

maintained with 1–2% halothane in oxygen supplemented by pancuronium. During two-lung ventilation, the PaO_2 was 486.0 ± 104.0 mmHg. Shifting to OLV lowered the PaO_2 to 191.0 ± 144.0 mmHg ($P < 0.005$). Applying CPAP 10 cmH₂O to the nonventilated lung increased the PaO_2 to 353.0 ± 178.0 mmHg when 7 l/min of oxygen was used ($P < 0.025$), and to 355.0 ± 175.0 mmHg when 1 l/min of oxygen was used ($P < 0.01$); there was no significant difference between the PaO_2 levels whether 1 or 7 l/min of oxygen was used ($P > 0.2$).

The underwater seal assembly offers a major advantage when compared with other CPAP devices that include a pressure relief valve.^{3,4} The pressure relief valve functions by limiting the escape of the inflowing oxygen and thereby creates constant distending pressure to the nonventilated lung. However, untoward occlusion of the pressure relief valve, an increase of the oxygen flow, or lung manipulation may result in an excessive rise of the airway pressure. The threshold resistor valves offer a better alternative to the pressure relief valves,⁵ but different threshold resistor valves are required to achieve different CPAP levels. Also, both the pressure relief and the threshold resistor valves cannot function as an indicator for bronchial sealing. In contrast, the underwater seal is used as an indicator for bronchial sealing,⁶ as well as a CPAP valve which can be adjusted according to the required CPAP level. The CPAP level shows minimal fluctuations secondary to changes of the oxygen flow or lung manipulation.

Anesthesiology
65:103, 1986

On the Acceleration of Epinephrine Absorption by Lidocaine

To the Editor:—We were interested in the recent report by Ueda *et al.*¹ describing the effect of lidocaine on the absorption of epinephrine. The authors compared 0.5% lidocaine containing 1:200,000 epinephrine with 1:200,000 epinephrine alone. No mention was made of how these solutions were prepared.

Moore² has noted that commercially prepared local anesthetic solutions containing epinephrine have a low pH and are not as effective for vasoconstriction as freshly prepared solutions. Guyton³ states that low pH will cause vasodilation.

We found the pH of commercially available 0.5% lidocaine with 1:200,000 epinephrine (Astra) to be 3.78, whereas a 1:200,000 solution of epinephrine prepared by diluting 1:1000 epinephrine with preservative-free normal saline had a pH of 5. If Ueda *et al.* used solutions made in this fashion, the pH differences alone may have been responsible for the variation in epinephrine uptake attributed to the lidocaine.

ANIS BARAKA, M.D.
ABDEL NOUR SIBAI, M.D.
MUSA MUALLEM, M.D.
MAURICE BAROODY, M.D.
SANIE HAROUN, M.D.
TALAL MEKKAOUI, M.D.
*Department of Anesthesiology
American University of Beirut
Beirut, Lebanon*

REFERENCES

1. Capan LM, Turndorf H, Chandrakant P, Patel C, Ramanathan S, Acinapura A, Chalon J: Optimization of oxygenation during one lung anesthesia. *Anesth Analg* 59:847–851, 1980
2. Jenkins AV: An endobronchial cuff indicator for use in thoracic surgery. *Br J Anaesth* 51:905–906, 1979
3. Thiagarajah S, Job C, Rao A: A device for applying CPAP to the nonventilated upper lung during one-lung ventilation. I. *ANESTHESIOLOGY* 60:253–254, 1984
4. Hannenberg AA, Satwicz PR, Dienes JRRS, O'Brien JC: A device for applying CPAP to the nonventilated lung during one-lung ventilation. II. *ANESTHESIOLOGY* 60:254–255, 1984
5. Lyons TE: A simplified method of CPAP delivery to the nonventilated lung during unilateral pulmonary ventilation. *ANESTHESIOLOGY* 61:216–217, 1984
6. Alfery DD, Benumof JL: Anesthesia for thoracic surgery, *Anesthesia*, vol 2. Edited by Miller RD. New York, Churchill Livingstone, 1981, pp 925–980

(Accepted for publication February 6, 1986.)

MITCHEL SOSIS, M.D., PH.D.
Clinical Assistant Professor of Anesthesiology

H. THOMAS TEMPLE
Medical Student

*Jefferson Medical College
Thomas Jefferson University
Philadelphia, Pennsylvania 19107*

REFERENCES

1. Ueda NK, Hirawana M, Mori K: Acceleration of epinephrine absorption by lidocaine. *ANESTHESIOLOGY* 63:717–720, 1985
2. Moore DC: *Regional Anesthesia*. Springfield, CC Thomas, 1981, p 38
3. Guyton AC: *Textbook of Medical Physiology*. Philadelphia, WB Saunders, 1981, p 244

(Accepted for publication February 10, 1986.)