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humans during a breathhold at FRC using a functional MRI technique, arterial spin labeling. Perfusion was measured in 3 non-overlapping 15mm thick sagittal slices covering most of the right lung in a 1.5T scanner using a phased-array torso coil. Lung density was measured in the same slices using a short echo 2D FLASH sequence via back-extrapolation to  $M_0$  based on published values of  $T_2^*$ . Coil inhomogeneity was corrected for using body coil images; values referenced to a water phantom, and 3 ROIs established in the vertical direction: nondependent (N), middle (M), and dependent(D). Perfusion was lowest in the N and not different between M and D ( $1.06 \pm 0.10$ (N),  $1.81 \pm 0.18$ (M),  $1.67 \pm 0.15$ (D); mean  $\pm$ SE; ml(blood)/min/ml(lung);  $p < 0.001$ ). Lung density showed a similar gradient ( $0.32 \pm 0.04$ ,  $0.38 \pm 0.03$ ,  $0.47 \pm 0.02$ ; gm/ml(lung);  $p < 0.05$ ). However flow/density was not gravitationally affected and showed the highest values in M ( $4.18 \pm 0.58$ ,  $5.25 \pm 0.58$ ,  $3.83 \pm 0.30$ ; ml(blood)/min/gm;  $p < 0.05$ ). The results show that gravitationally induced compression of lung tissue is a significant factor affecting the overall distribution of pulmonary perfusion.

**P2390****Lung diffusion capacity is reduced in patients with coronary artery disease and no signs of heart failure**

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Chronic heart failure (CHF) is known to impair lung function, especially gas diffusion, but few information is available about respiratory function test (RFT) in patients with a history of coronary artery disease (CAD) and no signs of CHF. AIM to assess RFT in a group of CAD patients (2-7 years from coronary bypass) attending a program of cardiac rehabilitation (aerobic exercise training 3 times/week). SUBJECTS and METHODS: in 11 M (age 55-72 years, 8 no smokers, 3 previous smokers  $\geq$  5years) with no exercise limitation (mean  $VO_{2max}$  89.3 of predicted, range 73%-115%, normal VE/VCO<sub>2</sub> slope during exercise), no reduced systolic function (EF  $64.7 \pm 1.9$ ), RFT and alveolar diffusing capacity (DLCO sb) at rest were assessed. RESULTS Standard pulmonary function tests were not affected but the diffusion capacity was moderately reduced. No significant correlation has been found between DLCO and any other index.

VC	FEV1	FEV1/VC	RV	KCO	DLCO VA	Hb
99% $\pm$ 2.2	93.2% $\pm$ 3.4	93.6% $\pm$ 1.2	106% $\pm$ 6.3	52.1% $\pm$ 3.8	63.6 $\pm$ 4	14.9 $\pm$ 0.2

RFT values are expressed as% of predicted. Hb g/dl.

**Conclusion:** in CAD patients with no evidence of heart failure, the pulmonary diffusion capacity is impaired; in the absence of obstructive and vascular lung disease and anaemia, the observed abnormalities could be related to ultrastructural changes in the endothelial alveolar membrane. The magnitude of these changes and their effect on exercise performance should be carefully followed up. Monitoring of pulmonary function seems indicated in patients with a history of CAD even if asymptomatic and may provide complementary information.

**P2391****Reference values for NO and CO transfers and the related variables Dm and Vc**

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The double NO/CO transfer method allow to calculate Dm and Vc in one step. It seems that no reference values using this new method have been published. 310 healthy subjects (166 men and 144 women) were recruited from two French cities (238 in Grenoble and 72 in Bordeaux). Ages ranged from 18 to 94 y.

NO and CO transfers were measured using the single breath method. The inspired mixture contained 40ppm NO and 2000ppm CO. The material used (Hypair compact with Expair software) was automated (Medisoft Dinant B). Measurements were duplicated. Few subjects were rejected as they did not performed reproductive measurements.

A linear multiple regression model for each of the variables was tested. The independent variables were gender, age, height, weight, BMI. If the level of significance of a variable was greater than 0.05, the variable was discarded from the model.

CO and NO transfers, Dm and Vc were all gender, age and height dependent (see table, units: ml, mmHg, min). The ratio Vc/VA was only age and BMI dependent. Interestingly the ratio Dm/Vc did not depend on any of these variables. The interval between the fifth and the ninety fifth quantiles of the univariate distribution of Dm/Vc was 0.63; 1.04.

## Coefficients estimates

Dependent variables	Intercept	Gender(0W,1M)	Age	Height	R <sup>2</sup>
TLCO	-18.14	4.53	-0.22	33.5	0.70
TLNO	-77.71	24.49	-1.05	153.8	0.69
DmCO	-40.79	12.92	-0.52	78.13	0.67
Vc	-54.66	12.62	-0.63	99.85	0.64

**229. Pulmonary gas exchange****P2389****Vertical distribution of pulmonary perfusion and lung density in humans: the Slinky® effect**

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Measurements of the vertical gradient in pulmonary perfusion from radioactive tracer studies *in-vivo*, and microsphere studies reach different conclusions as to the magnitude of the effect of gravity. We hypothesized that a significant proportion of the vertical gradient seen during *in-vivo* studies was due to gravitationally induced compression of the dependent lung increasing vascular density in those regions, the Slinky® effect. This effect would be expected to be absent in microsphere studies in which the lungs are inflated post-mortem. We studied 4 normal supine

Compared to previous works the present results point out the progressive declining effect of age on Vc and Dm. The differences observed with other works could be due to the wide range of age used in this study as well as to some methodological specificity.

**P2392**

**Ventilation-perfusion imbalance and GOLD stages in patients with COPD**  
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The objective of this analysis was to evaluate comprehensively ventilation-perfusion (V<sub>A</sub>/Q) distributions in patients with COPD according to GOLD stages. We retrospectively examined V<sub>A</sub>/Q abnormalities from 126 COPD patients (all but 2 males). All patients were smokers or ex-smokers (range, 6-180 pack-yr). Results were as follows (mean±SE):

	Stages 0-1	Stage 2	Stage 3	Stage 4
n	9	33	29	55
Age, yr	59±6	62±1	63±1	62±1
FEV <sub>1</sub> ,% pred	90±3	63±2*	41±1*	26±1*
FEV <sub>1</sub> /FVC	0.68±0.02	0.54±0.02*	0.41±0.01*	0.32±0.02*
PaO <sub>2</sub> ,mm Hg	88±4	79±2	74±2*	60±1*
PaCO <sub>2</sub> , mm Hg	37±1	37±1	39±1	45±1*
Log SD Q	0.70±0.13	0.87±0.04	0.96±0.03	0.99±0.03
Log SD V	0.56±0.09	0.78±0.04*	0.97±0.04*	1.03±0.03
DISP R-E*	6.4±2.0	9.0±0.7	12.4±0.7*	13.7±0.6

\*p<0.05 compared to previous stage

V<sub>A</sub>/Q descriptors showed different correlations with spirometric and routine gas exchange variables: with FEV<sub>1</sub> (%) (Log SDQ, -0.16 (ns), Log SDV, -0.53 and DISP R-E\*, -0.46 [p<0.05 each]); with PaO<sub>2</sub> (-0.60, -0.52 and, -0.67; p<0.001 each); and with DL<sub>CO</sub> (-0.25 [p<0.01], -0.42 and, -0.43; p<0.001 each). These findings indicate that FEV<sub>1</sub> correlates predominantly with the presence of underlying areas of high V<sub>A</sub>/Q units whereas both PaO<sub>2</sub> and DL<sub>CO</sub> correlate with areas of low and high V<sub>A</sub>/Q units. Supported by Red Respira.

**P2393**

**Ventilation distribution and gas trapping as affected by increased gravitational load and compression of the lower body**

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Increased gravito-inertial forces in the head-to-foot direction (+Gz) and compression of lower body half exert significant effects on FRC, volume of trapped gas (VTG) and ventilation distribution (JAP 94: 1353-1364, 2003). The study aimed to separate abdominal (ABD) and leg compression effects on FRC, VTG and ventilation distribution in +Gz. Eleven healthy males performed a multiple breath washout (MBW) of 4% SF<sub>6</sub> at +1 without inflation and at +2 and +3Gz with separate and combined inflation to 12 kPa of ABD and leg bladders. Lung clearance index (LCI) was used to assess ventilation inhomogeneity. Increased Gz produced an increase in FRC, LCI and VTG. At +2 and +3Gz separate compression of leg and ABD reduced FRC, marginally increased VTG with no effect on LCI. Combined leg and ABD compression did not affect LCI but caused a greater reduction in FRC and increase in VTG compared to either factor alone. That LCI was unaffected by combined leg and ABD compression despite a marked increase in VTG might be due to substantial basilar airway closures masking any additional ventilation inhomogeneity.

Relative changes in FRC, VTG, and LCI from 1Gz values

Gz-level	+2Gz		+2Gz		+3Gz		+3Gz	
	No inflation	Lower limbs	Abdomen	Combined inflation	No inflation	Lower limbs	Combined inflation	
FRC (%)	+6.1 *	-9.4 ###	-6.6 ###	-20.2 ###	+5.8 ns.	-2.0 ns.	-15.1 ##	
VTG (%)	+40 ***	+71 ns.	+70 ns.	+147 #	+102 ***	+119 ns.	+253 ##	
LCI (%)	+9.1 **	+12.3 ns.	+10.4 ns.	+7.3 ns.	+16.5 ***	+17.2 ns.	+18.0 ns.	

\*p<0.01; \*\*p<0.001; \*\*\*p<0.001; ns. +1Gz; # p<0.01; ## p<0.01; ### p<0.001; vs. +2Gz no AGS pressure; Paired T-test

**P2394**

**Increase in lung capillary blood volume in native highlanders: angiogenesis?**  
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Chronic hypoxia causes angiogenesis in the adult rat pulmonary circulation (Howell et al J Physiol 2003.547.113-45). Human native highlanders are submitted to chronic hypoxia. If this hypoxia stimulates angiogenesis these people should have high capillary lung volumes.

18 subjects, 21 to 71y, living in Oruro (Bolivia, 4000m altitude) volunteered. 10 of them complained from symptoms compatible with high altitude polycythemia (Monge disease) as the remaining 8 did not. The membrane (Dm) and capillary (Vc) components of pulmonary diffusion capacity were measured by a double CO and NO diffusion method described previously (Guénard et al. Respir Physiol 1987.70, 113-20). An automated material was used (Medisoft Dinant B). Vc was corrected for haemoglobin concentration. Arterial blood gases were measured (Roche Opti CCA). An echographic estimation of heart functions was performed. Mean TLCO corrected for haemoglobin concentration was 48.7 ml min<sup>-1</sup> ImmHg<sup>-1</sup> (23.6 to 77.5), mean TLNO 179 (79 to 312) and mean Vc 188.5 ml (88.5 to 361). The ratio DmCO/Vc was 0.51(0.39 to 0.72).

Compared to lowlanders, Vc in these subjects were about twice and Dm/Vc values were outside the range of lowlanders (0.63 to 1.04) in most cases (15/18). No difference between subjects complaining from symptoms and others, even in the high haemoglobin concentrations, was found but PaO<sub>2</sub> which was lower in the symptomatic group. Left atrial pressure estimated by echocardiography was normal, these results suggest that hypoxia might stimulate angiogenesis in human at least in native highlanders. Their low Dm/Vc ratio suggests that the growth in the surface of the lung does not paralleled that of the capillaries.

**P2395**

**An informative noninvasive shunt estimate in hereditary haemorrhagic telangiectasia**

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**Purpose:** Traditionally shunt measurements are invasive or use expensive equipment (100% oxygen breathing, radionuclide, or bubble echocardiogram shunt studies). We evaluated the non-invasive P<sub>i</sub>O<sub>2</sub>:S<sub>p</sub>O<sub>2</sub> diagram to assess shunt in hereditary haemorrhagic telangiectasia (HHT) patients compared to normal controls.

**Patients and Methods:** Six healthy controls and five patients assessed for familial HHT have been studied thus far. Available 100% oxygen breathing and bubble echocardiogram shunt study results were collated for HHT patients. Initial barometric pressure and then sequential pulse oximetry (S<sub>p</sub>O<sub>2</sub>) values after 3 minutes of stability were taken at F<sub>i</sub>O<sub>2</sub> values of 0.21, 0.24, 0.26, 0.28, 0.31, 0.35, 0.40, 0.50, and 1.0. XY plots of collective control and individual HHT P<sub>i</sub>O<sub>2</sub> versus S<sub>p</sub>O<sub>2</sub> values were fitted with curves using a Gompertz sigmoidal function. For illustration the collective control and a sample HHT patient's curves before and after malformation ablation were compared by visual inspection and an F-test.

**Results:** The collective control and individual HHT P<sub>i</sub>O<sub>2</sub>:S<sub>p</sub>O<sub>2</sub> curves were well fitted by the Gompertz sigmoidal function. Compared to the control curve there was progressive downward but not rightward displacement of the HHT patient's post- and pre-ablation curves corresponding to the known progression in shunt fraction. The combined control plus pre-ablation curve differed from the control curve (F 11.36, df1 50, df2 41, p < 0.05).

**Conclusion:** The P<sub>i</sub>O<sub>2</sub>:S<sub>p</sub>O<sub>2</sub> diagram is a simple, informative, non-invasive, and relatively inexpensive method to follow shunt in HHT patients.

**P2396**

**The lung microcirculation changes in patients with bronchial asthma**

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Changes of lung microcirculation were studied in 83 patients with exacerbation of the bronchial asthma by means of perfusion scintigraphy with Ts-99 m microspheres, under the influence of infusion glucocorticoid therapy. Diffuse and local defects of perfusion were revealed in 95, 6% (r<0, 05) of examined patients. They appeared as reduction of perfusion in lower lobes and its increase in upper (r<0, 05) zones, and, as well, redistribution of the microcirculation with its reduction in left lung (r<0, 05). We observed these changes even in patients with mild BA. The lung circulation disturbances were probably connected with inflammatory and bronchoobstructive syndromes as well as haemostasis system disturbances. Positive correlation was revealed between changes of lung microcirculation and increased coagulation activity, increased aggregation of erythrocytes, exacerbation of bronchial obstruction (data of FVD), and levels of Ig A, IgM and IgG, leukocyte

and neutrophil count as well as erythrocyte sedimentation rate. We observed the positive dynamic of scintigraphy picture in patients with BA after the glucocorticoid treatment course (90.2%). So the anti-inflammatory therapy (intravenous glucocorticoids) improved microcirculation due to decrease of inflammation and bronchoobstruction.

**P2398****Urinary uric acid excretion and overnight change in the urinary uric acid: creatinin ratio as a marker of tissue hypoxia in patients with COPD and OSA**

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**Objective:** Uric acid is ATP degradation end product due to hypoxia. In this study we aimed to evaluate the relationship between the hypoxia and urinary uric acid excretion (UUA/E) and the overnight change in the urinary uric acid: creatinin ratio ( $\Delta$ UA/Kr) in patients with OSA or COPD.

**Materials and methods:** Sixty patients had the diagnosis of OSA or COPD and 15 patients control group in this prospective study. OSA or COPD patients with an O<sub>2</sub> saturation lower than 90% existing at least 10% of night time were, subgrouped as hypoxic OSA or hypoxic COPD. Urine samples were collected twice, before and after PSG recordings, in order to calculate the UUA/E and  $\Delta$ UA/Kr ratio.

**Results:** UUA/E was significantly higher in the hypoxic group ( $0.72 \pm 0.19$ ) when compared with the nonhypoxic group ( $0.45 \pm 0.13$ ) and control group ( $0.37 \pm 0.14$ ) in COPD patients ( $p < 0.001$ ). Similar results were observed also for the OSA patients (hypoxic OSA:  $0.53 \pm 0.16$ , nonhypoxic OSA:  $0.36 \pm 0.08$ , control group  $0.37 \pm 0.14$ ;  $p < 0.001$ ).  $\Delta$ UA/Kr ratio was also significantly higher for hypoxic COPD and OSA patients ( $27.0 \pm 35.1$  and  $36.4 \pm 35.9$  respectively) when compared with the nonhypoxic groups ( $-4.2 \pm 11.7$  for COPD and  $-7.5 \pm 20.1$  for OSA) and control group ( $-13.5 \pm 23.6$ ) ( $p < 0.001$ ). UUA/E and  $\Delta$ UA/Kr both showed significant correlation with the duration of desaturation ( $\text{SaO}_2 < 90\%$ ) in all patients (UUA/E:  $p = 0.0023$ ,  $r = 0.58$ ,  $\Delta$ UA/Kr:  $p < 0.001$ ,  $r = 0.84$ ). **CONCLUSION:** As a result, we concluded that a simple and cheap method like can routinely be performed in order to assess tissue hypoxia especially in selected patient groups who do not have a disease or medical treatment that can affect UUA/E.

**P2399****The diagnostic value of exhaled carbonmonoxide and arterial carboxyhaemoglobin measurements in chronic obstructive pulmonary disease**

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In this study, it was aimed to show the value of COHb and COExh levels in assessing the severity of the disease in exsmokers with COPD.

44 patients and 13 controls were included in the study and their arterial COHb, venous COHb levels, (A-V)COHb gradients, COExh,  $\Delta$ CO (arterial CO-COExh) and FEV<sub>1</sub> were measured.

It was shown that the arterial COHb concentration was increased in stable COPD patients compared to controls ( $p < 0.001$ ). There was a statistically significant difference between the COPD stages IV, III and II regarding of the COHb concentration ( $P < 0.05$ ).

In COPD patients there was a negative correlation between COHb levels and %FEV<sub>1</sub> and PaCO<sub>2</sub>, COExh, venous COHb, (A-V)COHb and  $\Delta$ CO.

In COPD patients, the concentration of COExh was significantly higher compared to controls ( $p < 0.001$ ).

In COPD patients there was a negative correlation between COExh and %FEV<sub>1</sub>, PaO<sub>2</sub>, SaO<sub>2</sub> and a positive correlation between PaCO<sub>2</sub>, (A-V)COHb and venous COHb.

$\Delta$ CO values were higher in COPD patients compared to controls. There was a statistically significant  $\Delta$ CO difference in COPD patients only between II and IV stages. There was a negative correlation between  $\Delta$ CO values and %FEV<sub>1</sub> and a positive correlation between  $\Delta$ CO values and COExh.

We concluded that the COHb and COExh values in COPD patients can point out to the severity of the disease and COExh measurements may well be used for the assessment of airways inflammation and for oxidative-stress monitorization in stable exsmokers with COPD.

**P2400****Remarkable induction of brain natriuretic peptide in pulmonary emphysema after acute respiratory infection**

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Increase of plasma brain natriuretic peptide (BNP) is known to be a useful marker for right ventricular overload in COPD. Furthermore recent studies have confirmed that right ventricular hypertrophy is related to right ventricular induction of BNP and modulation of BNP receptors. Therefore the present study was carried out to elucidate which factor most contribute to core pulmonare by measuring the

quantity of plasma BNP in patients with pulmonary emphysema. We determine pulmonary emphysema by recognizing low attenuate areas on chest-computed tomography. Patients with heart disease and hypertension were excluded. We assessed pulmonary function by spirometer, hypoxia by analysis of arterial blood gas, serum C-reactive protein (CRP) as inflammatory marker by immunochemical assay and quantity of plasma BNP by immunoradiometric assay. The degree of increase in plasma BNP was significantly correlated with the increase of serum CRP. The value of CRP with cure declined and improved the value of serum BNP, too. Furthermore we found the tendency of increase of BNP accompanied with decrease of %predicted forced expiratory volume in 1sec value increase of alveolar-arterial oxygen tension gradient and oxygen desaturation of arterial blood. These results suggest that repeated airway inflammation on pulmonary emphysema which contribute to airway obstruction and hypoxia, promote induction of BNP and result in core pulmonare.

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