PAPER

Ogilvie Syndrome as a Postoperative Complication

Patty L. Tenofsky, MD; R. Larry Beamer, MD; R. Stephen Smith, MD

Hypothesis: Ogilvie syndrome is a postoperative complication.

Design: Case series.

Setting: University-affiliated tertiary-care hospital.

Patients and Methods: The medical records of patients diagnosed as having Ogilvie syndrome after trauma or operation between 1989 and 1998 were reviewed. Medical charts were examined for history, treatment, cecal diameter, and outcome.

Main Outcome Measures: Data were summarized in an attempt to identify patient populations at risk for Ogilvie syndrome.

Results: Ogilvie syndrome was diagnosed in 36 patients, 24 of whom were men. Average age at diagnosis was 68.9 years. Abdominal radiographs were obtained at time of diagnosis (mean cecal diameter, 13.4 cm; range, 8-20 cm). Operations preceding Ogilvie syndrome were orthopedic or spinal (n=14), cardiothoracic (n=12), abdominal (n=5), and vascular (n=2). Nonoperative trauma accounted for 3 cases. Coronary artery bypass grafting was the single most frequent procedure leading to Ogilvie syndrome (n=9 [25%]). Conservative treatment was successful in 52.8% of cases (n=19). Twenty colonoscopic decompressions were performed on 13 patients, with an overall success rate of 77% (n=10). Of the 3 patients in whom colonoscopic decompression failed, 2 died and 1 required operation. Five of the 36 patients required surgical intervention, with a mortality rate of 60% (n=3).

Conclusions: Previous studies have shown Ogilvie syndrome to occur most commonly after obstetrical/gynecologic, abdominal/pelvic, and orthopedic procedures. Our data confirm that patients undergoing orthopedic and spinal procedures are at higher risk, but that the surgical procedure most commonly leading to Ogilvie syndrome was coronary artery bypass grafting. Cardiothoracic surgeons, orthopedic surgeons, and neurosurgeons should be cognizant of this complication in the patient whose abdomen becomes distended postoperatively. If recognized early and treated appropriately, pseudo-obstruction will resolve in most patients. If surgical intervention is required, the subsequent mortality rate is high.

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From the Department of Surgery, The University of Kansas School of Medicine–Wichita.

In 1948, OGILVIE[1] described 2 cases of massive colonic dilation in the absence of mechanical obstruction. Both of his patients suffered from unsuspected malignant disease in the region of the celiac axis and semilunar ganglia. Ogilvie concluded that the nervous supply to the colon was affected by these tumors and that the neurological derangement led to a colonic "pseudo-obstruction." Multiple case reports have been published since that time; however, the actual mechanism that causes the colon to dilate in the absence of obstruction remains obscure. Most current research supports the theory that Ogilvie syndrome is secondary to large-bowel parasympathetic dysfunction. This theory has been supported by the successful use of parasympathomimetic agents in the treatment of Ogilvie syndrome. Agents that increase parasympathetic tone have been shown to resolve pseudo-obstruction without colonic decompression or surgical manipulation.[2] Turegano-Fuentes et al[3] demonstrated complete resolution of Ogilvie syndrome with the use of neostigmine methylsulfate in 12 of 18 patients.

Pseudo-obstruction of the colon can occur acutely or as a chronic condition. Chronic colonic pseudo-obstruction usually recurs or persists, whereas acute Ogilvie syndrome is a transient problem associated with systemic illness or the postoperative state. The acute syndrome must be recognized early and treated appropriately to avoid cecal perforation. Nonoperative management is usually attempted initially, with nasogastric suction, fluid resuscitation, enemas, and possibly neostigmine. If the patient's cecum is dangerously enlarged (>13 cm in diameter), or if conservative treatment fails, colonoscopic decompression is recom-

PATIENTS AND METHODS

Cases of Ogilvie syndrome occurring after trauma or operation between January 1, 1989, and December 31, 1998, were identified and the patients' medical records were retrospectively reviewed. Review of records took place at 2 university-affiliated tertiary care hospitals. Cases of Ogilvie syndrome were identified in the following manner. A listing of all charts from 1989 through 1998 that used the code of bowel obstruction was requested. This list was examined and all charts were eliminated in which (1) there was no operative procedure or traumatic event, or (2) there was evidence of a mechanical bowel obstruction (ie, cancer, adhesions, volvulus, or inflammatory bowel disease). The remaining charts were examined and any patient who met the criteria for Ogilvie syndrome was included. Patients were considered to have Ogilvie syndrome if radiographic findings demonstrated colonic distention greater than 8 cm without evidence of mechanical obstruction. It was assumed that all postoperative patients had no mechanical cause of Ogilvie syndrome, since they had no evidence of preexisting obstruction. If, however, the patient manifested Ogilvie syndrome without a precipitating cause, a mechanical obstruction was ruled out with a diatrizoate meglumine (Cystografin) enema. Diatrizoate not only diagnoses mechanical obstruction but has been shown to be therapeutic in some cases of Ogilvie syndrome.[8] Patients who had small-bowel dilation in addition to colonic dilation were considered to have a postoperative ileus and were excluded from the study. Charts were examined for patient demographics (age, sex, race), type of operation (general, cardiac, thoracic, orthopedic, gynecologic, or other), injury, anesthetic used, number of days from admission to operation, postoperative day of diagnosis of Ogilvie syndrome, interval from diagnosis to resolution or death, cecal diameter, preoperative and postoperative medications, preoperative medical history, treatment (conservative therapy, colonoscopic decompression, and/or operation), length of hospitalization, and outcome. Data were summarized and frequencies and means reported.

mended.[4] Failure of colonoscopic decompression usually requires operative treatment with cecostomy or a right hemicolectomy.

The postoperative state represents a significant risk factor for the development of Ogilvie syndrome. A review of the literature shows that 50% to 60% of acute colonic pseudo-obstructions occur after a surgical procedure or trauma.[4-7] Ogilvie syndrome in the postoperative patient is particularly worrisome, because the patient's abdominal distention may be confused with a simple postoperative ileus. It is important for the surgeon to be aware of the disorder, its risk factors, and its potentially lethal complications, since it may be successfully treated if recognized early. Therefore, the purpose of this study was to identify risk factors in patients who acquired Ogilvie syndrome after operation or injury.

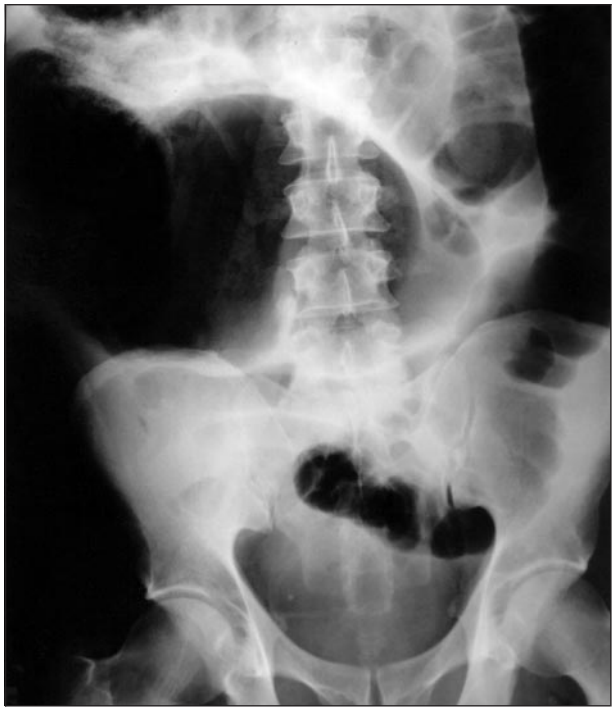

Figure 1. *Radiograph demonstrating massive dilation of the colon in a patient who developed Ogilvie syndrome after scapular fracture and repair.*

RESULTS

Thirty-six patients were diagnosed as having Ogilvie syndrome acutely after trauma or a surgical procedure between 1989 and 1998. Sixty-seven percent of patients were male (n=24), and mean±SD age at diagnosis was 68.9±12.5 years (range, 33-89 years). Most patients in this series had an extensive medical history. Hypertension was prevalent in 16 (44%), and 24 patients (67%) had coronary artery disease. Type 1 diabetes mellitus was present in 4 patients (11%). All patients underwent roentgenography of the abdomen in the supine and/or upright position at the time of diagnosis. The size of the cecum was noted on radiographic reports of 27 patients. The average size of the cecum at diagnosis was 13.4±3.0 cm (range, 8-20 cm). The abdominal roentgenogram shown in **Figure 1** is of a 51-year-old previously healthy man who underwent an open reduction and internal fixation of his left scapula. This case illustrates the magnitude to which the colon can dilate as a result of this syndrome.

The most common operation precipitating pseudo-obstruction was coronary artery bypass grafting, accounting for one fourth (n=9) of the procedures leading to Ogilvie syndrome (**Table 1**). Two other cardiac procedures (cardiac transplantation and coronary angioplasty) also led to pseudo-obstruction, as well as 1 thoracoscopic and 2 peripheral vascular procedures. If spinal operations or fractures were included as orthopedic procedures, this combination accounted for nearly half of the total (n=16 [44%]). There were 19 592 cardiopulmonary bypass procedures performed during the 10-year study period at the 2 institutions reviewed. The incidence after coronary bypass grafting is therefore very low (0.046%). Spinal procedures consisted of 4 fusions, 2 laminectomies, and 2

Table 1. Characterization of 36 Patients With Ogilvie Syndrome by Procedure or Trauma

Category	No. (%)
Coronary artery bypass grafting	9 (25)
Other cardiothoracic procedures	3 (8)
Orthopedic procedures	8 (22)
Spinal procedures or fractures	8 (22)
General surgical procedures	5 (14)
Nonoperative trauma	1 (3)
Peripheral vascular procedures	2 (6)

nonoperative traumatic fractures (1 cervical and 1 thoracic). Other orthopedic procedures included 3 total knee replacements, 1 total hip replacement, and 4 open reductions with internal fixation for trauma (fractures: scapula, 1; shoulder, 1; acetabulum, 1; and hip, 1). There were approximately 10 000 total knee and hip replacement procedures performed during the 10 years in the 2 institutions reviewed. Miscellaneous general surgical procedures and simple nonoperative trauma comprised only 6 cases (16.7%). These procedures included 2 laparoscopic cholecystectomies, 1 inguinal hernia repair, 1 ventral hernia repair, 1 resection of a gastric tumor, and a case of blunt trauma with rib fractures.

Of the patients acquiring Ogilvie syndrome, 26 (72%) had undergone general endotracheal intubation. Five patients (14%) received spinal catheters for anesthesia: 4 for orthopedic procedures and 1 for a vascular procedure. Only 1 epidural catheter was placed for a vascular procedure. One patient (3%) was given a local anesthetic, and 3 patients (8%) did not receive any anesthetic agents.

Data regarding intervals are shown in **Table 2**. The mean interval from operation to diagnosis of Ogilvie syndrome was 5.1 days. The time from diagnosis of the syndrome to resolution or death was 6.6 days. Patients who were diagnosed as having this syndrome generally had a lengthy hospitalization (18.5 days; range, 4-58 days).

The mortality rate for patients diagnosed as having Ogilvie syndrome was 14% (n=5). Conservative treatment consisting of nasogastric tube placement, fluid resuscitation, and enemas was successful in 19 patients (53%). No patients were given neostigmine. Thirteen patients underwent colonoscopy, with eventual resolution in all but 3 (77%). Twelve (92%) of the 13 patients had successful decompression of the colon after the initial colonoscopy; however, 6 patients (46%) had recurrence of symptoms, necessitating a second decompression. A third colonoscopy was required in 1 patient. One patient who underwent unsuccessful colonoscopic decompression required a right hemicolectomy and recovered. The remaining 2 patients who underwent unsuccessful colonoscopic decompression eventually died without surgical intervention. The first of these patients had ischemic bowel, and the family chose not to proceed with operative treatment. The second patient died of unknown causes; however, the patient's bowel function had returned to normal with initiation of tube feedings. Two patients underwent operation after conservative therapy and 2 underwent surgical intervention as their initial therapy. In 1 patient who underwent operation as the initial treatment, free air was suspected, prompting operative exploration with cecostomy. The second patient who had operative intervention as initial therapy underwent exploration for a massively dilated colon suspected to be complicated by ischemic injury. A total of 5 (14%) of the 36 patients required surgical intervention, which included 1 cecostomy, 3 right hemicolectomies, and a transverse colostomy.

Table 2. Interval Data for Patients With Ogilvie Syndrome

Variable	Mean ± SD	Range
Postoperative day of diagnosis (n = 36)	5.1 ± 3.6	1-17
Interval from diagnosis to resolution or death (n = 34)	6.6 ± 5.1	1.5-20
Length of hospital stay, d (n = 36)	18.5 ± 12.6	4-58

The mortality rate for patients who required operative intervention was high (60%), with 3 of the 5 surgical patients dying. When the records of the 5 patients who died were evaluated, it was evident that they all had considerable medical histories. One had suffered a myocardial infarction before operation, precipitating the patient's coronary artery bypass grafting procedure. All patients who died had coronary artery disease and hypertension; 2 had diabetes mellitus. Ogilvie syndrome was diagnosed between postoperative days 3 and 7 in patients who died. Three patients had evidence of colonic ischemia and underwent an operation as their initial treatment. Two patients underwent multiple, unsuccessful colonoscopies for their treatment. Although difficult to ascertain from retrospective analysis, it appears that a delay in diagnosis and inappropriate treatment affected outcome in these patients.

COMMENT

Colonic dilation in the absence of mechanical obstruction after operation is indicative of Ogilvie syndrome. The hallmark of the syndrome is abdominal distention with or without pain. Plain abdominal radiographs demonstrate massive colonic dilation, especially of the cecum and right colon.

Our data indicate that Ogilvie syndrome is most common in the seventh decade, with an average age of 68.9 years (median, 71 years; range, 33-89 years). The data also showed a 2:1 male-to-female ratio. The higher incidence in men was also documented by previous studies.[5,9,10] Clarke et al[10] studied 30 patients who developed Ogilvie syndrome after hip arthroplasty. They found a 3:1 male-to-female ratio and an average patient age of 74.3 years.

Few studies of Ogilvie syndrome have limited the scope to postoperative or injured patients. These types of cases account for approximately 50% to 60% of all patients with Ogilvie syndrome.[4-7] Previous retrospective studies have investigated which types of procedures most commonly lead to Ogilvie syndrome. In a large meta-analysis performed in 1986 by Vanek and Al-Salti,[7] the procedure most commonly preceding the syndrome was

cesarean section. In 1976, Spira et al[11] also found a 35% occurrence of Ogilvie syndrome after cesarean section or normal delivery. Interestingly, no patients in our retrospective review had undergone obstetric or gynecologic procedures. Our findings are similar to those of a retrospective study by Jetmore et al[5] in which only 1 of 56 patients with Ogilvie syndrome had an obstetric or gynecologic procedure. The association between Ogilvie syndrome and orthopedic procedures has been well established, with reported incidences between 5.4% and 22.2%.[10,12,13] Ogilvie syndrome has also been associated with lumbar spine surgery in previous reports.[14] Therefore, it is not surprising that 44% of the patients in our study group had either an orthopedic or a spinal procedure.

Jetmore et al[5] collected data between 1983 and 1989 and found that 27 (56%) of 56 patients with Ogilvie syndrome had undergone surgery. They found that the most frequent events leading to postoperative Ogilvie syndrome were cardiovascular or thoracic procedures.[5] This is in contrast to the findings of the 2 large meta-analyses, which reported the incidence of Ogilvie syndrome after cardiovascular or thoracic surgery to be between 2.9% and 3.5%.[6,7] The data collected in our study and the study by Jetmore et al are remarkably similar, which stresses that Ogilvie syndrome must be considered a potential complication after cardiothoracic operations.

The pathophysiological mechanism of Ogilvie syndrome after operation or trauma is unknown but most likely neurogenic. The prevailing theory to explain the pseudo-obstruction is deprivation of parasympathetic supply to the colon. The success of the parasympathomimetic drug neostigmine in the treatment of Ogilvie syndrome lends credence to this theory.[15,16] The cause of this parasympathetic neuropraxia is unknown. The pain and stress of operation or trauma may result in a sympathetic surge with relative loss of parasympathetics. In the case of coronary artery bypass grafting, the cardiopulmonary bypass may contribute to neurogenic derangement, but this is speculative.

Whatever the cause, Ogilvie syndrome should be recognized early and treated appropriately. Colonic distention may be rapidly progressive and lead to cecal necrosis and perforation. This significantly worsens the prognosis, as has been reported in previous studies that document mortality rates approaching 50%.[17] A high mortality rate was also demonstrated by our data, in which 3 (60%) of 5 patients who required operation for perforation, ischemia, or an acute abdomen died. Conservative treatment with nasogastric tube decompression, fluid resuscitation, and enemas was successful in 53% of our patients. We believe that, when patients are treated nonoperatively, abdominal radiographs should be repeated every 12 to 24 hours to monitor changes in cecal diameter. Colonoscopy may be used if conservative treatment fails, or if the cecum is on the verge of perforation. Many authors recommend colonoscopic or surgical decompression of the colon if the cecal dilation is greater than 12 cm.[7,9,13,18] Our overall success rate with colonoscopic decompression of 77% is similar to that in previous reports.[19] However, there were 2 deaths in the colonoscopy group. Although reports in the literature have documented favorable results with agents such as neostigmine,[2] no patients in our study received pharmacological treatment for Ogilvie syndrome.

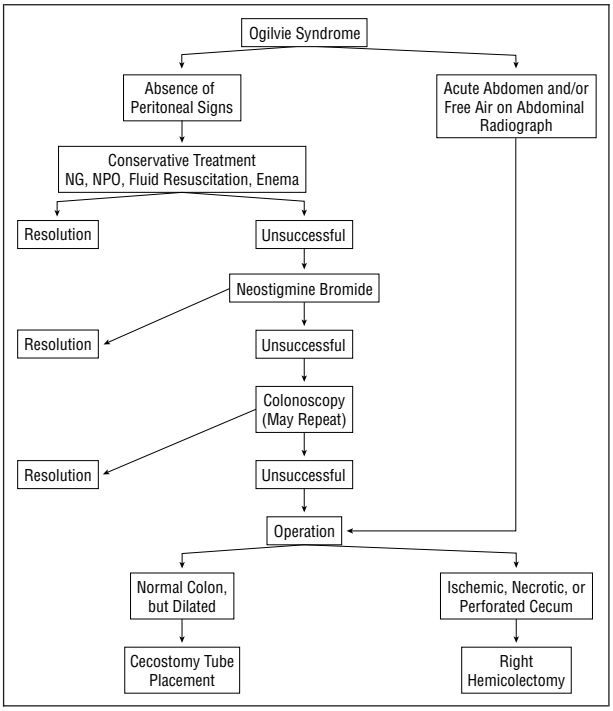

Figure 2. *Treatment algorithm for Ogilvie syndrome. NG indicates nasogastric tube; NPO, nothing by mouth.*

CONCLUSIONS

Ogilvie syndrome is an established postoperative complication. However, because it is rare, it may be overlooked or treated as an adynamic ileus. Unfortunately, no single cause of Ogilvie syndrome can be identified; however, it is apparent from the data that certain groups are at risk. Surgeons who perform cardiovascular and thoracic, orthopedic, or spinal procedures should include Ogilvie syndrome in the differential diagnosis when a postoperative patient's abdomen becomes markedly distended. If appropriately diagnosed, the syndrome can be treated conservatively with careful surveillance of the cecal diameter. Neostigmine has been shown to be an effective medical treatment and is a good addition to conservative management. Colonoscopy is a safe and effective method for treatment of this syndrome when conservative treatment has failed. Operative treatment is reserved for patients in whom colonoscopic decompression has failed or who develop peritoneal signs. On the basis of information gleaned from our study, as well as the current literature, we have devised a simple algorithm for treatment of patients diagnosed as having Ogilvie syndrome (**Figure 2**). Use of this algorithm should enable physicians to optimize their treatment of patients diagnosed as having Ogilvie syndrome.

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Corresponding author: R. Stephen Smith, MD, Department of Surgery, The University of Kansas School of Medicine–Wichita, 929 N St Francis, Wichita, KS 67214 (e-mail: Rsmith3@kumc.edu).

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DISCUSSION

Richard C. Thirlby, MD, Seattle, Wash: It is appropriate that this fine review of the presentation and treatment of patients with colonic pseudo-obstruction or Ogilvie syndrome be presented to the Western Surgical Association. The recognition of this syndrome and refinements in treatment have improved over the years in large part due to the contributions of our membership. In 1976, Kurkora and Tom Dent presented their classic paper entitled "Colonoscopic Decompression of Massive Nonobstructive Cecal Dilation" to the 84th annual meeting of the Western Surgical Association. This paper was instrumental in improving the recognition and treatment of these challenging patients (*Arch Surg*. 1977;112:512-517). Since their work was published in the ARCHIVES in 1977, colonoscopic decompression has become the standard intervention in patients refractory to conservative measures. However, since 1977 things have advanced. For example, at last year's SSAT (Society for Surgery of the Alimentary Tract) meeting, Dr Pitcher and his coworkers from nearby University of New Mexico reported that Cystografin enemas are effective treatment of these patients. In their series of 18 patients who developed Ogilvie syndrome while hospitalized for trauma or surgery, 15 of 18 were successfully treated with Cystografin enemas. They concluded that "the safety, efficacy, and ease of this procedure make Cystografin enema optimal first-line treatment for acute colonic pseudo-obstruction"(*J Gastrointest Surg*. 1999;3:173-177).

For years, Ogilvie syndrome was a surgical disease. Operation was frequently necessary. When Tom Dent introduced colonoscopy into the treatment algorithm, Ogilvie syndrome became, in many institutions, a gastroenterologist's disease. Not surprisingly, the search for a pharmacological treatment for this condition intensified when it became apparent that it was also a nighttime and/or weekend disease. A gastroenterologist, Michael Kimmey, recently published a study that I believe establishes intravenous neostigmine as the first-line treatment of patients with acute colonic pseudo-obstruction (*N Engl J Med*. 1999;341:137-141). In this blinded-randomized trial, 10 of 11 patients receiving neostigmine had prompt colonic decompression as compared to none of the 10 patients who received placebo. The median time to response was 4 minutes. Two patients had recurrences and required colonoscopy; one patient eventually required colectomy. Side effects, such as excess salivation and bradycardia, were not severe, although all patients require EKG monitoring, and atropine must be available at the bedside. This treatment is so quick and so effective that patients must be lying on a bedpan when it is administered.

In my opinion, the current state of the art in the treatment of these patients is as follows. The diagnosis is usually clear. These usually elderly patients have marked abdominal distention and radiographs that demonstrate colonic distention without evidence of obstruction. Cecal diameter is at least 10 cm, usually greater than 12 cm. If there is any question about the diagnosis, a diagnostic and potentially therapeutic Cystografin enema should be performed. This procedure requires 2 to 3 L of solution left in place for 15 to 20 minutes. The next intervention in most patients is to reverse any correctable causes of poor colonic motility. There are frequently 2. The most common is narcotic use. Narcotics must be eliminated: not reduced, but eliminated. This may require the help of your anesthesia colleagues with placement of epidural catheters or other regional techniques, etc. When possible, NSAIDs should be used for pain. Other drugs that impair colonic motility must be stopped; otherwise, any intervention short of colectomy—whether it be neostigmine, Gastrografin enemas, or colonoscopy—will fail since the pseudo-obstruction will rapidly recur. Other harmful drugs include the calcium-channel blockers and anticholinergic psychiatric medications.

The second intervention gets to the original cause proposed by Ogilvie: that is, imbalance between sympathetic and parasympathetic innervation to the gut. Sympathetic tone impairs motility; parasympathetic tone enhances motility (hence the efficacy of neostigmine). Sympathetic blockade with regional analgesia techniques, such as epidural bupivacaine, will not only reduce narcotic use but also enhance motility independently. If conservative measures such as elimination of narcotics are not working quickly, the next step is the administration of 2 mg of neostigmine intravenously. It will work. It cannot be emphasized enough, however, that if precipitating factors for pseudo-obstruction are not corrected, recurrence is likely with any treatment. If neostigmine does not work, a Gastrografin enema is worth a try. If that does not work, then exploration should be performed urgently.

With these opinions in mind, I have the following questions and comments for Drs Tenofsky, Beamer, and Smith. First, I am a bit troubled by your entry criteria. Patients were iden-

tified for review when they were coded at discharge with a diagnosis of bowel obstruction. I suspect that some patients with Ogilvie syndrome resolve quickly and never get coded for bowel obstruction. These missed patients could affect your findings and conclusions. Second, the average size of the cecum at diagnosis was 13.4 cm, but the range was as low as 8 cm. Most authors require a cecal diameter of 10 or even 12 cm prior to making the diagnosis of colonic pseudo-obstruction. Could you comment on how you make the diagnosis? What do you think the role of contrast studies is in the workup of these patients?

Third, your series is unique in that a significant percentage of patients were recovering from cardiac operations. Do you have any explanation? A take-home message is that we should familiarize our cardiac surgery colleagues with the prevalence and lethality of this condition. Fourth, your manuscript does not list any concomitant medications. Do you know what percent of patients were on narcotics? Many patients were hypertensive. Do you know how many of these patients were on calcium-channel blockers, a class of drug known to impair colonic motility? Finally, since this review has been completed, have you any experience with either therapeutic Hypaque or Gastrografin enemas or neostigmine?

In summary, this is a timely, informative review of an important clinical syndrome. Ogilvie syndrome is a marker for very sick patients. Despite all efforts, at least 10% of these patients will never leave the hospital. There are many suggested treatments involving radiologists, gastroenterologists, and surgeons. Optimal treatment of these patients mandates that surgeons be the quarterbacks. Only then will a logical, safe, and effective multidisciplinary approach be consistently applied to the treatment of these patients.

Merril T. Dayton, MD, Salt Lake City, Utah: The use of neostigmine really has made a difference, but in a series like this, the cases that will teach us the most are those that were not operated on soon enough and ended up perforating or becoming ischemic. Are you able to identify in this series risk factors or a common predictor of those that perforated? Were you able to tell, for example, how long they had their pseudo-obstruction? We presume that is probably the major risk factor; if so, what is the time period that we should look at as being a critical time period?

James G. Tyburski, MD, Detroit, Mich: I have 2 questions. One is, were any of these patients on diet and this condition was unrecognized on the orthopedic or cardiothoracic services? Secondly, how sick were they? Were they in the ICU? Is there some kind of ischemia-reperfusion problem with the colon when people have cardiopulmonary bypass or a major orthopedic procedure?

James J. Peck, MD, Portland, Ore: I wondered if your protocol requires that the patient be monitored if you use neostigmine and if the physician needs to be present.

Frank A. Folk, MD, Naperville, Ill: I wonder about the high incidence following cardiac procedures. Since those patients are on large doses of cardiac medication as well as those on neurotropic medication, they may be predisposed to Ogilvie syndrome. Is there some way to quantify the role of such medications among patients who develop Ogilvie syndrome?

Dr Smith: How did we make the diagnosis? It was a combination of clinical examination with findings of abdominal distention as well as radiographic confirmation of dilatation of the cecum. Why did we choose 8 cm as our threshold? There are several reasons. First of all, the definition of Ogilvie syndrome is actually quite nebulous in the literature. Previous authors have used diameters from 9 to 12 cm. We chose to use 8 cm because we wanted to throw a broad net to include all potential patients with Ogilvie syndrome in our retrospective review. Additionally, we were interested in detecting patients who were early in the course of the process and we wanted to include these patients in our review as well. For these reasons we chose 8 cm as a cecal diameter consistent with Ogilvie syndrome.

As far as contrast studies are concerned, we have not used Gastrografin for either diagnosis or treatment. In fact, if we had a question regarding mechanical obstruction of the distal colon, we preferred to proceed directly to colonoscopy for both evaluation of the distal colon as well as decompression. It is very tempting, however, to relocate this potentially messy and unpleasant task to the radiology suite, with the radiologist attending, as opposed to the endoscopy suite, where the surgeon was in charge.

We agree that it is very important to inform our cardiac surgery colleagues of the incidence of Ogilvie syndrome in their patient population.

Why was there such a great number of patients from this group in our series? We believe it is strongly a matter of the denominator. During the study period at the 2 institutions involved, there were approximately 17 000 procedures utilizing cardiopulmonary bypass. The size of this group is responsible for the large number of cases of Ogilvie syndrome identified in this group.

Regarding operative medications, there are certainly a variety of substances that have been implicated as causative factors in the development of this syndrome. In fact, one of Ogilvie's first 2 patients, a lawyer, attributed the development of his abdominal discomfort and distention to "an injudicious feast of whiskey and gooseberries." To answer Dr Thirlby's question, all of the patients in our series, each and every one of them, had received significant analgesic doses of narcotics before colonic pseudo-obstruction developed. A majority of our patients were receiving antihypertensive medications as well as antiarrhythmics, including more than 50% of patients who were receiving calcium-channel blockers as well as digoxin.

Why did we not use neostigmine? To date there have been only 3 or 4 reports in the literature regarding the use of this drug before the very recent report by Pevec, Saunders, and Kimmy. There was a great deal of concern that, with the potential side effects of neostigmine, which include bradycardia, hypotension, bronchospasm, and others, significant complications might develop. This was of particular concern because of the large number of our patients who had a history of recent cardiac surgery or cardiac disease. However, the protocol that we have adopted includes the early use of neostigmine. Neostigmine will play a significant role in the future treatment of this syndrome.

We believe that the administration of neostigmine should be done with ECG monitoring and a physician should be present for approximately 20 minutes after the administration of neostigmine.

Dr Dayton, were we unable to predict unfavorable outcomes by examining parameters such as cecal diameter or the period of the patient's cecal dilatation? It seemed that there was no particular correlation, either between cecal diameter or the period of time that the patients had had this problem as related to the subsequent development of perforation or ischemia.

Dr Tyburski, the majority of these patients were out of the ICU and on the wards and were receiving oral intake. Some were receiving a regular diet at the time of the diagnosis. We also considered the possibility of this being some sort of a reperfusion syndrome or a syndrome related to the lack of pulsatile blood flow during cardiopulmonary bypass, but unfortunately that is entirely speculative at this point.

Dr Donovan, we did not require the presence of a splenic cutoff sign on KUB [abdominal roentgenogram] to include a patient with other signs and symptoms of Ogilvie syndrome in our study group.

Dr Folk, higher doses of medications such as narcotics and calcium-channel blockers did not seem to correlate with a greater chance for the development of this syndrome.