RELAXATION RATE OF MOUTH, NOSE AND OESOPHAGEAL PRESSURES DURING SNIFFS REFLECT RESPIRATORY MUSCLE FATIGUE

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The maximum relaxation rate (MRR, % force loss/10 ms) of mouth pressure during an inspiratory gasp is reduced with respiratory muscle fatigue (Levy et al. Am Rev Respir Dis 1984, 130: 381-384). We have recently shown that pressures in the mouth reflect oesophageal pressures during sniffs, a manoeuvre easily performed by patients (Koulouris et al, Clin Sci 1987, 86P). We have now investigated whether MRR of pressures generated in the mouth (Pmx), inopharynx (Pnp) and oesophagus (Poes) by voluntary sniffs decreases with inspiratory muscle fatigue. In 8 normal subjects we measured simultaneously Poes, Pnp and Pmx with balloon catheters. The naso and mouth balloons (5cm length) were positioned in the nasopharynx 10cm from the anterior nares and in the oral cavity with the lips closed without a nose clip. MRR was determined from 10 sniffs for Poes, Pnp and Pmx after fatigue. To produce inspiratory muscle fatigue, each subject breathed through an inspiratory resistance such that 80% of maximum inspiratory pressure was generated with each breath for as long as possible (5-6 mins). Following fatigue sniffs were performed during the first 30 secs and subsequently at 30 sec intervals to 10 min recovery. Artificialised blood samples were drawn during the entire study to ensure that there was no change in O2, CO2 and pH. Immediately following fatigue all subjects showed a decrease in MRR. For Poes this was 33% (range 20-42) from 9.97±0.46 (mean±SD) to 6.64±0.85, p<0.001; for Pnp 32% (range 20-42) from 10.15±0.53 to 6.93±0.77, p<0.001; and for Pmx 33% (range 21-42) from 10.20±0.65 to 6.84±0.59, p<0.001. All measures of MRR returned to normal by 10 mins. Studies were repeated in 3 subjects with similar results. We conclude that fatigue of the respiratory muscles in pressures measured in the mouth, nose and oesophagus during sniffs. These measurements may prove useful for detecting and monitoring respiratory muscle fatigue.

18 MEASUREMENT OF PULMONARY ARTERIOVENOUS SHUNT: COMPARISON OF TC-99m ALBUMIN MICROSPHERES WITH THE 100% OXYGEN METHOD

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Quantification of the right to left shunt in patients with pulmonary arteriovenous malformations (PAVMs) is important in assessing the effect of therapeutic embolization. Since in normal lung albumin particles do not pass through the pulmonary capillary bed passage into the systemic circulation, it reflects shunt fraction. Previous techniques using labelled macroaggregates in which lung activity has been compared with systemic activity have correlated poorly with physiological measurements. We describe an alternative approach using TC-99m albumin microspheres (TC-99mM 7.2-24um) for shunt quantification. Five male subjects with angiographically proven PAVMs were studied in the resting supine position. After breathing 100% oxygen for 20 min, PaO2, oxygen saturation (SO2) and IB were measured and used to calculate shunt from the classical equation. TC-99mM (110MBq) were injected i.v. while the patient remained supine breathing 100% oxygen. After 5 min. a timed perfusion gamma camera image was obtained and excitation of interest closely applied to the outline of the right kidney. Injected dose was obtained by subtracting pre- and post-injection activity in the syringe on the face of the gamma camera. After corrections for isotope decay, the fraction of injected dose in the right kidney was calculated using a depth correction based on a right lateral image. Assuming a right kidney blood flow of 10% of cardiac output the following shunt values were obtained for each patient: 5.8, 21, 29, 34 and 47%. Similar values were obtained using the oxygen method: 7.6, 19, 29, 29 and 48% respectively (r=0.995). This radiolabelling technique allows precise quantification of the right to left shunt over a wide range of values in patients with PAVMs.

17 THE EFFECT OF NEDOCROMIL SODIUM AND OXITROPIUM BROMIDE ON SODIUM METABISULPHITE INDUCED BRONCHOCONSTRICION

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Sodium metabisulphite (MBS) is a widely used preservative which can cause bronchoconstriction on ingestion. The mechanism of bronchoconstriction is unclear though SO2 liberation may be involved. We have investigated the effect of nedocromil sodium (N), which inhibits mast cell degranulation in vitro, and oxitropium bromide (OB), a muscarinic receptor blocker, both of which inhibit SO2-induced bronchoconstriction. Six asthmatic non-asthmatics were pre-treated with N (Amp), OB (200μg) or matched placebo (P) in a double-blind, randomised design. Thirty minutes later a dose of MBS that previously had been shown to cause 34.5% fall in specific airways conductance (sgaw) was inhaled and sgaw recorded, by computerised body plethysmography, for 20 min. Mean sgaw fell by 56.9% (meansSEM) 1 min after P, and by 35±20% after OB (P<0.05). Nedocromil prevented any significant fall (p<0.05) and in our mild asthmatics inhaling increasing doses of MBS until 1 greater than 35% fall in sgaw was achieved. On subsequent study day the drugs were administered, 30 min before a repeat MBS dose response, and the dose causing a 35% fall in sgaw (PD35) was calculated. Geometric mean PD35 was 8.39μmol after P (5.0-13.9μmol 95% confidence limits), 22.7μmol (12.9-39.8μmol) after OB and 79.4μmol (35.5-177.8μmol) after N. In 3 subjects the maximum fall after N pre-treatment was 12%; a PD120μmol was used for analysis. The mechanism of MBS-induced bronchoconstriction is the same in atopic and asthmatic subjects. Nedocromil is more effective than oxitropium in blocking the effect suggesting that mast cell degranulation may be involved.