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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 O_2 concentration (FI_{O_2}) 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 Pa_{O_2} values were used to calculate least-square regression lines. Pa_{O_2} fell linearly with time, and the slopes were -17 , -21 , -27 , and -28 mmHg Pa_{O_2} per min for the four donors. Since the Pa_{O_2} just before ventilation stopped was between 360–515 mmHg for these donors, hypoxemia ($Pa_{O_2} < 90$ 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 Pa_{O_2} , during apnea after breathing 100% O_2 , decreased at an average rate of 57.5 mmHg/min. Our donors differed from their patients in that several major sources of O_2 utilization were absent; the kidneys were removed before the blood was sampled, and the brains were surely capable of only minimal metabolism. The Pa_{CO_2} 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 Pa_{CO_2} is greater than 60 mmHg and the Pa_{O_2} less than 50 mmHg after discontinuation of mechanical ventilation. Although such a patient would still have kidneys, brain death could slow the Pa_{CO_2} ascent and Pa_{O_2} 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.

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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

contrast clonidine, a relatively specific alpha-2 agonist that acts indirectly by reducing central nervous system sympathetic outflow, would not be blocked by the actions of ethacrynic acid.⁴ The mechanism for poor absorption in critically ill patients proposed by the authors may account for the ability of rectally administered clonidine to control hypertension in their patient when oral clonidine failed.

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In reply: We thank Dr. Hosking for his comments and concern that the use of ethacrynic acid may have interfered with the vasodilator actions of both nitroglycerin and nitropruside.

We reexamined our cases and noted that the first patient received ethacrynic acid, but as a secondary measure late in the course of managing her hypertension. None of the other five cases received ethacrynic acid. Therefore, we do not feel that ethacrynic acid interfered with the vasodilators in the first case. We will certainly keep this drug interaction in mind for future reference.

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Fail-safe Oxygen Analyzers

To the Editor:—Whenever nitrous oxide is supplied to an anesthesia circuit, there is a real possibility that a hypoxic mixture will be delivered secondary to human error in setting the flow controls. Less commonly, inaccurate or leaky flowmeters in conjunction with N₂O can lead to hypoxia. Use of an oxygen analyzer allows early detection of these errors but is often vitiated by another human error, failure to turn the analyzer on. A modification of

the anesthesia machine could guarantee that nitrous oxide will only be used when the oxygen analyzer is on.

An electromechanical fail-safe valve should be placed in the nitrous oxide intermediate pressure line. This valve would only be opened by a solenoid that is activated when the oxygen analyzer is turned on. Nitrous oxide could not be delivered unless the O₂ analyzer is turned on! If the machine is equipped with air or N₂, these lines should also have electromechanical fail-safes. Of course the low O₂ alarm must have a default preventing settings below 20%.¹ Perhaps the fail-safe analyzer should be further refined to shut the valves automatically whenever reading below 20%, thus allowing only oxygen to be delivered to

The opinions and assertions contained herein are the private views of the author and are not to be construed as reflecting the views of the Department of the Army or the Department of Defense.