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 thermomodulation 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 re-warming, hypertension (mean arterial pressure >100 mmHg) developed which 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 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) 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 systematically. 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.

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Early Warning Sign of an Accidental Endobronchial Intubation:
A Sudden Drop or Sudden Rise in \( P_{A_{CO_2}} \)?

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. 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, the CO\(_2\) rise (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 P\textsubscript{ACO\textsubscript{2}} is
\[ \dot{V}_A = \frac{\dot{V}_{CO_2}}{P_{ACO_2}} \times (PB - 47). \] At a constant CO\textsubscript{2} production, the equation can be simplified as \[ \dot{V}_A = K/P_{ACO_2}. \] Thus alveolar P\textsubscript{CO\textsubscript{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\textsubscript{CO\textsubscript{2}} in the ventilated lung, increase in P\textsubscript{ACO\textsubscript{2}} in the nonventilated lung with a significant drop in arterial P\textsubscript{O\textsubscript{2}}, and minimal increase in P\textsubscript{ACO\textsubscript{2}}. As mass spectrometry during accidental endobronchial intubation measures the alveolar CO\textsubscript{2} from the ventilated lung only, initially, it would show a marked fall in end-tidal CO\textsubscript{2}.

What the authors have reported is a case of hypventilation of a ventilated lung. Their suggestion that an acute elevation of PET\textsubscript{CO\textsubscript{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\textsubscript{2} in that lung.\textsuperscript{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\textsubscript{2}.

We would like to emphasize that while using an end-tidal CO\textsubscript{2} monitor, an early warning sign of an accidental endobronchial intubation is a sudden drop in P\textsubscript{ACO\textsubscript{2}} and not a sudden rise in P\textsubscript{ACO\textsubscript{2}}.

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REFERENCES


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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 P\textsubscript{ECO\textsubscript{2}} (this may be missed when using a time-shared spectrometry system), a minimal rise in P\textsubscript{ACO\textsubscript{2}}, and markedly increased airway pressure.

The incident we reported describes a case of hypventilation 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.

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