Hyperventilation and Rehypnotization: A GasMan Simulation.
I: Effect of Hyperventilation

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Introduction:

- alveolar minute ventilation (V_A) should have less effect on emergence with less soluble agents (1)
- resurgent interest in the use of hyperventilation to hasten recovery from general anesthesia (2-4), but studies are flawed:
  - initial hypoventilation (2, 3) in control group
  - concomitant CO_2 administration causes hypercarbia (4)
  - hypercarbia by itself shortens emergence by increasing cerebral blood flow (5).
- we used GasMan® (MedManSimulations, Inc., Chestnut Hill, MA)
  - to further theoretically address the effect of hyperventilation on recovery
  - to compare the effects of hyperventilation on recovery with 3 different agents (isoflurane, sevoflurane, and desflurane) after prolonged anesthesia

Methods:

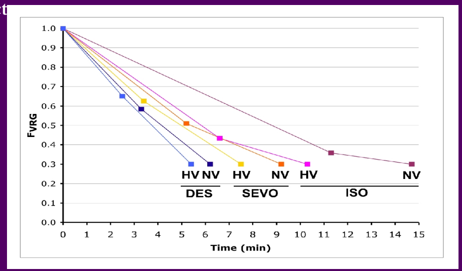
- GasMan® simulation
- 1 MAC of isoflurane, sevoflurane, and desflurane for 8 h
- normoventilation (5 L/min)
- saturation of the vessel rich group (VRG)
- near-saturation of the muscle group (MG)
- after 8 h, agent administration is stopped
  - fresh gas flow to 10 L/min to avoid rebreathing
  - wash-out with normoventilation (V_A 5 L/min) or hyperventilation (V_A 10 L/min)
- time for partial pressure in VRG (F_{VRG}) to reach 0.3 MAC (MACawake) is determined

Results:

Times to reach 0.3 MAC in the VRG:

	Normoventilation	Hyperventilation	Difference (abs.)	Difference (%)
Isoflurane	14 min 42 sec	10 min 18 sec	4 min 24 sec	30 %
Sevoflurane	9 min 12 sec	7 min 30 sec	1 min 42 sec	18 %
Desflurane	6 min 12 sec	5 min 24 sec	48 sec	13 %

Figure 1.



Discussion:

- our simulations confirm Eger's predictions
- hyperventilation is less effective with lower soluble agents
- hyperventilation in GasMan does not result in hypocarbia
  - similar to normocapnic hyperventilation in patients
  - normocapnic hyperventilation requires anesthesia circuit modifications
    - adding CO_2
    - rebreathing of CO_2 and activated charcoal to eliminate agent
  - hypocapnic hyperventilation reduces clearance of agent from the brain
    - decreased cerebral blood flow
    - prolongs emergence

Conclusion:

- normocapnic hyperventilation with desflurane or sevoflurane is theoretically of limited value
- clinical studies are required to confirm these findings

References:

1. Eger E. Anesthetic Uptake and Action, Williams & Wilkins, 1974, pp 228-48.
2. Vesely A et al. Br J Anaesth 2003;91:787-92.
3. Katznelson R et al. Anesth Analg 2008;106:486-91.
4. Sakata D et al. Anesth Analg 2007;105:79-82.
5. Gopalakrishnan N et al. Anesth Analg 2007;104:815-21.

Appendix: What determines emergence? F_{CNS} ≤ MAC awake (0.3 MAC)

1. Relationship between F_{CNS} and F_A

- GasMan®: $F_{CNS} = F_{VRG}$, and $F_A = F_a$ (F_a = arterial partial pressure)
- decrease in F_A determines decrease in F_{CNS} to ≤ MACawake
- there is no instantaneous equilibration: there is a time delay between F_a and FCNS
  - this is an exponential process
  - is determined by CNS's time constant (τ)
  - there is 95 % equilibration after 3 τ

$$\tau = \frac{volume}{flow} = \frac{capacity\ for\ agent}{amount\ of\ agent\ delivered\ to\ tissue} \quad ; \quad \tau_{CNS} = \frac{volume_{CNS} \times \lambda_{T/G\ CNS} \times F_A}{Q_{CNS} \times \lambda_{B/G} \times F_A}$$

- τ_{CNS} is shortest with desflurane: there is faster equilibration between F_{CNS} and F_A for desflurane

Figure 2.

Weight (kg)	70.0	FA des (%)	6.0%	FRC (L)	2.5
CO (L/min)	5.0	FA sevo (%)	2.0%	VRG (L)	6
VA (L/min)	5.0	FA iso (%)	1.2%		

	tissue/gas solubility λ des	λ sevo	λ iso	Volume mL	Blood flow % CO	Blood flow mL/min
CNS	0.54	1.15	2.09	1470	15.8	790
Liver	0.55	1.25	2.34	3990	30.0	1500
Heart	0.54	1.21	2.18	280	4.7	235
Kidneys	0.4	0.78	1.39	280	25.3	1265
Total VRG					*75.8*	*3790*
MG	0.94	2.4	4.5	33000	18.0	900
Fat	13	34	70	14500	6.0	300
Blood	0.42	0.65	1.3	5000		

desflurane	Capacity mL	Capacity FRC units	Agent transport mL/min	τ min
CNS	47.63	0.32	19.91	2.39
Liver	131.67	0.88	37.80	3.48
Heart	9.07	0.06	5.92	1.53
Kidneys	6.72	0.04	31.88	0.21
Total VRG	*195.09*	*1.30*	*95.51*	
MG	1861.20	12.41	22.68	82.06
Fat	11310.00	75.40	7.56	1496.03
Blood	126.00	0.84		
FRC	150.00			

sevoflurane	Capacity mL	Capacity FRC units	Agent transport mL/min	τ min
CNS	33.81	0.68	10.27	3.29
Liver	99.75	2.00	19.50	5.12
Heart	6.78	0.14	3.06	2.22
Kidneys	4.37	0.09	16.45	0.27
Total VRG	*144.70*	*2.89*	*49.27*	
MG	1584.00	31.68	11.70	135.38
Fat	9860.00	197.20	3.90	2528.21
Blood	65.00	1.30		
FRC	50.00			

isoflurane	Capacity mL	Capacity FRC units	Agent transport mL/min	τ min
CNS	36.87	1.23	12.32	2.99
Liver	112.04	3.73	23.40	4.79
Heart	7.32	0.24	3.67	2.00
Kidneys	4.67	0.16	19.73	0.24
Total VRG	*160.90*	*5.36*	*59.12*	
MG	1782.00	59.40	14.04	126.92
Fat	12180.00	406.00	4.68	2602.56
Blood	78.00	2.60		
FRC	30.00			

2. The rate of decrease in F_A depends on the amount of agent transferred from venous blood to the alveoli minus the amount of agent exhaled

- amount of agent returned to the alveoli with venous blood = $Q \times \lambda_{B/G} \times F_{ven}$
- when Q, $\lambda_{B/G}$, or F_{ven} is small, the amount of agent returned to the alveoli will be less
- F_{ven} is determined by the amount of agent released by the tissues, which is determined by τ_{tissue} and $F_{tissue} - F_a$ (the degree of saturation of the tissue)
- the VRG is washed out very quickly, and the fat group has a very long time constant, and therefore once F_{VRG} ≤ MACawake, only the MG contributes to F_{ven}
- the transfer of agent from venous blood to alveoli = $Q \times \lambda_{B/G} \times (F_{ven} - F_A)$
- the amount of agent that is exhaled = $V_A \times F_A$

Figure 3.