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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 (FIO₂) of 1.0 during bilateral nephrectomy, then disconnected from the ventilator. ECG evidence of death commonly occurred 15-20 min later. This 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, hypoxemic levels.

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REFERENCES


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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