requires a knowledge of the specific surgical technique used to place the venous cannulae and the exact site of drug injection. We report an incident involving a new type of Swan-Ganz catheter in which an infused drug did not enter the circulating bloodstream as expected.

A Paceport™ Swan-Ganz thermodilution catheter* was used in a patient who was undergoing coronary artery bypass grafting. This new catheter resembles the more commonly used venous infusion port ("VIP") catheter in that it has an extra port for drug infusion. Instead of the extra port exiting 30 cm from the distal end (normally in the right atrium), the extra port of a Paceport™ catheter exits at 19 cm, usually within the cavity of the right *ventricle.* This right ventricular port is designed to accept a separately packaged wire for ventricular pacing,† or it can be used to infuse drugs.

The right atrium of our patient was drained by a single venous cannula‡ during hypothermic CPB. While rewarming, hypertension (mean arterial pressure >100 mmHg) developed that did not respond to additional fentanyl (given into the oxygenator). A nitroprusside (NP) infusion was begun through the extra port of the Paceport™ catheter, and progressively increased to 3.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$ without apparent response. The blood pressure, however, decreased rapidly in response to 3 mg trimethaphan given into the oxygenator. When the aortic clamp was removed and the heart began to eject blood, a transient abnormal episode of severe hypotension (35 mmHg) occurred, which responded quickly to phenylephrine.

When a single right atrial cannula is used for CPB, a drug infused into the right atrium should quickly arrive at the pump and be circulated systemically. Unlike the situation with a "VIP" catheter, our patient's NP infusion was delivered to the right *ventricle,* not the right atrium. We hypothesize that the drug pooled inside the right ventricle, with the low right atrial pressure acting to keep the tricuspid valve closed. This would explain the patient's apparent resistance to the hypotensive actions of NP. When full CPB was changed to partial bypass and blood ejected from the heart, the pooled drug was flushed into the systemic circulation, causing the transient hypotension.

The problem we encountered with the Paceport™ catheter could also occur with a "VIP" or standard two-lumen catheter if the "atrial" infusion port has been advanced into the right ventricle; this might occur while floating the balloon into the wedge position in tall patients or those with dilated pulmonary arteries.

This potential complication is easily avoided by ensuring that all drugs during full CPB are delivered either: 1) to the bypass pump oxygenator; 2) to the superior vena cava; or 3) to the right atrium—but only if the bypass pump is withdrawing blood from this chamber.

This letter should serve to reemphasize the point that no drug should ever be administered to a patient without first examining exactly where it is being delivered. In our patient, infusion of drug into the pump oxygenator, the right atrial *monitoring* port, or the catheter introducer "side port" would have prevented this incident.

DONALD L. DODSON, M.D.
DAVID R. LARACH, M.D., PHD.
FREDERICK A. HENSLEY, M.D.

Department of Anesthesia
The Pennsylvania State University College of Medicine
P.O. Box 850
Hershey, Pennsylvania 17033

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* American Edwards Labs, Anasco PR 00610. Model no. 93A-931H-7.5F.

† American Edwards Labs, Chandler™ Transluminal V-Pacing Probe, Model 98-100H.

‡ Sarns, Inc., Ann Arbor, MI 48103. Model 12340.

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Early Warning Sign of an Accidental Endobronchial Intubation: A Sudden Drop or Sudden Rise in PA_{CO_2}?

To the Editor:—A recent case report endorses routine monitoring of end-tidal CO_2 wave forms as an early warning system of accidental endobronchial intubation.[1] The authors contend that during endotracheal (ET) anesthesia, a sudden rise in end-tidal CO_2 without changes in total CO_2 production in the body may be suggestive of inadvertent migration of the ET tube in the endobronchial position. Although they noticed that the end-tidal CO_2 went up from 4.6% to 6.1% within minutes after surgical manipulation made the tube move from the trachea to the left bronchus, their observation must be considered a very unusual one. With tidal volume maintained (as is the case with the volume-cycled ventilator), if an ET tube were to go down the main bronchus, the ventilation/per-

fusion (V/Q) ratio in the ventilated lung would almost double. Relationship of alveolar ventilation and PA_{CO_2} is $\dot{V}_A = \frac{\dot{V}_{CO_2}}{PA_{CO_2}} \times (PB - 47)$. At a constant CO_2 production, the equation can be simplified as $V_A = K/PA_{CO_2}$.[2] Thus alveolar P_{CO_2} and alveolar ventilation have an inverse relationship. Doubling the alveolar ventilation of one lung, as would happen with endobronchial intubation at least initially, will lead to a marked decrease in alveolar P_{CO_2} in the ventilated lung, increase in PA_{CO_2} in the nonventilated lung with a significant drop in arterial P_{O_2}, and minimal increase in PA_{CO_2}.[3] As mass spectrometry during accidental endobronchial intubation measures the alveolar CO_2 from the ventilated lung only, initially, it would show a marked *fall* in end-tidal CO_2.

What the authors have reported is a case of hypoventilation of a ventilated lung. Their suggestion that an acute elevation of PET_{CO_2} may result from an increase in dead space is not true. The immediate effect of increased physiologic dead space by endobronchial intubation, would be to lower the alveolar and expired CO_2 in that lung.[4]

Could reduced tidal volume to the ventilated lung be due to partial obstruction in an uncuffed tube? There is no mention in the report whether the authors used a volume-cycled or a pressure-cycled ventilator and what expired tidal volume was during one-lung ventilation. A use of a pressure-cycled ventilator with partial obstruction in an uncuffed tube could explain the reduced tidal volume in a ventilated lung and increased end-tidal CO_2.

We would like to emphasize that while using an end-tidal CO_2 monitor, an early warning sign of an accidental endobronchial intubation is a sudden drop in PA_{CO_2}* and not a sudden rise in PA_{CO_2}.

SHANTILAL K. GANDHI, M.D., F.F.A.R.C.S.
Associate Professor
Medical College of Wisconsin

C. A. MUNSHI, M.D.
Associate Professor
Medical College of Wisconsin

JOHN P. KAMPINE, M.D., PH.D.
Professor and Chairman
Department of Anesthesiology
Professor, Physiology
Medical College of Wisconsin
Milwaukee, Wisconsin 53226

* Jameson L: Clinical applications of mass spectrometry. American Society of Anesthesiologists Annual Refresher Course Lectures, 1985, p 223.

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In reply:—We agree that the first explanation offered in our report was an unlikely possibility. That is, unimpeded ventilation of one lung is generally associated with an initial fall in PE_{CO_2} (this may be missed when using a time-shared spectrometry system), a minimal rise in Pa_{CO_2}, and markedly increased airway pressure.

The incident we reported describes a case of hypoventilation of an intubated lung. We postulated that the position of the endotracheal tube in relation to the bronchial wall of the ventilated lung was such that resistance of the intubated lung was markedly greater than that of the alternative pathways for flow; namely, the upper airway, contralateral lung, and pressure-limiting valve of the ventilator. Because breath sounds could not be heard over the contralateral lung, we assumed gas was escaping to the atmosphere around the endotracheal tube in sufficient quantity to result in hypoventilation. However, since expired tidal volume was not measured during the course of the anesthetic, this explanation must remain speculative.

R. H. RILEY, M.B., B.S.
K. E. FINUCANE, M.B., B.S., F.R.A.C.P.
Departments of Anaesthesia and Pulmonary Physiology
Sir Charles Gairdner Hospital
Nedlands, Western Australia 6009

J. H. MARCY, M.D.
Department of Anesthesiology
Childrens Hospital of Pittsburgh
Pittsburgh, Pennsylvania 15213

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