Title : VENTILATION-PERFUSION EFFECTS OF NITROGLYCERIN

Authors : P.S. Colley, M.D., F.W. Cheney, M.D., M.P. Hlastala, Ph.D.

Affiliation: Department of Anesthesiology and the Anesthesia Research Center; Department of Medicine, Division of Respiratory Diseases and Department of Physiology and Biophysics, University of Washington School of Medicine, Seattle, WA 98195

Introduction. Previous studies[1] have indicated that nitroglycerin (NTG) interferes less with hypoxic pulmonary vasoconstriction (HPV) in a nitrogen ventilated lung than does sodium nitroprusside. NTG therefore, should produce little impairment of pulmonary gas exchange and be a safer drug to use particularly in patients with ventilation perfusion abnormalities. The purpose of this study was to examine the effects of iv NTG on PaO_2, $\dot{V}_A/\dot{Q}$ distribution, venous admixture ($\dot{Q}_{VA}/\dot{Q}t$) and pulmonary vascular resistance (PVR) in diffuse lung injury. In order to change the degree of HPV, both normal and high inspired oxygen concentrations were used.

Methods. Intravenous oleic acid was used to produce diffuse pulmonary injury in 11 dogs. Twenty-four hours following oleic acid injection, the animals were anesthetized with pentobarbital, intubated and mechanically ventilated. NTG was infused and mean arterial pressure (MAP) lowered by 33% first during ventilation at F_IO_2=.21 and then with F_IO_2=1.0. Measurements of vascular pressures, cardiac output ($\dot{Q}t$), blood gases, $\dot{V}_A/\dot{Q}$ distribution and shunt by the multiple inert gas elimination method were done before, during and after NTG infusion at both F_IO_2 levels.

Results. (See Table) During air ventilation, NTG caused a small decrease in PaO_2, a small increase in $\dot{Q}_{VA}/\dot{Q}t$ (oxygen method), a decrease in $\dot{Q}t$ and large and similar decreases in systemic vascular resistance (SVR) and PVR. There was no significant change in $\dot{V}_A/\dot{Q}$ maldistribution as evidenced by changes in the log standard deviation (SD) of the perfusion distribution. The shunt component (flow to completely unventilated lung) of venous admixture was also unchanged. There was no correlation between increases in $\dot{Q}_{VA}/\dot{Q}t$ and decreases in PVR (r=0.2711). A 36% decrease in PVR occurred when ventilation was initiated with F_IO_2=1.0 and confirmed the presence of HPV during F_IO_2=.21. During oxygen ventilation NTG caused the PaO_2 to fall slightly, but there was no significant change in $\dot{Q}s/\dot{Q}t$, $\dot{Q}t$ or PVR.

Discussion. During air ventilation, some of the PaO_2 decrease was due to the fall in $\dot{Q}t$. The hypothesis, however, that NTG decreases PaO_2, at least in part, by increasing $\dot{Q}_{VA}/\dot{Q}t$ was confirmed. In comparision, during oxygen ventilation when HPV was minimal, there was no change in $\dot{Q}s/\dot{Q}t$ with NTG. These results indicate that NTG increases $\dot{Q}_{VA}/\dot{Q}t$ by inhibiting HPV. The failure of NTG to significantly increase the inert gas shunt component and log SD perfusion may be because NTG is a relatively weak inhibitor of HPV[1]. The disproportionately large decrease in PVR during air ventilation, compared to the small increase in $\dot{Q}_{VA}/\dot{Q}t$, indicates that NTG also has a significant vasodilatory effect on pulmonary vessels perfusing normal lung areas.

Table

Air Ventilation	Pre		NTG		Post
$\dot{Q}t$ (L/min)	3.45 ±0.69	*	2.84 ±0.64	*	3.35 ±0.64
MAP (torr)	135 ±26	*	90 ±21	*	116 ±28
SVR (dyne. sec cm^{-5})	3252 ±557	*	2491 ±553		2921 ±694
PVR (dyne. sec cm^{-5})	502 ±187	*	372 ±103	*	546 ±195
PaO_2 (torr)	64 ±8	*	55 ±9		60 ±7
$\dot{Q}_{VA}/\dot{Q}t$ (%)	28 ±12	*	36 ±14		31 ±10
Shunt (%) Component	11 ±12		12 ±0		10 ±8
Log SD (perfusion)	.743 ±.188		.771 ±.150		.675 ±.122
100% Oxygen Ventilation					
	Pre		NTG		Post
$\dot{Q}t$ (L/min)	3.21 ±0.163		3.20 ±0.63		3.36 ±0.58
MAP (torr)	123 ±24	*	90 ±19	*	116 ±27
SVR (dyne. sec cm^{-5})	3118 ±603	*	2303 ±618	*	2813 ±754
PVR (dyne. sec cm^{-5})	358 ±136		297 ±138		336 ±169
PaO_2 (torr)	407 ±118	*	389 ±117		387 ±111
$\dot{Q}s/\dot{Q}t$ (%)	17 ±7		18 ±9		19 ±9
Log SD (perfusion)	.905 ±.452		.694 ±.317		.697 ±261

n=11, *=p<.05 between values, mean values ±SD

References:

1. Webster LR, et al.: Comparison of intravenous nitroglycerin and sodium nitroprusside on hypoxic pulmonary vasoconstriction. ASA Annual Meeting, Abstract, 1979, pp 67-68

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