HBO AND CARDIAC FUNCTION

- Hypothesis: HBO should be beneficial to the failing or damaged heart
- HBO should:
  - Decrease cardiac output and work

References
- Savitt, 1994
- Hahnloser, 1966
- Eggers, 1962 (humans at 1 ATA)

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- Increase oxygen availability
- Increase survival after MI in dogs (Kuhn 1965)
- Questionable effects on left ventricular contractility:
  - No change--Savitt, 1994
  - Increase--Ishikawa, 1981
  - Decrease-- Kioschos, 1969

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- Increase afterload to damaged heart
  - Increased peripheral resistance

PR= (MAP- RAP)/CO

  - but no change in arterial pressure hence no change in afterload
- However, HBO is contraindicated in CHF because of reported cases of rapidly developing **pulmonary edema.**

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Measurement of Pulmonary Blood Volume

- Measure mean transit time of indicator (ascorbic acid
- $MTT = \frac{\int c(t) \cdot t\, dt}{\int (t)\, dt}$
- PBV = MTT * CO

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- End Systolic Elastance or ESPVR
  - First proposed by Sagawa, 1981
  - Independent of afterload, preload, amd heart rate
  - Has become the "gold standard" but
    - Requires measurement of pressure-volume loops
    - Requires major alteration in venous return or aortic pressure to measure change in Pressure-Volume loops

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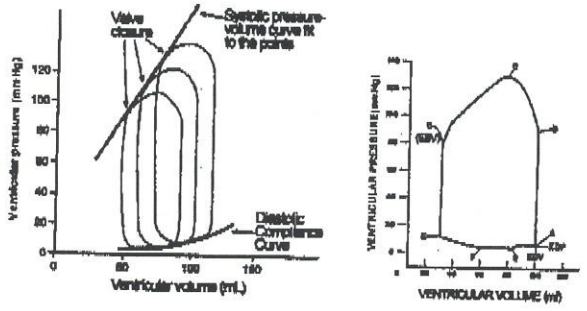
Pressure Volume Loops



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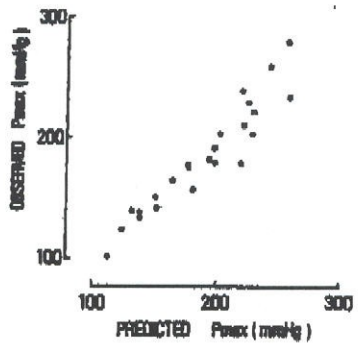
Sunagawa's Results



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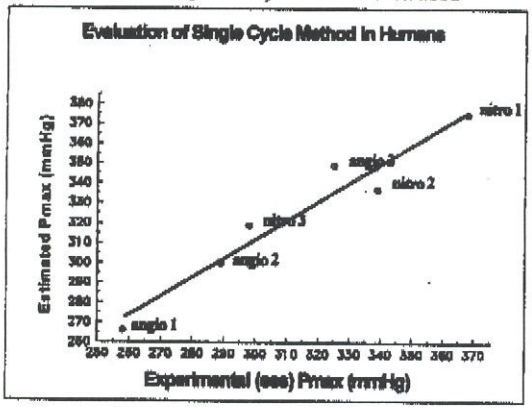
Takeuchi, 1991, Human Studies



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Our Modification

- Pmax = max dp/dt * Tau/ Pi

  Tau = cycle length

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HBO RESULTS

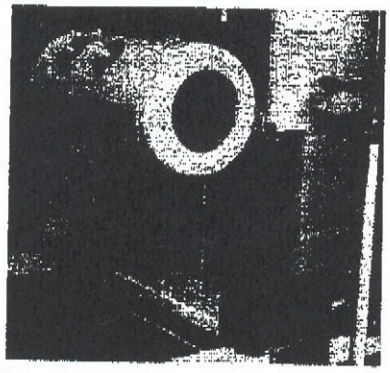

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FIG. 1. OVERALL HEMODYNAMIC EFFECTS

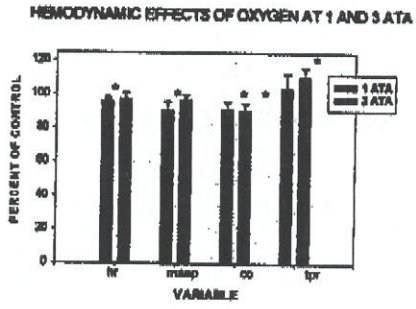

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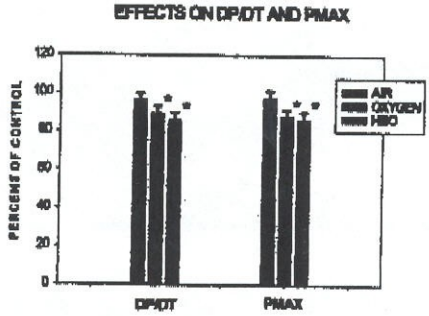
FIG . 2 EFFECTS OF HBO ON MAXIMAL DP/DT AND PMAX



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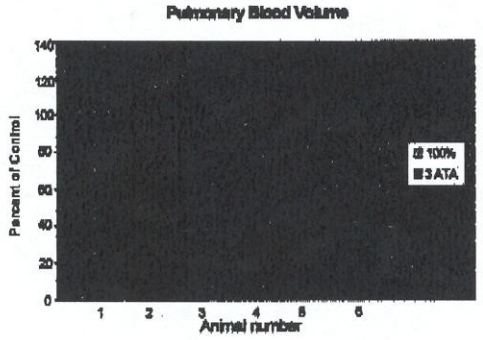
EFFECTS ON PULMONARY VASCULAR VOLUME



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CONCLUSIONS

- Left ventricular function is depressed with HBO.
- Right ventricular function is unchanged with HBO.
- Pulmonary edema may be due to ventricular imbalance--10 min of 2% imbalance= death
- While measured PBV did not change significantly, none of our animals were in CHF.

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