

References

1. Sullivan MT, Neff WB: A modified Saunders ventilating system for rigid wall bronchoscopy. *ANESTHESIOLOGY* 1979; 50:473-5
2. Dumon F, Shapshay S, Bourcureau J, Cavaliere S, Meric B, Garbi N, Beamis J: Principles for safety in application of Neodymium-YAG laser in bronchology. *Chest* 1984; 86:163-8
3. Vorc'h G, Fischler M, Personne C, Colchen A: Anesthetic management during Nd:YAG laser resection for major tracheobronchial obstructing tumors. *ANESTHESIOLOGY* 1984; 61:636-7
4. Peachey T, Eason J, Moxhan J, Jarvis D, Driver M: Systemic air embolism during laser bronchoscopy. *Anaesthesia* 1988; 43:872-5
5. Ross D, Mohsenifar Z, Potkin R, Roston W, Shapiro S, Alexander J: Pathogenesis of cerebral air embolism during Neodymium-YAG laser photoresection. *Chest* 1988; 94:660-1
6. Lang N, Wait G, Read R: Cardio-cerebrovascular complications from Nd:YAG laser treatment of lung cancer. *Am J Surg* 1991; 162:629-32

7. Van Allen CM, Hrdina LS, Clark J: Air embolism from the pulmonary vein. *Arch Surg* 1929; 19:567-99
8. Evans D, Hardenbergh E, Hallenbeck JM: Cardiovascular effects of arterial air embolism, Arterial Air Embolism and Acute Stroke. Edited by Hallenbeck JM, Greenbaum LJ. Rockville, MD, Undersea Med Soc, 1978, pp 20-33
9. Evans D, Kobrine A, Weathersby P, Bradley M: Cardiovascular effects of cerebral air embolism. *Stroke* 1981; 12:338-441
10. Giesecke AH, Gerbershagen HU, Dortman C, Lee D: Comparison of the ventilating and injection bronchoscopes. *ANESTHESIOLOGY* 1973; 38:298-303
11. Benumoff JL: Anesthesia for special elective procedures, Anesthesia for Thoracic Surgery, 2nd Edition. Edited by Benumoff JL. Philadelphia, WB Saunders, 1995, pp 500-1
12. Warner ME, Warner MA, Leonard P: Anesthesia for Neodymium: YAG (nd:YAG) laser resection of major airway obstructing tumors. *ANESTHESIOLOGY* 1984; 60:230-2

Anesthesiology
1997; 86:1390-2

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Perioperative Acute Renal Failure Associated with Preoperative Intake of Ibuprofen

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NONSTEROIDAL antiinflammatory drugs (NSAIDs) inhibit prostaglandin synthesis. Because renal blood flow depends on prostaglandin, particularly when circulating blood volume is decreased, a recommendation has been made that NSAIDs be withheld before surgery because of the risk of renal dysfunction.¹ However, there has been no report of perioperative renal dysfunction attributable to preoperative administration of NSAIDs, and a

recent review has endorsed preoperative administration of NSAIDs for minor outpatient procedures and a combined multimodality drug therapy including NSAIDs after surgery.² The authors report a case of perioperative acute renal failure after lumbar discectomy in a young patient who was taking ibuprofen preoperatively.

Case Report

A 94-kg man, aged 38 yr, with a 10-yr history of back pain and a herniated nucleus pulposus compressing the L5 nerve root was admitted for lumbar discectomy. Past medical history revealed alcohol abuse and drug-seeking behavior with claims of recovery and abstinence in the past year. The patient admitted to taking acetaminophen-codeine (300 mg - 30 mg) tablets and meperidine (50 mg) tablets that were prescribed to him at 6-h intervals until the day before surgery. Physical examination revealed no systemic abnormalities. Complete hemogram and urinalysis were within normal limits, and on the eve of his operation, concentrations of blood urea nitrogen (BUN) and serum creatinine were 17 mg/dl and 0.7 mg/dl, respectively. A lumbar laminectomy using general anesthesia was planned.

After uneventful induction of anesthesia and orotracheal intubation

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Received from the Anesthesiology Service, Veterans Affairs Puget Sound Health Care System—Seattle Division, and the Department of Anesthesiology, University of Washington School of Medicine, Seattle, Washington. Submitted for publication September 19, 1996. Accepted for publication February 19, 1997.

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Key words: Nonsteroidal antiinflammatory drugs. Renal dysfunction, perioperative.

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using metocurine, 2 mg, sodium thiopental, 500 mg, and succinylcholine, 120 mg, an indwelling urinary catheter was inserted into the bladder, and 200 ml of residual urine was drained. The patient was then placed in the prone position on a Wilson frame with the hips and knees in approximately 60° flexion. Anesthesia was maintained using nitrous oxide and isoflurane and intermittent doses of intravenous morphine. The patient was hemodynamically stable throughout the operation, which lasted approximately 5 h. During the first h, the patient received intravenous lactated Ringers solution, 1200 ml, and the urine output was 200 ml. Thereafter, the urine output decreased markedly with 0 ml, 10 ml, 5 ml, and 5 ml recorded at hourly intervals, whereas the cumulative volumes of lactated Ringers solution administered intravenously were 1700 ml, 3200 ml, 4000 ml, and 4700 ml, respectively. Total blood loss was estimated to be approximately 250 ml.

Emergence from anesthesia was uneventful, but oliguria persisted in the postanesthesia care unit and in the ward overnight. Results of blood tests and urinalysis on the first postoperative day were the following: BUN, 38 mg/dl; serum creatinine, 3.7 mg/dl; urine sodium, 41 mEq/l; urine osmolality, 347 mOsm/kg; and granular casts in urine. Renal ultrasonogram showed no evidence of urinary tract obstruction. On further questioning, the patient admitted to taking ibuprofen 600 mg, four times a day until the evening before surgery, despite having been asked to discontinue it 1 week before surgery. Acute tubular necrosis was diagnosed, and he was thereafter administered only intravenous potassium-free solutions (normal saline and dextrose 5% solutions). Peak concentrations of BUN and serum creatinine were 44 mg/dl and 4.8 mg/dl, respectively, on the third postoperative day, declining to 31 mg/dl and 2.9 mg/dl, respectively, on the ninth postoperative day when the patient was discharged from the hospital. At the follow-up clinic visit 6 weeks later, there was full recovery, with concentrations of BUN and serum creatinine returning to normal levels.

Discussion

Nonsteroidal antiinflammatory drugs have no effect on renal function in healthy human beings.¹ In patients with a decreased circulating blood volume or with congestive heart failure, NSAIDs produce a decrease in glomerular filtration rate and creatinine clearance.¹ In these patients, adrenergic and renin-angiotensin activation produces renal vasoconstriction. Local release of vasodilatory prostaglandins, also brought about by angiotension II and catecholamines, is necessary for maintaining renal blood flow. Therefore, inhibition of prostaglandin synthesis by NSAIDs can result in decreases in renal blood flow and function in these patients.¹ Such effects have been well documented, and there are many reports of renal dysfunction in healthy patients associated with the administration of ketorolac tromethamine.³⁻⁶ In most of these cases, the patients received repeated doses of ketorolac after surgery.³⁻⁶ In one patient, acute renal failure was associated with a single dose of ketorolac administered intraoperatively.⁴ In con-

trast, our patient was taking ibuprofen only preoperatively in doses that were large but still within recommended limits.⁷ Blood pressure and heart rate were stable during the operation, fluid administration was adequate — approximately 10 ml/kg/h — and blood loss was only 250 ml, suggesting that a decrease in circulating blood volume was not a contributing factor in renal dysfunction. It is well established that surgical stress results in adrenergic and renin-angiotensin activation.⁸ The plasma half-life of ibuprofen is 2–4 h,⁷ but the authors were unable to determine the duration of its effect on prostaglandin synthesis from any published source. It is possible that ibuprofen produced persistent inhibition of prostaglandin synthesis that resulted in decrease in renal blood flow because of adrenergic and renin-angiotensin activation during the operation, leading to an acute tubular necrosis.

Other factors also may have contributed to acute tubular necrosis in this patient. It is possible that the patient, who gave unreliable history, was taking other medications or substances that may have contributed to acute tubular necrosis. Although perioperative acute renal failure in patients free of systemic diseases who undergo uncomplicated operations is rare, isoflurane produces moderate but reversible decreases in renal blood flow and glomerular filtration rate.⁸ Although the effect of prone position on renal function is not well documented, improper prone position with pressure on the abdomen may cause compression of abdominal vessels⁹ that might have decreased the blood supply to the kidneys. It is possible that the combination of preoperative intake of ibuprofen, general anesthesia using isoflurane, and prone position during the operation might have made the patient susceptible to acute tubular necrosis.

Acute tubular necrosis caused by NSAIDs has a good prognosis if recognized early. Discontinuation of the drug and conservative management using parenteral solutions free of potassium salts usually results in prompt recovery of renal function^{1,3-5} as was observed in the present case. The authors were unable to determine from a review of the literature whether management using diuretics or dopamine is of benefit in reversing acute tubular necrosis caused by NSAIDs. In general, management of acute tubular necrosis using diuretics or dopamine does not influence recovery of renal function.¹⁰

References

1. Clive DM, Stoff JS: Renal syndromes associated with nonsteroidal antiinflammatory drugs. *N Engl J Med* 1984; 310:563–72

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2. Souter AJ, Fredman B, White PF: Controversies in the perioperative use of nonsteroidal antiinflammatory drugs. *Anesth Analg* 1994; 79:1178-90
3. Pearce CJ, Gonzalez FM, Wallin JD: Renal failure and hyperkalemia associated with ketorolac tromethamine. *Arch Intern Med* 1993; 153:1000-2
4. Smith K, Halliwell RMT, Lawrence S, Klineberg PL, O'Connell PO: Acute renal failure associated with intramuscular ketorolac. *Anaesth Intensive Care* 1993; 21:700-3
5. Patel NY, Landercasper J: Ketorolac induced acute renal failure: A case report. *Wis Med J* 1995; 94:445-7
6. Buck ML, Norwood VF: Ketorolac-induced acute renal failure in a previously healthy adolescent. *Pediatrics* 1996; 98:294-6

7. Insel PA: Analgesic-antipyretic and antiinflammatory agents and drugs employed in the treatment of gout, Goodman and Gilman's Pharmacological basis of therapeutics, Ninth Edition. Edited by Hardman JG, Limbird LE. New York, McGraw Hill, 1996, pp 617-57
8. Sladen RN: Renal physiology, Anesthesia, Fourth Edition. Edited by Miller RD. New York, Churchill Livingstone, 1994, pp 663-88
9. Martin JT: Patient positioning, Clinical Anesthesia, Second Edition. Edited by Barash PG, Cullen BF, Stoelting RK. Philadelphia, JB Lippincott, 1992, pp 709-36
10. Brady HR, Brenner BM: Acute renal failure, Harrison's Principles of Internal Medicine, 13th Edition. Edited by Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL. New York, McGraw-Hill Inc., 1994, pp 1265-74

Anesthesiology
1997; 86:1392-4
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Sevoflurane Inhalation Induction for Emergency Cesarean Section in a Parturient with no Intravenous Access

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RAPID sequence induction of general anesthesia and paralysis, with intubation of the trachea, is the standard of care for patients who require emergency cesarean section because of severe fetal distress in the absence of preexisting regional anesthesia. As a result of the impaired gastric emptying associated with pregnancy and labor, these patients are treated as if their stomachs are full; there is a serious risk of maternal morbidity or mortality if aspiration occurs.¹ Rapid sequence induction requires an intravenous route for drug administration. In rare instances, intravenous access may not be available, and the severity of the fetal distress may man-

date delivery before access can be obtained. The authors describe a case wherein inhalation induction of anesthesia with a volatile anesthetic that does not irritate the airways proved to be a suitable alternative.

Case Report

Our anesthesia call team was emergently summoned to the labor and delivery floor to care for a uniparous, 29-yr-old woman, gravida 2, at 38 weeks gestation, who presented a few minutes earlier complaining of labor pain. During the initial vaginal examination, her membranes ruptured to reveal a double footling breech fetus with 15-20 cm of prolapsed umbilical cord. The obstetrician had applied a heel electrode and was manually attempting to keep the fetus in the uterus as the patient was wheeled into the delivery room. When we arrived, the patient was on her hands and knees on the operating table. The fetal heart rate was 50 beats/min, and an obstetrician was performing a bimanual manipulation of the fetus. Nurses and technicians were preparing for emergency cesarean section, and the obstetrician stated, "The baby has to come out now." There was no intravenous access, and no monitors had been applied to the mother.

Within 1 min of our arrival, the patient was rolled onto her back with left uterine displacement, and the abdomen was hastily painted with Betadine®. During this time, both arms and the neck were inspected for a vein that could be used for intravenous catheter insertion or direct injection of induction drugs; unfortunately, no veins were apparent. The necessity for *immediate* delivery because

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Received from the Department of Anesthesiology, University of Connecticut School of Medicine, Farmington, Connecticut. Submitted for publication December 17, 1996. Accepted for publication February 28, 1997.

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Key words: Anesthetics, inhaled: sevoflurane. Obstetric anesthesia: emergency cesarean section.