

These methods include: (1) use of ventilation-perfusion scanning to guide in the choice of initial lavage site, (2) use of the fiberoptic bronchoscope and an airway leak detector to effect precise separation of the two lungs, and (3) use of temporary unilateral pulmonary artery balloon occlusion to improve oxygenation.

REFERENCES

1. Kylstra JA, Rausch DC, Hall KD, Spock A: Volume controlled lung lavage in the treatment of asthma, bronchiectasis and mucoviscidosis. *Am Rev Respir Dis* 103:651-662, 1971
2. Ramirez-RJ, Kieffer RF Jr, Ball WC Jr: Bronchopulmonary lavage in man. *Ann Intern Med* 63:819-828, 1965
3. Selecky PA, Wasserman K, Benfield JR, Lippman, M: The clinical and physiological effect of whole lung lavage in pulmonary alveolar proteinosis: A ten year experience. *Ann Thorac Surg* 24:451-461, 1977
4. Rogers RM, Tantam KR: Bronchopulmonary lavage: A "new" approach to old problems. *Med Clin North Am* 54:755-771, 1977
5. Kao D, Wasserman K, Costley D, Benfield J. Advances in the treatment of pulmonary alveolar proteinosis. *Am Rev Respir Dis* 111:361-363, 1975
6. Smith JD, Miller JE, Safer P, Robin ED: Intrathoracic pressure, pulmonary vascular pressures and gas exchange during pulmonary lavage. *ANESTHESIOLOGY* 33:401-405, 1970
7. Tisi GM, Landis GA, Miale A Jr, Moser KM: Quantitation of regional pulmonary blood flow: Validity and potential sources of error. *Am Rev Respir Dis* 97:843-850, 1968
8. Altose MD, Hicks RE, Edwards MW Jr: Extracorporeal membrane oxygenation during bronchopulmonary lavage. *Arch Surg* 111:1148-1153, 1976
9. Cooper JD, Duffin J, Glynn MF, et al: Combination of membrane oxygenator support and pulmonary lavage for acute respiratory failure. *J Thorac Cardiovasc Surg* 71:304-308, 1976
10. West JB: *Ventilation/Blood Flow and Gas Exchange*. Oxford, Blackwell Scientific Publications, 1970, p 25

Anesthesiology
57:538-539, 1982

Slowing of Heart Rate during Cardiac Output Measurement by Thermodilution

TOSHIAKI NISHIKAWA, M.D.,* AND SHUJI DOHI, M.D.†

With measurements of cardiac output and pulmonary hemodynamics using the flow-directed balloon-tipped (Swan-Ganz®) catheter, several complications¹ have been described which usually are related to the insertion of the catheter, but not to the measurement of cardiac output by thermodilution *per se*. However, Weisel *et al.*² observed a transient arrhythmia following the injection of cold injectate. We recently observed a case in which bradycardia developed abruptly following the injection of the cold tracer during measurements of cardiac output.

REPORT OF A CASE

A 52-year-old, 40 kg woman underwent a craniotomy for removal of a brain tumor. Her past history included pulmonary tuberculosis 30 years ago, and previous surgery for a brain tumor four years ago. ECG and other laboratory data revealed no abnormality.

One hour before anesthesia, 0.4 mg atropine was administered im. Anesthesia was induced with 250 mg thiamylal, iv, and the trachea intubated after administration of 50 mg succinylcholine, iv. During

anesthesia with halothane, nitrous oxide, and controlled ventilation, a 7F thermistor-tipped, flow-directed (Swan-Ganz®) catheter was inserted percutaneously through the right internal jugular vein to monitor the cardiopulmonary status (the *in vivo* catheter length was 40 cm). A computer (9520, Edwards Laboratory) was used to determine cardiac output by the thermodilution technique. A second catheter was inserted into the left radial artery for monitoring of arterial blood pressure and blood sampling for blood-gas analysis.

Cardiac output determined two hours after the induction of anesthesia, was 3.34 l/min. At this time, heart rate was 72 beats/min, arterial blood pressure 100/60 mmHg, pulmonary arterial pressure 23/10 mmHg, right atrial pressure 9.0 cm H₂O, pulmonary capillary wedge pressure 8.0 mmHg. The *pH_a* was 7.41, PaCO₂ 37 mmHg, PaO₂ 163 mmHg, and BE 0.7 mEq/l. The rectal temperature was 36.9° C.

One hour later, however, when 10 ml of cold 5% dextrose solution were injected into the central venous port of the catheter, the heart rate, which was calculated electronically, decreased suddenly from 68 to 60 beats/min and then shortly returned to its previous level (fig. 1A). Two sequential determinations of cardiac output produced similar changes (fig. 1B and C). On these occasions, the values of cardiac output measured were 4.38, 4.32, 4.08 l/min, respectively. Arterial and pulmonary arterial pressure showed little change (fig. 1A, B, C) and the ECG did not change with the exception of transient slowing of normal sinus rhythm.

Thereafter, 10 ml of 5% dextrose in water (2° C) were injected into the distal port of the catheter. No change as appeared above occurred, however. Although 10 ml of 5% dextrose solution (37° C) were again injected into the central venous port of the catheter, no change in heart rate occurred. In one of the last series of measurements, decreases in both the radial artery and pulmonary artery pressure as well as a decrease in heart rate occurred at the time of the injection of the cold tracer (fig. 1D). The value of cardiac output was 2.74 l/min. The catheter was then retracted about 3 cm. The above episode did not occur again.

* Instructor.

† Associate Professor of Anesthesiology, Institute of Clinical Medicine, The University of Tsukuba, Sakuramura, Ibaraki, 305.

Received from the Department of Anesthesiology, Sapporo Medical College and Hospital, Chuoku, Sapporo, Hokkaido 060, Japan. Accepted for publication June 22, 1982.

Address reprint requests to Dr. Dohi.

Key words: Equipment: catheters, flow-directed. Complications: bradycardia.

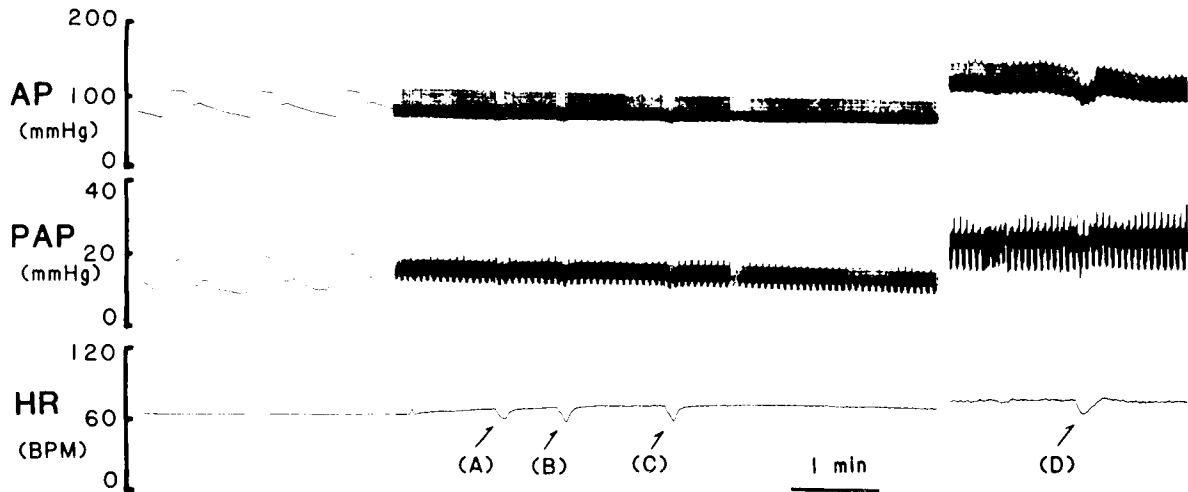


FIG. 1. Polygraph tracings of arterial blood pressure (AP), pulmonary arterial pressure (PAP) and heart rate (HR). At A, B, C, and D, 10 ml of the cold 5% dextrose solution were injected into the right atrium through the proximal orifice of the Swan-Ganz¹ thermodilution catheter. Note the transient decrease in heart rate at each injection. For further discussion, see the text.

DISCUSSION

Transient arrhythmias during cardiac output measurements using the thermodilution technique have been described previously.² In our case, we definitely observed a series of transient bradycardias related to injections of cold injectate. Local cooling of the sinus node could cause slowing of the sinus pacemaker, since warming the sinoatrial node increases its spontaneous activity and slope of diastolic depolarization.³ We could not, however, find any clinical study to substantiate this possibility. Experimentally, Marshall⁴ found in single fibers of the rabbit atrium that although pacemaker fibers were the most resistant to cooling, slowing of the atrial rate in association with a decreased rate of diastolic depolarization and a fall in diastolic membrane potential occurred as the temperature declined. When 10 ml of cold solution are injected into the right atrium, the temperature of the sinus node should decrease considerably because the central venous port of the catheter should lie near the sinus node. Therefore, the phenomenon described above seems to have been due to slowing of the sinus pacemaker discharge induced by local cooling with the cold dextrose solution.

Since migration of the catheter with the pulsatile propelling force of the blood flow can occur,⁵ no bradycardia observed at the first measurement might be related to this migration of the catheter which probably caused bradycardia in subsequent determinations of cardiac output. Bradycardia during cardiac output measurement using thermodilution method has not been previously described; however, it is possible that we could have missed this occurrence without the direct write out. Additionally, with the premedication of atropine, bradycardia, which could be a vagotonic response, did occur in our case; this failure to suppress the va-

gotonic response could be related to the small dose of atropine given.⁶

The first three events in the present case showed few changes in arterial and pulmonary artery pressure, but the last showed a significant decrease in arterial blood pressure as well as in heart rate (fig. 1D). This, incidentally, seems to be due to excitement of the vagal receptors in the heart which is accompanied by bradycardia and arterial hypotension, largely due to systemic vasodilatation and resembling the Bezold-Jarisch reflex.⁷ Although the described cardiovascular changes may be rare, heart rate should be monitored when cold tracer is injected during cardiac output measurements using the thermodilution method. Conceivably, cardiac output measurements also could be transiently affected by the cardiovascular change induced by cold tracer.

The authors thank Dr. Maki Matsumoto, Department of Anesthesiology, Yale University School of Medicine, for his assistance.

REFERENCES

1. Elliott CG, Zimmerman GA, Clemmer TP: Complications of pulmonary artery catheterization in the care of critically ill patients. A prospective study. *Chest* 76:647-652, 1979
2. Weisel RD, Berger RL, Hechtman HB: Measurement of cardiac output by thermodilution. *N Engl J Med* 292:682-684, 1975
3. Smith JJ, Kampine JP: *Circulatory Physiology—the Essentials*. Baltimore, Williams & Wilkins, 1980, p 83
4. Marshall JM: Effects of low temperatures on transmembrane potentials of single fibers of the rabbit atrium. *Cir Res* 5:664-669, 1957
5. Benumof JL, Saidman LJ, Arkin DB, Diamant M: Where pulmonary arterial catheters go: Intrathoracic distribution. *ANESTHESIOLOGY* 46:336-338, 1977
6. Churchill-Davidson HC: *A Practice of Anaesthesia*. 4th edition. London, Lloyd-Luke Ltd, 1978, p 801
7. Mancia G, Lorenz RR, Shepherd JT: Reflex control of circulation by heart and lungs. *International Review of Physiology. Cardiovascular Physiology II, Volume 9*. Edited by Guyton AC, Cowley AW. Baltimore, University Park Press, 1976, pp 111-165