Title: MINIMIZING INTRAOPERATIVE HYPOXEMIA DOES NOT AFFECT POST PNEUMONECTOMY LUNG GROWTH

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Objectives: Partial lung resection in rats and other small mammals has long been known to promote growth of the remaining tissue, such that lung mass is restored within 1 to 2 weeks. Understanding regulation of post-PNX lung growth may help to clarify the mechanisms which underlie severe lung growth retardation, as seen for example in infants with congenital diaphragmatic hernia, or which account for generation of abnormal tissue after other forms of lung injury. Initiation and control of this rapid hyperplastic compensatory growth after pneumonectomy (PNX) are not well understood, but post-resection hypoxemia has been proposed as an important factor. Increased lung size is a well-described adaptive response to a hypoxic environment in high-altitude natives; similar increases in size have been shown in animals raised in simulated high altitude environments. Nattie et.al.(1) showed mild and inconstant hypoxia in awake rats the day of PNX; by the following day the hypoxia had resolved. However, arterial blood gases (ABG) were not performed until 2 h after surgery. It is now known that biochemical changes associated with hyperplastic growth are measurable as soon as 3 h post PNX (2). The current studies examined the effects on post-PNX RL growth when the acute hypoxemia common during PNX was largely prevented.

Methods: Male Sprague-Dawley rats (284±2 g) were subjected to left PNX. Spontaneously ventilating (SV) animals breathed room air during surgery, with pharyngeal positive pressure ventilation (PPV) used only for resuscitation; this is the conventional approach. Intermittent PPV (IPPV) animals were intubated with a translaryngeal teflon catheter, and ventilated with air during PNX (PIP = 15 cm H_2O, PEEP = 2 cm H_2O, f = 70). Arterial oxygen saturation (SaO_2) was measured continuously during and immediately after the procedure with a pulse oximeter (Nellcor) placed over the femoral artery. Indirect SaO_2 values were confirmed by 3 direct SaO_2 determinations (Radiometer) before, during, and 5-8 min after thoracotomy; ABG were measured on the same sample (Corning).

Results: After induction of anesthesia, SaO_2 averaged 93±1% in both groups. At thoracotomy SV animals developed severe desaturation: SaO_2 was less than 50% in all animals for 2.6±0.5 min and less than 30% in all but one for 1.8 ± 0.4 min (Table 1.). Hypoxemia persisted until after pneumothorax evacuation. IPPV animals better maintained SaO_2: 8/13 had brief periods of moderate hypoxemia (0.3 ± 0.2 min SaO_2 < 50%) and only one was severely hypoxemic (0.7 min at SaO_2 < 30%). During the immediate post-operative period however, SaO_2 had returned to more than 90% of the pre-operative value in the SV group; but SaO_2 in IPPV animals declined further. Arterial samples correlated well with the pulse oximeter: the slope of the regression line (n=54) was 0.90 (corr. 0.91). Statistical analysis of ABG 2 min after thoracotomy showed decreases in pH and PO_2, and increased PCO_2 in SV vs. IPPV. Rats were killed and lungs weighed on postoperative day 14. There was no difference in RL weights (265±12 mg dry; mean ± SE of pooled data), RL wet weight to body weight (BW) ratio (387±14 mg/100g BW), lung dry to wet weight ratio (0.204±0.002), or tissue content of RNA (17.3±0.3mg.g^{-1} dry), DNA (2.42±0.07 mg.g^{-1} dry), or protein (566±8 mg.g^{-1} dry) between the two groups.

Conclusions: The results indicate that pulse oximetry is useful for non-invasive monitoring of arterial oxygen saturation in rats undergoing PNX, that tracheal intubation and IPPV effectively maintain oxygenation during PNX. The persistent post-operative desaturation seen in IPPV animals may represent mild airway edema. Lung mass was restored equally in both groups; the stable protein/DNA ratio is usually interpreted to mean that the response at the cellular level is hyperplastic. Avoiding acute hypoxemia during PNX does not affect compensatory lung growth; factors other than hypoxemia are likely to be more important in its initiation and control.

Support: American Lung Association of Pennsylvania, Parker B. Francis Foundation, HL 20344.

References:

1. Nattie EE, Wiley CW, Bartlett D: Adaptive growth of the lung following pneumonectomy in rats. J Appl Physiol 37(4): 491-495, 1974

2. Rannels DE, Addison JL, Bennett RA: Increased pulmonary uptake of exogenous polyamines after unilateral pneumonectomy. Am J Physiol 250 (Endocrinol Metab 13): E435-E440, 1986

EXPERIMENTAL GROUP	BODY WEIGHT g	SaO_2< 50% minutes	SaO_2< 30% minutes	RIGHT LUNG mg wet
SV	288 ± 3	2.6 ± 0.5	1.8 ± 0.4	1310 ± 93
IPPV	281 ± 3	0.3 ± 0.2[a]	0.05	1285 ± 60

Table 1. Intraoperative hypoxemia and lung weights after left PNX. Values are means ± S.E.M. of 8-15 observations. Spontaneous (SV) or positive pressure (IPPV) ventilation was performed as described above. Animals were sacrificed and their lungs removed 14 days after PNX. Differences between means are significant where indicated as follows:
[a] $p < 0.001$ vs. SV

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