Anesthesiology
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Naloxone, Hypertension, and Ruptured Cerebral Aneurysm

To the Editor:—When used to antagonize narcotics, the administration of naloxone can cause hypertension,[1] pulmonary edema,[2] and cardiac arrhythmias.[3,4] Despite these reports and the manufacturer's[5] advice of careful titration of naloxone, the originally recommended dose of 0.4 mg is often given for reversing the respiratory depressant effects of narcotics.

We observed a sudden increase in arterial blood pressure from 150/90 to 260/140 torr which apparently caused rupture of a cerebral aneurysm, five minutes following naloxone reversal of fentanyl. The patient, whose hypertension had been medically controlled with hydralazine and chlorthalidone for ten years, was admitted with a diagnosis of a subdural hematoma. Computerized axial tomography (CAT scan) revealed an aneurysm of the anterior communicating artery. On admission, arterial blood pressure was 130/90 torr. Arterial blood pressure ranged from 110/70 torr to 130/90 torr during her hospitalization of one-month duration. Twenty-six days after her last bleeding episode, a repeat CAT scan indicated hydrocephalus, for which a ventriculoperitoneal shunt procedure was performed.

Anesthesia was induced with the intravenous (iv) administration of Innovar,® 3 ml, and thiopental, 200 mg. Pancuronium, 6 mg, was used to aid orotracheal intubation. Anesthesia was maintained with 66 per cent nitrous oxide and intermittent iv fentanyl, 0.05 mg, as needed during the two-hour procedure. Residual paralysis was reversed with 2.5 mg prostigmine and 1 mg atropine at the end of the procedure. Tidal volume and respiratory rate were less than 150 ml and 10/min, respectively. Naloxone, 0.4 mg, was given iv with an increase in tidal volume to 500 ml and respiratory rate to 20/min. Consciousness returned and she answered questions coherently. Blood pressure was 150/90 torr and heart rate 78/min immediately before transfer to the recovery room. Upon arrival in the recovery room she was still responsive to verbal stimuli; however, arterial blood pressure had increased to 260/140 torr. Heart rate was essentially unchanged at 84/min and respiratory rate increased to 32/min. She soon became unresponsive. The intravenous administration of hydralazine 20 mg, and morphine, 3 mg, decreased blood pressure to 150/90 within 30 min. Four hours later, she was alert and coherent but had developed a right hemiparesis. A repeat CAT scan revealed rebleeding of the aneurysm.

The recommended dose of naloxone for rapid awakening is 1 μg/kg, iv, which has not been associated with undesirable effects. This recommended dose is considerably smaller than that given in our case. The pressor effect of naloxone following opiate pretreatment has been reported by Dashwood[6] in cats. Because of this and the previous clinical reports,[1–4] doses of naloxone larger than 1 μg/kg should be given with caution, especially to known hypertensive patients.

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REFERENCES

1. Tanaka GY: Hypertensive reaction of naloxone. JAMA 228: 25–26, 1974
2. Flacke JW: Acute pulmonary edema following naloxone reversal to high dose morphine anesthesia. ANESTHESIOLOGY 47:376–387, 1977
3. Michaelis LL, Hicky PR, Clark TA, et al: Ventricular irritability associated with the use of naloxone hydrochloride. Ann Thorac Surg 18:608–614, 1974
4. Azar I, Turndorf H: Severe hypertension and multiple atrial premature contractions following naloxone administration. Anesth Analg (Cleve) 58:524–525, 1979
5. Endo Lab. Drug Insert since 1976
6. Dashwood MR: A pressor response to naloxone: Evidence for release of endogenous opiod peptide. J Physiol (Lond) 281:30P–31P, 1978

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Technique for Changing Endotracheal Tube is Neither New nor Better

To the Editor:—The technique for changing endotracheal tubes described by Desai and Fencl[1] had been previously described and published by our group in 1978[2]. As can be seen in our previous publication, it is not necessary to cut off "as much as possible" of the existing tube, thus, avoiding the risk of "losing" the

remaining tube in the pharynx. In addition, the tube changer we described is of such a diameter that it provides an adequate lumen through which the patient can ventilate while the tubes are being exchanged. The tube changer we described, which is commerically available,* is graduated so that its depth within the trachea and endotracheal tube can be determined and therefore, we have found that new tubes can be properly positioned without requiring an additional radiograph.

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REFERENCES

1. Desai, S. P., Fencl, V.: A safe technique for changing endotracheal tubes. ANESTHESIOLOGY 53:267, 1980
2. Millen, J. E., Glauser, F. L.: A rapid, simple technique for changing endotracheal tubes. Anesth Analg (Cleve) 57: 735–736, 1978

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Simplified Pulmonary Artery/Right Atrial Pressure Monitoring System

To the Editor:—Triple lumen and quadruple lumen, thermodilution, flow-directed, pulmonary-artery catheters permit simultaneous measurement of pulmonary artery (PA) pressure (PAP) and right atrial pressure (RAP) or central venous pressure (CVP). Pulmonary capillary wedge pressure (PCWP) can also be measured by inflating the balloon on the catheters. Because of physiologic noise caused by patient movement and by respiratory variations, these pressures can seldom be measured continuously. To prevent pulmonary embolism, the balloon is inflated for only a short time, thus precluding continuous reading of PCWP. We have developed a simple method which makes it possible to measure these pressures with one transducer/amplifier system. The system continuously flushes both the PA and CVP catheter lumens. Switching from measurement of CVP to PAP requires 90° rotation of a single stopcock. The mechanism is simpler and less expensive than a similar one proposed by Kotter.[1]

The system is outlined in figure 1. Stopcock 1 (a conventional 3-way stopcock) connects either the PA catheter lumen or the CVP catheter lumen to the pressure transducer. In position "A" the transducer is connected to the CVP lumen, while in the more common "B" position the transducer is connected to the PA lumen. Both lumens of the catheter are continuously flushed with two devices which can be connected to a single pressurized fluid source. Stopcock 1 can be connected to the CVP lumen by a 3-in piece of male-male pressure tubing or by using preassembled, sterile, single-use kits on which the tubing connection at stopcock 1 is bonded.

Stopcocks 2 and 3 are conventional 3-way stopcocks.

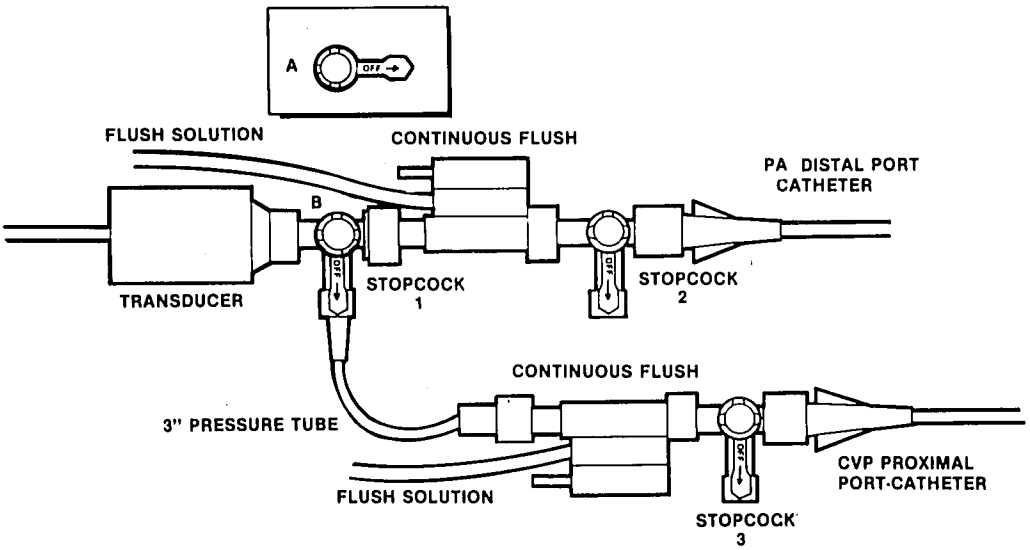

FIG. 1. Diagram of a simplified pulmonary artery/right atrial pressure monitoring system.