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Preventing Delivery of Hypoxic Gas Mixtures

To the Editor:—The letter published by Richards¹ reports yet another way that the chain proportioning device coupling Modulus[®] flow control valves can fail with the possibility of delivering hypoxic gas mixtures; there are doubtless many more to report.

Of greater concern is the way in which the failure was discovered. This practice of depending on the device to increase oxygen flow while the operator increases nitrous oxide flow is one against which I have already commented in ANESTHESIOLOGY.² To use an extreme example, what if some calamity befell the anesthetist before the failure of the proportioning device was noticed. In such a case, the oxygen flow rate would not be increased and thus a "self created" hazardous situation would not be corrected.

Safety devices are perhaps misnamed if they lead us into habits revealed as unsafe when the devices fail. Safe technique uses devices only as back ups against the inevitable danger of operator error.

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In Reply:—Dr. Herbst misconstrued my description of in-use evaluation of a safety function of an anesthesia machine. My use of the proportioning device is founded on the expectation, not the assumption, of an increase in oxygen flow. This safety device malfunction¹ would lurk undetected in the face of prim adherence to a ranked sequence of flow control knob manipulation. Safe technique includes assessment of back-up devices in recognition of the multifactorial nature of anesthetic mishaps.² It is incongruous to recommend reliance upon safety devices only at the inopportune moment of operator error.

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Ventricular Tachycardia Associated with Injection of Prostaglandin F₂ α into the Uterine Cervix during Anesthesia

To the Editor:—The use of prostaglandin F₂ α to control postpartum hemorrhage due to uterine atony resistant to usual medical management has been found to be effective and generally safe.¹ We report a case in which injection of prostaglandin F₂ α into the uterine cervix during anesthesia resulted in life-threatening ventricular tachycardia that resolved by appropriate management.

CASE REPORT

A healthy, 30-yr-old, 64-kg woman presented with vaginal bleeding 1 week after spontaneous delivery. Upon examination, an enlarged uterus the size of 12 gestational weeks was found. Ultrasound exam-

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ination revealed a suspected retained placenta. Her history included an uneventful dilatation and curettage under general anesthesia 3 yr earlier during which oxytocin and methylergonovine had been administered without untoward effects. There was no history of cardiac disease. In the operating room, blood pressure was 130/80 mmHg and heart rate was 96 beats per min. Anesthesia was induced with methohexital (1.5 mg/kg) and fentanyl 0.1 mg, and maintained using small incremental doses (10-20 mg) of methohexital as needed. The trachea was not intubated and the patient spontaneously breathed a 66/33 N₂O/O₂ mixture. The blood pressure was 110/70 mmHg and the pulse was 86 beats per min. Cervical dilatation was unnecessary and during curettage placental residua were evacuated. The uterus

was atonic and bleeding continued despite uterine massage, iv oxytocin 10 IU, and methylergonovine maleate 0.2 mg. Fifteen minutes after induction of anesthesia, blood pressure was 100/60 mmHg and the pulse 96 beats per min. In an attempt to control bleeding prostaglandin F₂ α (Prostin F₂ α, Upjohn Limited, West Sussex) 2.5 mg was injected directly into the uterine cervix. One minute later the ECG showed bigeminy and trigeminy of premature ventricular beats, followed 30 s later by ventricular tachycardia. The blood pressure was 80/50 mmHg, arterial blood pH was 7.38, P_{O₂} 110 mmHg (F_IO₂ 0.33), P_{CO₂} 39 mmHg; base excess -1.5 mEq/l, HCO₃⁻ 23 mEq/l, O₂ saturation 98%, and hemoglobin 11.2 g %. Plasma electrolyte concentrations were within normal limits. Lidocaine 1 mg/kg was given iv, followed immediately by a second dose, and within 1 min the ECG reverted to normal sinus rhythm and stabilized, thereafter remaining within 10 mmHg of the baseline level. Administration of the prostaglandin F₂ α was followed by contraction of the uterus and cessation of hemorrhage. Following the procedure, the patient was alert, blood pressure remained within the normal range, and she was discharged the next day. A month later an electrophysiologic examination of the heart was normal.

Possible hazards of prostaglandin administration have been much discussed.^{2,3} Little doubt remains that many prostaglandins of the F series, and their structurally related synthetic analogues, exert diverse effects on the cardiovascular system.⁴ It is unlikely that any of the anesthetics used in the presented case was responsible for the arrhythmia. The arterial blood gas values and plasma electrolyte concentrations did not suggest hypoxia, hypercarbia, or electrolyte imbalance as etiologic factors. The onset of ventricular tachycardia almost immediately following the injection of prostaglandin F₂ α suggests the possibility that an inadvertent intravenous injection was wholly or partly accountable for this response. Physicians who use prostaglandins intravenously, intramyometrially, or intra-amniotically should be alert to the possibility of such a life-threatening complication and should have immediate access to resuscitation facilities.

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