



FIG. 1. Schematic line drawing of expiratory pressure relief (EPR) valve components. During inspiration, the EPR valve becomes closed with a positive pressure through the connecting tubing so that the anesthetic circuit becomes a gas-tight system. During expiration, the excess gases are released passively through the EPR valve. Illustration by Elita E. Chang.

JAY J. CHOI, M.D.
Assistant Professor

JOSEPH GUIDA, M.D.
Resident

WEN-HSIEN WU, M.D., M.S.
Professor and Chairman

Department of Anesthesiology
UMDNJ-New Jersey Medical School
100 Bergen Street
Newark, New Jersey 07103

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Organization of an Emergency Airway Management Kit

To the Editor:—In addition to providing anesthesia in the operating room (OR), anesthesiologists are also called for emergency airway management outside of the OR. This includes the management of trauma victims in the emergency room and cardiac arrests in any of the other hospital care areas. Traditionally, anesthesia personnel have responded to such calls, taking with them a "tackle box" filled with equipment and drugs that they anticipated using. We have experienced delays in establishing airways because of the disarray of drugs and equipment in the box. Consequently, we designed an efficient case (fig. 1) in which equipment necessary for airway management is easily available.

Impressions of equipment and drugs were cut into foam rubber cushions, which were placed inside an aluminum attaché case. Because each item has a designated location as indicated by the foam rubber impression, users can easily determine if something is missing when the box is checked. The organization of equipment provided by this system has resulted in more efficient management of airway emergencies throughout the hospital.



FIG. 1. Aluminum attaché case for emergency airway management containing foam rubber cutouts for equipment and drugs.

M. C. S. JESUDIAN, M.D.
Instructor

MICHAEL ESTES, M.D.
Assistant Professor

Department of Anesthesiology
Medical College of Virginia
P. O. Box 695
Richmond, Virginia 23298

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Blood Gas Values Change Slowly in Apneic Organ Donors

To the Editor:—Brain-dead organ donors were ventilated with a fractional inspired O₂ concentration (F_IO₂) of 1.0 during bilateral nephrectomy, then disconnected from the ventilator. ECG evidence of death commonly occurred 15–20 min later. This time was longer than expected, and prompted a study exempted from Institutional Review Board review so long as anonymity of the patient was preserved. Samples were taken each minute after ventilation was discontinued, for a total of ten samples, from arterial lines in each of four donors. Endotracheal tubes were in place, open to room air. The PaO₂ values were used to calculate least-square regression lines. PaO₂ fell linearly with time, and the slopes were –17, –21, –27, and –28 mmHg PaO₂ per min for the four donors. Since the PaO₂ just before ventilation stopped was between 360–515 mmHg for these donors, hypoxemia (PaO₂ < 90 mmHg) did not develop nearly as quickly as would be true of living patients, such as those studied by Heller and Watson.¹ Those authors reported four patients whose PaO₂, during apnea after breathing 100% O₂, decreased at an average rate of 57.5 mmHg/min. Our donors differed from their patients in that several major sources of O₂ utilization were absent; the kidneys were removed before the blood was sampled, and the brains were surely capable of only minimal metabolism. The PaCO₂ rose slowly, at a mean rate of 1.7 mmHg/min, presumably for

the same reason. This may be of interest to those attempting to establish brain death by the apnea test described by Grenvik,² which requires that apnea be present when the PaCO₂ is greater than 60 mmHg and the PaO₂ less than 50 mmHg after discontinuation of mechanical ventilation. Although such a patient would still have kidneys, brain death could slow the PaCO₂ ascent and PaO₂ descent enough to make the wait for such values to be reached a long one, indeed, if the test were begun at hypocarbic, hyperoxic levels.

DAVID L. BRUCE, M.D.
Professor and Chairman
Department of Anesthesiology
University of Mississippi Medical Center
2500 N. State Street
Jackson, Mississippi 39216

REFERENCES

1. Heller ML, Watson TR Jr: Polarographic study of arterial oxygenation during apnea in man. *N Engl J Med* 264:326–330, 1961
2. Grenvik A: Brain death and permanently lost consciousness, *Society of Critical Care Medicine: Textbook of Critical Care*. Edited by Shoemaker WC, Thompson WL, Holbrook PR. Philadelphia, WB Saunders, 1984, p 969

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Ethacrynic Acid Interferes with Vasodilators

To the Editor:—Johnston *et al.*¹ report a case of apparent rebound hypertension following abrupt cessation of clonidine therapy. In the immediate postoperative period, their patient developed hypertension that was refractory to a number of pharmacologic interventions, including intravenous infusions of nitroglycerin and nitroprusside. By chance, one of the other therapeutic agents used to control the hypertension was ethacrynic acid. It is possible that the use of ethacrynic acid may have contributed to the lack of response to intravenous vasodilator therapy.

Many of the direct vasodilating agents act *via* a common intermediate reaction involving sulfhydryl groups within the vascular smooth muscle membrane.² Ethacrynic acid reacts with sulfhydryl groups and may block this common intermediate vasodilator reaction.³ This effect may cause a refractory response to vasodilator therapy.

If ethacrynic acid was administered to the patient prior to institution of intravenous vasodilator therapy, such therapy could account, in part, for the lack of response despite large doses of nitroglycerin and nitroprusside. In