CLINICAL REPORTS

Ronald D. Miller, M.D., Editor

Anesthesiology
61:454–456, 1984

Intraoperative Hypoxia and Hypotension Caused by Gastrografin-induced Hypovolemia

FARROKH R. MANEKSHA, M.B., B.S.,* JOANN BETTA, M.D.,* MARLENE ZAWIN, M.D.,†
PAUL J. POPPERS, M.D.‡

Uncomplicated meconium ileus can be treated with gastrografin enemas.[1,2] We describe an infant who developed severe hypoxemia following intestinal administration of gastrografin during an exploratory laparotomy.

REPORT OF A CASE

A 10-day-old, 2,900-g male infant born after 39 weeks of gestation was scheduled for an exploratory laparotomy, for necrotizing enterocolitis. After birth, ventilatory support was required for respiratory distress. He had since improved, not requiring such support. Because he had become increasingly jaundiced, biliary atresia was suspected. Also the additional diagnosis of cystic fibrosis was confirmed by abnormal sweat electrolytes. To allow proper removal of the inspissated secretions from the upper airway, trachea was reintubated with a 3.0-mm tube in the neonatal intensive care unit.

An iv infusion of 10% dextrose in a solution containing sodium 25 mEq/l, potassium 20 mEq/l, magnesium 3 mEq/l, chloride 24 mEq/l, lactate 23 mEq/l, and phosphate 3 mEq/l (D10 E_{48} Solution) was in progress at a rate of 12 ml/h via a 22-gauge cutdown catheter in the right saphenous vein when the infant was brought to the operating room. The preoperative hematocrit was 32% and serum electrolytes were as follows: sodium 132 mEq/l, potassium 4.2 mEq/l, chloride 98 mEq/l, and bicarbonate 22 mEq/l.

The infant was monitored with a precordial stethoscope, an electrocardiograph, an automatic oscillometric blood pressure monitor, a rectal temperature probe, and a transcutaneous oxygen tension ($P_{tc}O_2$) monitor. Anesthesia was induced with a mixture of air, oxygen, and halothane. All gases were humidified, warmed, and delivered via a Bain® circuit connected directly to the endotracheal tube.

During exploration of the abdomen, there was no evidence of ischemic changes in the wall of the intestine or of peritonitis. The diagnosis of meconium ileus with microcolon was made. An enterostomy was made in the distal ileum for evacuation of the meconium with gastrografin. Approximately 70 ml undiluted gastrografin was instilled into the lumen. Large quantities of meconium subsequently were removed.

Until this time, the anesthetic course had been unremarkable. Blood loss had been replaced continuously during the procedure with 1 ml packed erythrocytes and 1 ml of 0.9% saline solution for each 2 ml of blood lost. Periodic tracheal suctioning was performed during the procedure and ventilation was easily maintained keeping the $P_{tc}O_2$ between 60 and 80 mmHg.

A few minutes after the meconium was removed the $P_{tc}O_2$ fell from 80–15 mmHg. This was followed by a decrease in systolic blood pressure from 55 to 30 mmHg. There was no excessive bleeding, decrease in lung compliance, or any other difficulty in ventilation. Halothane administration was discontinued and ventilation controlled with a $F_{I_{O_2}}$ of 1.0. Pancuronium 0.2 mg was injected iv to prevent the infant from moving. These measures did not produce any improvement in arterial blood pressure or $P_{tc}O_2$.

Intravenous fluid administration then was increased; in the course of 15 min, 50 ml 5% dextrose in lactated Ringer's solution and 50 ml 5% albumin were infused. Subsequently, both $P_{tc}O_2$ and arterial blood pressure quickly returned to previous levels. Halothane anesthesia was resumed and was well tolerated for the remainder of the procedure. Total blood loss was estimated at 140 ml and was replaced intraoperatively with 72 ml packed erythrocytes. Total fluids given over 3 h of surgery included 50 ml of D10 E_{48} solution, 75 ml of 0.9% saline solution, 50 ml of 5% dextrose in lactated Ringer's solution, and 50 ml of 5% albumin. The postoperative hematocrit was 38% and serum electrolytes were as follows: sodium 148 mEq/l, potassium 3.5 mEq/l, chloride 112 mEq/l, and bicarbonate 18 mEq/l.

The postoperative course in the anesthesia intensive care unit was uneventful, and the patient thereafter was transferred to the neonatal intensive care unit where controlled ventilation was continued for 24 h.

DISCUSSION

Several factors could explain the hypoxia and hypotension. Cystic fibrosis is notorious for the thick viscous secretions that are usually present in the respiratory tract. They frequently are the cause of pulmonary shunting, hypoxemia, and hypotension. To prevent this, anesthetic gases were warmed and humidified and frequent suctioning of the trachea performed. When the episode of hypoxemia and hypotension occurred, there was no change in lung compliance or breath sounds.

With necrotizing enterocolitis, sepsis and hypovolemia are often part of the clinical picture that may include a fall in $P_{tc}O_2$ and hypotension. However, this preoperative diagnosis was ruled out after exploration of the abdomen.

Hypotension and decreased peripheral perfusion could occur if iv fluid management was improper or blood

* Assistant Professor, Department of Anesthesiology.
† Resident, Department of Radiology.
‡ Professor and Chairman, Department of Anesthesiology.

Received from the Departments of Anesthesiology and Radiology, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, New York 11794. Accepted for publication February 24, 1984.

Address correspondence to Dr. Maneksha.

Key words: Anesthesia: pediatric. Complications: hypotension, hypovolemia. Gastrografin.

loss was not replaced adequately. Again, this explanation is not a likely one in view of the careful calculation of normal fluid requirements and of the evaporative and third space losses during surgery. Blood loss was measured meticulously and replaced intraoperatively. However, the immediate and favorable response in $P_{tc}O_2$ and arterial blood pressure to additional iv fluid administration indicates that hypovolemia did exist.

The most likely cause of the intraoperative episode of severe hypovolemia is the use of the gastrografin. Gastrografin is a water-soluble iodinated contrast agent for radiographic examination of the gastrointestinal tract. It is composed of a 76% aqueous solution of methylglucamine diatrizoate, 0.1% Tween 80, and 37% firmly bound iodine. Relatively nontoxic when introduced into body cavities, gastrografin is preferable to barium in situations where a perforation is suspected.

In neonates with meconium ileus, it is the contrast agent of choice as it is a totally miscible solution and therefore does not concretize behind an obstruction as does barium. Gastrografin usually is introduced per rectum in the radiologic suite under fluoroscopic guidance. If it is seen to flow through a narrow, unused colon, to reflux around inspissated meconium in the ileum, and then to enter the dilated proximal bowel, the diagnosis of meconium ileus is established. In the absence of atresia, volvulus, perforation, or peritonitis, the ileus is considered uncomplicated; the procedure in this circumstance has a therapeutic as well as a diagnostic role. The gastrografin promotes the expulsion of the meconium by the infant and thus serves as a nonoperative alternative in the reduction of the obstruction.

The features that enable gastrografin to be a therapeutic enema agent are its physical nature and the chemical properties of its three major components.[3] Since gastrografin is a water-soluble, miscible solution, it is able to flow freely through the microcolon when introduced into the bowel. When it reaches the area of obstruction it can pass through and around the bowel contents and in so doing, soften the tenacious meconium and create an interphase between the intestinal mucosa and the meconium. The meconium then is dislodged from the bowel wall, thus facilitating its expulsion.

Methylglucamine diatrizoate has an osmolality of 1,730 mOsm/l, making it the major contributor to the total osmolality of gastrografin, which is 1,900 mOsm/l. When gastrografin is introduced into the bowel, a high osmotic gradient is created that draws fluid into the intestinal lumen from the vascular compartment, resulting in further softening and loosening of the meconium.[4]

Tween 80 or Polysorbate 80 is the second major component of gastrografin. It is an oily and slippery saturated fat that also softens the bowel contents. It is a surface-active agent and so facilitates the passage of the gastrografin through and around the thick tenacious meconium, resulting in its expulsion per rectum by the infant.[3,5] The iodinated portion of this solution is responsible for its radiopacity, which permits the performance of the examination in the radiographic suite under fluoroscopic guidance. Thus, the amount of gastrografin administered, the level to which it has been introduced, and the amount that is present in the bowel following evacuation all can be evaluated.

That a gastrografin enema can result in acute hyperosmolar dehydration in these infants is explainable on the basis of two mechanisms. The first is the high osmotic gradient that is created, drawing iv fluid into the bowel lumen and causing a sudden increase in serum osmolality and hematocrit, as well as a significant decrease in the cardiac output and heart rate.[4,6] The second mechanism involves the absorption of osmotically active solutes found in gastrografin by the intestinal mucosa and their introduction into the vascular compartment, causing an osmotic diuresis.[7]

In addition to the systemic hazards, local changes can occur following the introduction of significant amounts of fluid into an already distended and stressed segment of bowel. This can further compromise the integrity of the mucosa and can lead to bowel necrosis and perforation.[8]

Yet, despite these risks, gastrografin enemas remain the treatment of choice in patients with an uncomplicated meconium ileus. The survival rate of these infants is significantly higher than of those treated surgically (whose mortality can be as high as 50%),[2,6] since the latter are subjected to the risks of surgery, general anesthesia, and the postoperative pulmonary complications, which are exacerbated further by their underlying abnormality, cystic fibrosis.[6]

In our situation, where decompression done intraoperatively with direct visualization and manipulation of the obstructed meconium filled segment, the radiopacity of the solution is no longer a significant factor. Perhaps Tween-80 may be the most useful component for the actual loosening and softening of the meconium and so may be used either in combination with an isotonic gastrografin solution or even alone, with relief of the obstruction and no untoward side effects.[3,10]

This case stresses the importance of $P_{tc}O_2$ monitoring in neonates for surgical procedures. Not only is it useful to prevent oxygen toxicity or arterial hypoxemia, but it is also a good indicator of decreased cardiac output causing inadequate perfusion and tissue hypoxia. In adults, when cardiac output decreases, the $P_{tc}O_2$ will decrease in relation to the cardiac output, despite Pa_{O_2} being maintained at normal levels.[11] In this instance, a similar mechanism has been demonstrated in the neonate. It is a warning that the $P_{tc}O_2$ will change in relation to

the Pa_{O_2} only if the cardiac output is maintained at normal levels. To differentiate between arterial hypoxemia and a decrease in cardiac output, analysis of arterial blood gases should be performed. In this infant it was not done because an arterial sample could not be obtained. The lack of response to increasing the FI_{O_2} to 1.0 and the prompt improvement following iv fluid administration confirmed that the change in $P_{tc}O_2$ was secondary to a low cardiac output state.

Also demonstrated are the consequences of instilling a hyperosmolar solution into an extravascular body space. An immediate flux of water from the intravascular and extracellular fluid spaces into the newly formed "third space" occurs. It results in hypovolemia and hyperosmolarity of the body fluids, which can be corrected by administration of hypotonic intravenous fluids. To confirm the presence of a hyperosmolar state, changes in serum osmolality, serum electrolytes, and hematocrit can be measured.[6] We were unable to obtain a blood sample intraoperatively. Postoperatively, the hematocrit obtained was of no significant value because of the major blood loss during the case. The serum sodium and chloride concentrations were elevated postoperatively, showing some degree of hyperosmolarity. Serum osmolality unfortunately was not measured.

In this instance, lactated Ringer's solution and 5% serum albumin were used to correct the intravascular volume deficit, leaving the hyperosmolar state untreated. This could have resulted in intracranial bleeding and renal failure. Fortunately, these consequences were not encountered in this infant.

References

1. Noblett HR: Treatment of uncomplicated meconium ileus by Gastrografin enema: A preliminary report. J Pediatr Surg 4:190–197, 1969
2. Wagget J, Bishop MC, Koop CE: Experience with Gastrografin enema in the treatment of meconium ileus. J Pediatr Surg 5:649–654, 1970
3. Wood BP, Katzberg RW, Ryan DM, Karch FE: Diatrizoate enemas: Facts and fallacies of colonic toxicity. Radiology 126:441–444, 1978
4. Rowe MI, Furst AJ, Altman DM: The neonatal response to Gastrografin enema. Pediatrics 48:29–35, 1971
5. Wood BP, Katzberg RW: Tween 80/Diatrizoate enemas in bowel obstruction. AJR 132:747–750, 1978
6. Rowe MI, Seagram G, Weinberger M: Gastrografin-induced hypertoxicity—the pathogenesis of a neonatal hazard. Am J Surg 125:185–188, 1973
7. Poole CA, Rowe MI: Clinical evidence of intestinal absorption of Gastrografin. Radiology 118:151–153, 1976
8. Leonidas JC, Buny VF, Fellows RA, Beatty EC: Possible adverse effect of Methylglucamine Diatrizoate compounds on the bowel of newborn infants with meconium ileus. Radiology 121:693–696, 1976
9. Kuhns LR, Kanellitsas C: Use of isotonic water-soluble contrast agents for gastrointestinal examinations in infants. Radiology 144:411, 1982
10. Wood BP, Katzberg RW: Tween 80/Diatrizoate enemas in bowel obstruction: AJR 132:747–750, 1978
11. Tremper KK, Shoemaker WC: Transcutaneous oxygen monitoring of critically ill adults, with and without low flow shock. Crit Care Med 9:706–709, 1981

Anesthesiology
61:456–459, 1984

Internal Jugular Vein Function after Swan–Ganz® Catheterization

NANCY A. K. PERKINS, M.D.,* WAYNE S. CAIL, M.D.,† ROBERT F. BEDFORD, M.D.,‡
JOHN H. ELINGER, M.D.,* ANTHONY J. BUSCHI, M.D.†

Thromboembolic phenomena are recognized complications resulting from use of flow-directed (Swan–Ganz®) pulmonary artery catheters,[1–5] and a high incidence (66%) of internal jugular vein thrombosis has been reported in medical patients.[6] Internal jugular vein thrombosis is a potentially devastating complication in patients with neurologic disorders.[7–9] Since we routinely use pulmonary artery pressure monitoring during neurosurgery,[10] we sought to evaluate the incidence and

* Research Fellow, Department of Anesthesiology.
† Clinical Assistant Professor, Department of Radiology.
‡ Associate Professor of Anesthesiology and Neurological Surgery.

Received from the Departments of Anesthesiology and Radiology, University of Virginia School of Medicine, Charlottesville, Virginia 22908. Accepted for publication February 24, 1984. Supported (in part) by research training grant #T32-GM-07590-04 from the National Institutes of Health (Dr. Perkins). Presented in part at the 1981 and 1982 annual meetings of the American Society of Anesthesiologists.

Address reprint requests to Dr. Bedford, Box 238, Department of Anesthesiology, University of Virginia School of Medicine, Charlottesville, Virginia 22908.

Key words: Anesthesia: neurosurgical. Complications: internal jugular vein thrombosis. Equipment: catheters, flow-directed.