confirmed resistant hypertension, randomized to conventional treatment or to CPAP

mmHg	Baseline	Conventional treatment	p	Baseline	CPAP	p
Daytime SBP	142.6±12.39	141.92±15.88	0.76	143.16±12.12	137.8±13.3	0.03
Daytime DBP	83.57±12.46	83.92±14.2	0.75	83.41±10.26	80.08±9.93	0.02
Nighttime SBP	132.5±17	133.57±19.82	0.75	130.5±15.6	124.45±12.41	0.08
Nighttime DBP	77.35±15.02	77.2±18.1	0.94	74.2±9.3	69.1±7.7	0.007
24-h SBP	139.57±12.4	141.93±15.88	0.3	140.08±10.95	137.8±13.3	0.31
24-h DBP	81.4±12.65	81.6±14.97	0.84	80.8±9.35	76.41±9.07	0.002
Serum aldosterone (ng/dL)	25.24±11.27	24.45±10.23	0.6	25.28±9.23	21.76±9.32	0.01

Conclusion: The association between OSA and RH could be mediated at least in part by an effect on the RAAS.

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The effect of simulated obstructive hypopnea and apnea on thoracic aortic wall transmural pressures

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Background: Preliminary evidence supports an association between OSA and thoracic aortic dilatation, although potential causative mechanisms are incompletely understood; these may include an increase in aortic wall transmural pressures, induced by obstructive apnoeas and hypopnoeas. **Methods:** In patients undergoing cardiac catheterization mean blood pressure (MBP) in the thoracic aorta and oesophageal pressure were simultaneously recorded by an indwelling aortic pigtail catheter and an oesophageal catheter in randomized order during: normal breathing, simulated obstructive hypopnoea (inspiration through a threshold load), simulated obstructive apnoea (Mueller manoeuvre), and end-expiratory central apnoea. Transmural aortic pressure (aortic MBP minus oesophageal pressure) was calculated as a measure of aortic wall stress. **Results:** 10 patients with a median age (range) of 64 (46-75) years were studied. Inspiration through a threshold load, Mueller manoeuvre and end-expiratory central apnoea were successfully performed and recorded in 10, 7 and 9 patients, respectively. The difference between aortic MBP and oesophageal pressure (and thus the extra aortic dilatory force) was median (quartiles) +9.3 (+5.4, +18.6) mmHg, p=0.02 during inspiration through a threshold load, +16.3 (+12.8, +19.4) mmHg, p=0.02 during the Mueller manoeuvre, and +0.4 (-4.5, +4.8) mmHg, p=0.80 during end-expiratory central apnoea.

Conclusions: Simulated obstructive apnoea and hypopnoea increase aortic wall dilatory transmural pressures because intra-aortic pressures fall less than oesophageal pressures. Thus OSA may mechanically promote thoracic aortic dilatation.

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Acute improvement of pulmonary hemodynamics does not alleviate Cheyne-Stokes respiration in chronic heart failure - a randomized, controlled, double-blind, crossover trial

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Background: Studies confirmed Cheyne-Stokes respiration (CSR) to be associated with elevated pulmonary capillary wedge pressure (PCWP) in chronic heart failure (CHF). This study aimed to investigate the acute effects of lowering PCWP and pulmonary artery pressure (PAP) on CSR severity.

Methods: 21 consecutive patients with CHF and CSR (apnea-hypopnea-index (AHI≥15/h)) underwent right heart catheterization, followed by infusion of glyceryltrinitrate (GTN), and inhalation of iloprost. Throughout the procedure PAP and PCWP was measured invasively. Afterwards maximum tolerable dosage of GTN and iloprost were randomly applied in 2 split-night procedures versus i.v. or inhalative NaCl 0.9% under full polysomnography monitoring.

Results: Mean (m)PAP was significantly reduced by GTN (20.1±9.0 to 11.6±4.2mmHg, p<0.001; infusion rate 6.2±1.5ml/h) and iloprost (16.9±7.9 to 14.2±6.4mmHg, p<0.01; dosage: 10 mcg/ml), whereas mPCWP was lowered exclusively by GTN (14.0±5.6 to 7.2±3.9 mmHg, p<0.001; iloprost: 11.7±6.2 to 11.0±6.3mmHg, p=n.s.). Compared to placebo sleep studies revealed no significant improvement of AHI (GTN: 39.0±17.7/h vs. 35.3±16.0/h, p=n.s.; iloprost: 34.9±21.9/h vs. 33.3±19.4/h, p=n.s.) and central apnea index (GTN:14.4±16.5/h vs. 11.5±16.37h, p=n.s.; iloprost:11.0±16.3/h vs.12.5±19.1/h, p=n.s.) following GTN or iloprost treatment, respectively.

Conclusion: GTN and Iloprost led to a significant reduction in PAP, whereas PCWP was lowered by GTN exclusively. However, acute improvement of pulmonary congestion had no impact on CSR severity. Extended treatment periods appear crucial for successful causal therapies.

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OSAS severity is associated to decreased heart rate turbulence slope

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Obstructive sleep apnea syndrome (OSAS) has been associated to impaired baroreflex sensitivity (BRS) which has recently been shown to be non-invasively assessed by heart rate turbulence (HRT) analysis. Although HRT seems to be better suited than traditional heart rate variability indexes for autonomic assessment in presence of respiratory and arrhythmic disorders, very few papers addressed its evaluation in OSAS. Aim of the study is to find out whether and to which extend HRT is associated to OSAS severity. HRT consists of sinus cycle length fluctuations following spontaneous isolated VPC composed by an early rate acceleration phase, namely turbulence onset (TO), and by a late deceleration phase, namely turbulence slope (TS). We studied HRT in polysomnographic recordings of 82 mild (5<AHI<15), 74 moderate (16<AHI<30) and 65 severe (AHI>30) OSAS pts. (age 62±14, 71% males). Results showed that, while TO values did not significantly differ between mild (-0,78±1,50), moderate (-0,89±1,78) and severe (-0,70±1,28) pts., TS significantly decreases (Kruskal-Wallis P value <0.05) from mild (3,27±2,7) to moderate (2,6±2,6) and severe (1,98±2,5) pts., with a significant Dunn's multiple comparisons post test only between mild vs. severe OSAS pts. Data indicate that the main BRS alterations do not appear in the early HRT phase triggered by transient vagal inhibition, with TO<0 normal values in mild to severe pts., but during the slow one, due to the sympathetic hyperactivity affecting the heart rate recovery, with TS<2.5 abnormal values associated to increasing OSAS severity. These findings support the conclusion that HRT assessment could have a prognostic value related to the development of cardiovascular disease in OSAS.