Bowel Perforation Caused by Peritoneal Shunt Catheters: Diagnosis and Treatment

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OBJECTIVE: The peritoneum is the preferred site for insertion of shunts used for the treatment of hydrocephalus. Bowel perforation by peritoneal catheters (BPPC) is a rare but devastating complication. Its pathophysiology, diagnosis, and treatment are debated.

METHODS: Retrospective review of cases of BPPC in a series of 1956 patients having a peritoneal catheter followed up for a mean duration of 10.0 years.

RESULTS: Nineteen observations of BPPC, representing 1.0% of the total series. Nine of 19 patients were nonambulatory, and nine of 19 had a previous history of meningeal infection. At the time of diagnosis, only three of the 19 patients had anal extrusion of the catheter, 14 had fever, nine had abdominal signs and symptoms, and six had cutaneous signs of infection. Radiological investigations were often negative or inconclusive. In seven of the patients, the initial diagnosis was shunt failure, and BPPC was diagnosed only during shunt revision. Once the correct diagnosis was made, the treatment was total shunt removal, external drainage, and antibiotic therapy for 2 weeks. Three patients, all severely impaired before BPPC, died, one of meningeal sepsis, the others of multiorgan failure related to spastic tetraparesis. Three were considered shunt-independent, two had a ventriculoatrial shunt, and the others had a new shunt with a peritoneal catheter without complication.

CONCLUSION: BPPC is a neurosurgical emergency. Anal extrusion is present in only a minority of patients; the diagnosis of BPPC is often difficult, delayed, and its incidence is likely underestimated. The majority of patients can be treated with a new peritoneal shunt after cure of the infection.

KEY WORDS: Bowel perforation, Hydrocephalus, Peritoneal shunt

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Since the introduction of shunting for the treatment of hydrocephalus, the peritoneum has gained a wide acceptance as the best absorption site for cerebrospinal fluid. Bowel perforation by a peritoneal catheter (BPPC) is a rare complication, but has generated a large number of clinical reports, detailing perforation of virtually every possible hollow viscus in the abdomen (3, 5, 7, 11, 12, 14, 17). The diagnosis of BPPC is self-evident when the catheter protrudes through the anus; however, many cases of BPPC are recognized only during shunt revision for what was misdiagnosed as shunt obstruction. Several authors warned that "the diagnosis. . .is not always elementary" (15) and that "bowel perforation may be totally unexpected" (14). The pathophysiology of bowel perforation is debated; Di Rocco (3) suggested that bowel erosion results from inflammation caused by pre-existing shunt infection. The surgical management of BPPC is also the matter of some debate, and the indications for exploratory laparotomy, as well as the possibility of further use of the peritoneum, are discussed.

Because few studies have reported more than a few cases, the real incidence, pathophysiology, predisposing factors, elements of diagnosis of BPPC, and the principles of its treatment are unclear. To improve our knowledge of BPPC, we reviewed our database for cases of pediatric hydrocephalus treated for BPPC in our institution.

MATERIALS AND METHODS

We retrospectively reviewed our neurosurgery database for patients treated for hydrocephalus during childhood in our institution who had a diagnosis of BPPC. Our institution is the only referral center for pediatric neurosurgery, serving an area with a population of 4 million. Patients shunted during childhood are systematically followed up in clinics for life, first in

the pediatric and, later, in the adult department of neurosurgery. The diagnosis of BPPC was based on bacteriological findings of bacteria or yeasts of intestinal origin or on such clinical evidence as anal extrusion or the finding of a catheter end dyed by biliary or fecal pigments during operation. We defined the time of the diagnosis of BPPC as the day of catheter removal. The delay to diagnosis of BPPC was the interval between the first clinical manifestations related to BPPC and the day of shunt removal.

At the time of shunt insertion, peritoneal catheters were always inserted under direct vision and never with the trocar technique. The types of shunts have changed during the period of the study, but spring-loaded catheters were never used. We used shunts with a proximal valve, and the length of the distal catheter was shortened as needed to fit the patient's prospective stature; lateral slits were added along the last 10 cm of the catheter to provide extra drainage. The treatment of BPPC was based on total shunt removal, external drainage, and intravenous antibiotics during 2 weeks, followed by the insertion of a new ventriculoperitoneal shunt whenever possible.

We collected information on potential predisposing factors such as the age at shunt insertion; number of shunt revisions before the occurrence of BPPC per annum of follow-up; other abdominal or open neurosurgical operation; ambulatory status; and previous episodes of meningeal infection. We then compared these risk factors in the BPPC and non-BPPC groups. We collected clinical, radiological and operative findings, and bacteriological data in cases of BPPC.

For statistical analysis, continuous variables were studied using Student's *t* test, and binary variables were studied using the χ^2 test, using the commercially available software SPSS 11.5 for Windows (SPSS, Inc., Chicago, IL).

RESULTS

Between March 1980 and January 2004, we diagnosed and treated 19 cases of BPPC. During the same period, we treated 1956 children with a peritoneal shunt catheter, with a mean follow-up duration of 10.0 years. These 1956 children underwent a total of 6041 shunt operations. BPPC affected 1.0% of the patients and represented 5.1% of the occurrences of shunt infection in the whole series. The medical history and clinical presentation of these 19 patients are summarized in *Table 1*. Their surgical treatment, bacteriological data, and outcome are summarized in *Table 2*.

Predisposing Factors

The age at shunt insertion and the number of previous shunt revisions were not significantly different in the BPPC group and in the rest of the series. Although the numbers of nonambulatory patients as well as of patients with a previous history of infection were markedly higher in the BPPC group, the χ^2 could not be calculated because of too small a number of patient in the BPPC group. These results are presented in *Table 3*.

The mean age at the time of BPPC was 9.6 years (3.5 mo–30.5 yr). Among these 19 patients, nine (47.4%) were nonambulatory, eight (42.1%) had mental retardation, nine (47.4%) had a history of meningeal infection or inflammation in the perinatal period or a previous episode of shunt infection, four (21.1%) had a history of abdominal surgery, and three others had bowel dysfunction related to myelomeningocele. Two patients had open neurosurgery, one for posterior fossa decompression and the other for spinal cord untethering before BPPC. Overall, only four patients had none of these potential predisposing factors.

Diagnosis

The median interval between the last shunt revision or neurosurgical operation and the diagnosis of BPPC was 2.4 years (5 d–10.3 yr). One patient had intraoperative BPPC; this patient had enteritis before surgery, which may have facilitated perforation, and he developed abdominal symptoms and fever within the days after shunt insertion. All other patients developed BPPC at a distance from surgery. The median interval between the first symptoms and the diagnosis of BPPC was 30 days (5 d–6 mo). At the time of diagnosis, fever was present in 14 patients (73.7%); abdominal signs and symptoms such as constipation, diarrhea, abdominal pain, and tenderness, were present in nine (47.4%) patients. Signs of inflammation along the shunt track were present in seven (36.8%) patients, but anal extrusion of the catheter was noted in only three (15.8%). Signs of raised intracranial pressure and meningism were present in 12 (63.2%)and six (31.6%) patients, respectively. Raised intracranial pressure was the sole presenting symptom in two patients.

The patient with intraoperative bowel perforation had radiological evidence of pneumoperitoneum (*Fig. 1*). In the other patients, abnormal radiological features, such as the absence of mobility of the peritoneal catheter (*Fig. 2, A–C*), were noted inconstantly and only in retrospect. Computed tomographic scanning and ultrasonography showed occasionally a thickened bowel wall and mucosa and focal peritonitis (*Fig. 2D*). Overall, in the majority of cases, the radiological study of the abdomen was negative or unspecific, except for one patient who had shuntography, which showed opacification of an intestinal loop. On the other hand, brain computed tomographic scanning showed enlarged ventricles in eight patients.

Biological signs of inflammation were present in 11 patients; a cerebrospinal fluid study showed pleocytosis with positive culture in 10 patients. In 10 patients, the culture of the peritoneal catheter grew three or more different organisms; the most common bacteria were Escherichia coli and Group D Streptococcus (10 patients each).

Surgery

The patient with intraoperative BPPC underwent emergency laparotomy. Emergency laparotomy was also per-

TABLE 1. Indication for shunting, medical history, and clinical findings at the time of the diagnosis of bowel perforation[a]

Patient no.	Age (yr)/ sex	Antecedents	Delay since last surgery (mo)	Duration of symptoms (d)	Inflammation	Signs of shunt infection	CSF cytosis and culture	Abdominal symptoms
1	14/M	Meningitis, Listeria; Chiari decompression	8.6	183	Fever, blood	None	Positive	Occlusion
2	22/F	Apert syndrome ; appendicitis	18.3	21	Fever, blood	Meningitis	Positive	Pain
3	0.5/M	Neonatal hemorrhage; meningitis, Strepto B	5.0	0	Blood	Extrusion	Positive	None
4	5/F	Epidermolysis, meningitis, Staph A	10.8	48	Fever, blood	None	Negative	Occlusion
5	11/F	Meningitis, Proteus	123.0	50	Fever, blood	Meningitis	Positive	Occlusion
6	0.3/M	Operated pyloric stenosis; enteritis	5 days	4	Fever	None	NA	Peritonitis
7	14/M	Myelomeningocele; spinal cord untethering	20.3	16	Fever	Meningitis	Positive	None
8	20/F	Appendicitis, peritonitis, shunt nephritis	31.5	170	Fever, blood	Skin, meningitis	Positive	None
9	3/M	None	11.8	30	Fever, blood	Skin	Negative	None
10	1/F	None	6.9	90	Fever	Skin, extrusion	Negative	None
11	0.8/M	Congenital toxoplasmosis	4.5	NA	Fever	None	Positive	Pain
12	26/M	None	105.8	53	Fever	Meningitis	Positive	Peritonitis
13	2/M	Occipital meningocele; shunt infection, Staph	4.7	NA	None	None	Positive	None
14	12/F	Shunt infection, Staph	51.9	7	Fever, blood	None	Negative	Diarrhea
15	3/F	Myelomeningocele	7.6	23	Fever, blood	Skin	NA	Peritonitis
16	30/M	None	35.8	59	Blood	Meningitis	Positive	None
17	0.9/M	None	7.3	NA	Fever, blood	Skin, extrusion	Negative	None
18	11/M	Glutaric aciduria; intestinal occlusion	97.6	30	None	Skin	NA	None
19	5/M	Myelomeningocele	4.8	NA	NA	None	NA	None

[a] CSF, cerebrospinal fluid; Strepto, Streptocoques; Staph, Staphylococcal; NA, not available.

formed in two cases of delayed BPPC because the patient presented initially with high fever and an acute abdomen, and the relation to the shunt was not suspected. In seven (36.8%) other patients, the initial diagnosis was shunt obstruction, and the diagnosis of BPPC was made only during shunt revision. Overall, an abnormal color of the peritoneal catheter was noted in 12 patients. In one patient, the distal end of the peritoneal catheter was extracted with difficulty because it was firmly adhering. Although its color was not abnormal, the culture grew Group D Streptococcus.

When the diagnosis of shunt infection was made, the general practice was to remove the peritoneal catheter and maintain external drainage and intravenous antibiotics for 2 weeks. When cutaneous inflammation was present, the whole shunt was removed, and a frontal external drain was inserted. After the infection was cured, a new shunt with a peritoneal catheter was inserted in 14 patients. Three patients were considered shunt independent. Two patients had a ventriculoatrial shunt inserted; in one of these (Patient 19), the peritoneal shunt was converted to atrial as initial surgery. That patient later developed shunt nephritis because of continuing infection, which required total shunt removal followed by insertion of a new peritoneal shunt.

Outcome

Three (15.8%) patients died 17 days to 6 months after BPPC. All of these were severely debilitated with spastic tetraparesis before BPPC; one died of uncontrolled meningitis despite shunt removal and external drainage; the two others died in spite of a good control of the infection because of multiorgan failure related to their pre-existing status. These three patients represented 2.4% of nontumor-related deaths in the whole series. The duration of follow-up after BPPC for the 16 survivors was 93.8 months, during which five underwent a new shunt revision, always using the peritoneum as absorption site.

DISCUSSION

Incidence

From our data, we can calculate that the risk for a given shunted patient to develop BPPC is one every 1000 years. In

TABLE 2. Surgical treatment for bowel perforation, bacteriological data, final shunt insertion, and outcome[a]

Patient no.	Surgery	Bacteriology	New shunt	Outcome	Follow-up (mo)
1	Laparotomy, external drainage	Haemophilus, E. coli, Acinetobacter, Pseudomonas, Strepto D	Peritoneal	Dead, tetraparesis	0.6
2	External drainage	E. coli, Proteus, Strepto D, Candida	Peritoneal	Good	42.2
3	External drainage	E. coli, Morganella, Staph A, Strepto D	Peritoneal	Good	22.2
4	Removal	Strepto D, E. coli, Klebsiella	Peritoneal	Good	20.3
5	External drainage	E. coli, Proteus, Pseudomonas	Peritoneal	Good	93.5
6	Removal	E. coli, Proteus, Strepto D	Peritoneal	Good	63.7
7	External drainage	Multiple bacteria, Yeast	Peritoneal	Good	35.9
8	External drainage	E. coli, Proteus, Strepto D	Peritoneal	Good	108.1
9	External drainage	E. coli, Strepto D, Pseudomonas	Peritoneal	Good	119.4
10	Removal	E. coli, Proteus, Klebsiella	Peritoneal	Dead, infection	6.0
11	External drainage	Strepto D, gram-negative rod	Peritoneal	Good	260.1
12	Laparotomy, external drainage	Strepto D, multiple germs	Atrial	Sigmoidectomy for diverticulosis	18.2
13	External drainage	E. coli, Strepto C	Peritoneal	Good	212.6
14	External drainage	Citrobacter	Peritoneal	Good	6.9
15	External drainage	Staph A	Peritoneal	Good	157.8
16	External drainage	Citrobacter	None	Dead, infection	2.3
17	Removal	Proteus	None	Good	3.9
18	Removal	NA	None	Good	151.8
19	Conversion to atrial, laparotomy	NA	Atrial	Shunt nephritis, conversion to peritoneal	185.0

[a] E. coli, Escherichia coli; Strepto, Streptocoques; Staph, Staphylococcal; NA, not available.

TABLE 3. Comparison of the study group (n = 19) with the contrast group (n = 1937) treated with peritoneal shunts during the same period of time[a]

	Perforation	No perforation
No. of patients	19	1937
Age at shunt insertion (mo)	13.9	26.9
History of infection (%)	9 (47.4)	491 (25.1)
Nonambulatory (%)	9 (47.4)	176 (9.0)

[a] Although none of these differences attained statistical significance, the patients with bowel perforation by a peritoneal catheter were much more likely to be nonambulatory and have a history of meningeal infection.

most publications on BPPC, its incidence is estimated between 0.1 and 0.7% of shunted patients. Our figure of 1.0% is higher, which can be ascribed to our relatively long follow-up, but also to a high proportion of cases without anal extrusion. Literature reviews show catheter extrusion as the presenting symptom in approximately half the cases of BPPC (9, 13, 19), but this finding is probably over-represented in case reports. BPPC without catheter extrusion is likely under-diagnosed and might account for a number of cases of unexplained shunt infection and sudden death. Byard et al. (2) reported a case of BPPC diagnosed postmortem and advised pathologists to examine systematically the tip of the peritoneal catheter during the autopsy of shunted patients.

Pathophysiology

Although spring-loaded catheters have become notorious for their ability to perforate virtually any viscus in the abdomen (3, 5, 7, 12), other reports have shown that unloaded silicon catheters are also responsible for bowel perforation (9, 11, 14, 17).

Bowel perforation can occur intraoperatively, as a complication of catheter introduction. Although the use of the trocar method is considered safe (6), many authors prefer to approach the peritoneum under direct vision (4). Although we adhere to this principle, intraoperative BPPC occurred in one of our patients who had a scarred abdomen and enteritis. In the majority of cases, however, BPPC was delayed after surgery, suggesting that it resulted from a chronic inflammatory process rather than a traumatic event. Occasionally, the catheter perforates the viscus at the site of a previous surgery (5, 10). Di Rocco (3) suggests that patients with myelomeningocele may be at increased risk for BPPC. In our study, three patients had a myelomeningocele, and nonambulatory patients represented almost half of the series. Although these data suggest that an altered motility of the intestines might facilitate mechanical trauma by the catheter, more patients will be needed to test this hypothesis.

BPPC is obviously a cause of shunt infection, but an interesting hypothesis is that it might in some cases be the result of occult shunt infection caused by intraoperative contamination (3). Several cases of contamination by Staphylococcus aureus (11) or Staphylococcus epidermidis (7, 8) have been reported in cases of BPPC. In several occurrences, skin inflammation or breakdown over the catheter was present long before the diagnosis of bowel perforation was made (1, 15), suggesting that the shunt was infected before perforating the bowel. In this perspective, BPPC can be likened to a mechanism of rejection of an infected foreign body.

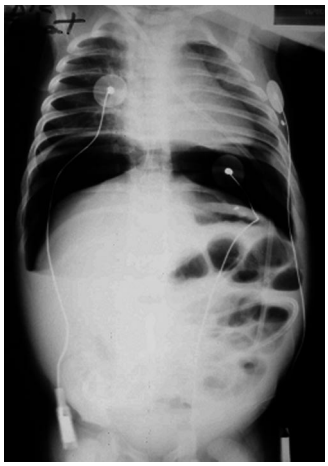

FIGURE 1. *Patient 6. Three-month-old male with history of operated hypertrophic pyloric stenosis, shunted for subdural collection. In addition, patient had enteritis at the time of operation. Five days after surgery, he presented with acute abdomen and high-grade fever. Plain abdomen radiographs showed obvious pneumoperitoneum. The child underwent removal of shunt and emergency laparotomy with suture of bowel perforation. A new subduroperitoneal shunt was inserted 3 weeks later without complication.*

Diagnosis

Bowel perforation may manifest itself immediately after surgery or several years later (3, 16). The diagnosis is evident in the case of anal extrusion (*Fig. 3*). However, in our experience, this represents only a minority of cases. In another subset of patients, signs of sepsis along the catheter track raise suspicion of shunt infection (3, 9, 10). In our series, fever and biological markers of inflammation were rarely lacking in the event of BPPC, but these features are often considered trivial in children. Abdominal symptoms were present in less than half of the cases and were easily overlooked, especially in severely disabled and nonverbal patients.

The radiological findings are often negative or inconclusive in cases of BPPC. One interesting feature on plain radiographs is the absence of motion of the catheter on seriated images (3, 8). The abdominal computed tomographic scan with contrast and ultrasonography may be helpful, showing local inflammation and a thickened muscular layer and mucosa, but is often negative. The shuntogram makes the diagnosis of BPPC when it shows direct visualization of the haustral pattern of the intestine (14, 15), but it is rarely performed.

The main differential diagnosis of BPPC is shunt obstruction, to which it is commonly associated. In our series, BPPC was often diagnosed only during shunt revision when the tainted peritoneal catheter was exposed. Such late diagnosis incurs the risk of meningeal contamination. From our experience, we have learned to be very vigilant for abdominal signs and clinical and biological signs of inflammation. In any case of doubt, cerebrospinal fluid should be obtained from both lumbar puncture and tapping of the shunt, and a shuntogram should be performed.

Treatment

The treatment of BPPC is a surgical emergency. It is based on shunt removal, external drainage, and intravenous, broad-spectrum antibiotics, followed by insertion of a new peritoneal shunt after 2 weeks. Atrial shunt is indicated only if the abdominal problem is not solved at the time of shunt insertion. Laparotomy is made only in cases presenting with an acute abdomen, particularly in case of intraoperative BPPC; in all other cases, a consensus has emerged in favor of simple

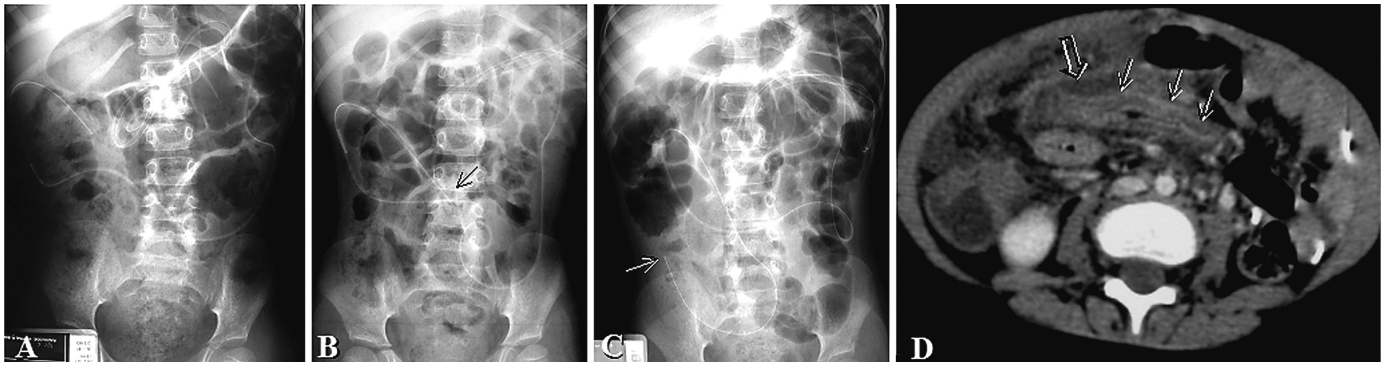

FIGURE 2. *Patient 4. Five-year-old female with epidermolysis diagnosed at birth, complicated with staphylococcal meningitis and septated hydrocephalus requiring two peritoneal shunts. Shunt on left side of abdomen was last revised in January 2003.* A, *postoperative abdomen radiograph obtained in January 2003, after last shunt revision.* B, *systematic abdomen radiograph obtained in May 2003 showing that, whereas the revised catheter was free in the left part of the peritoneal cavity, the right catheter was stuck in the right part of the abdomen* (arrow). C, *in October 2003, the child was hospitalized again for severe abdominal pain and low-grade fever; C-reactive protein was over 200 mg/L. More recent radiograph showing that the right catheter is still stuck and suggests it has perforated the bowel and progressed inside the lumen* (arrow). D, *abdominal computed tomographic scan showing thickened bowel wall* (small arrows) *and limited peritoneal collection* (large arrow). *Both shunts were removed the following day; the peritoneal catheter was dyed with biliary pigment and grew three different organisms. A new peritoneal shunt was inserted 2 weeks later with good outcome.*

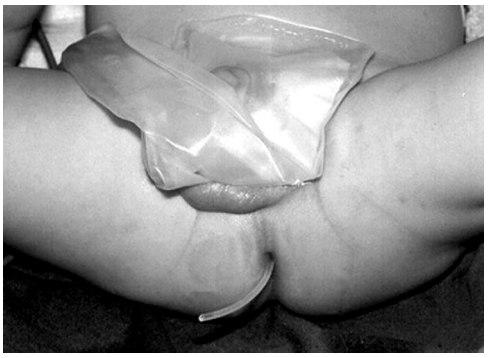

FIGURE 3. *Patient 3. Seven-month-old male with hydrocephalus caused by neonatal ventricular hemorrhage. Catheter was found protruding from an otherwise normal child; he was immediately referred to the neurosurgery department and underwent operation the same day. The peritoneal catheter was exposed, sectioned, and its proximal segment connected to external drainage bag while its distal segment was removed by simply pulling it down through the anus. Cerebrospinal fluid showed pleocytosis and grew four different strains of bacteria. After 16 days of external drainage and intravenous antibiotics, a new ventriculoperitoneal shunt was inserted with good outcome.*

surveillance and fasting for 2 days before resuming oral uptake (3, 9). Recent reports have shown that colonoscopy can be helpful to remove the distal catheter, especially when the extruded catheter has retracted inside the patient's body (1, 19). In cases without catheter extrusion or when the catheter had not retracted, however, we do not think that endoscopy is indicated.

BPPC should be prevented as much as possible. We prefer to introduce peritoneal catheters under direct vision, especially in scarred abdomens. Prevention of delayed bowel perforation justifies systematic removal of all unnecessary catheters (5, 18).

CONCLUSION

The diagnosis of BPPC is often difficult and delayed because anal extrusion is present in only a minority of cases, and signs of shunt infection are often lacking or are overlooked. The diagnosis of visceral perforation should be considered in all shunted patients presenting with unexplained fever or prominent abdominal symptoms. When these patients are referred for shunt failure, a high level of suspicion is necessary to correct the diagnosis. The surgical treatment is based on shunt removal, external drainage, followed by insertion of a new shunt. After BPPC, the peritoneum retains its absorptive capacity and is still the preferred site for shunt placement.

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COMMENTS

The authors have nicely reviewed their substantial experience with bowel perforation complicating peritoneal shunts. The number of patients accessible for study reflects the fact that the shunted children, as they reach adulthood, are followed in the same neurosurgical clinic. It also relates presumably to a relatively stable population. I agree that the higher incidence of this complication among their shunted patients is likely a reflection of the longer period of follow-up, rather than some other factor in their management. It is instructive that the likelihood of bowel perforation appears to be higher in nonambulatory patients. This is disquieting in light of the fact that these are also the patients who are less likely to effectively communicate their symptoms to the physician, thus delaying diagnosis and treatment.

Paul H. Chapman
Boston, Massachusetts

This is a retrospective review identifying 19 of 1956 patients who experienced what the authors determined was intestinal perforation of a peritoneal catheter over an almost 50 year period. The authors identified these patients by clear evidence of intestinal perforation or growth of multiple entereal organisms in the presence of a distal peritoneal catheter problem. While three patients presented with the dramatic anal extrusion, the others presented in a variety of ways, often with signs of infection, although some presented as what appeared to be straightforward shunt obstruction. All of the patients were treated by having total shunt removal, external drainage and antibiotic therapy for two weeks, followed almost always by re-insertion into the abdomen. Three patients died. More of the patients with the intestinal perforation were non ambulatory or had a history of previous infection compared to the patients without this complication.

This is an excellent review of a rare but very serious problem. By looking at a very large group of patients followed for a long time period they identified a substantial number of patients. In addition to alerting surgeons to the possibility of this complication, they outline how they may be successfully managed without a laparotomy and temporary colostomy.

James M. Drake
Toronto, Ontario, Canada

This is a good analysis of a rare but important complication of V-P shunt surgery. A casual reading of the manuscript might lead the reader to the conclusion that BPPC is common enough to be suspected in close to 1% of V-P shunt patients, when in fact there were only 19 incidents documented in this study where 6,041 total shunt operations were done. Some of those operations were to treat BPPC, but that would represent a very small proportion of the cases. Most of the cases were the result of infection or poor protoplasm as opposed to technical errors and the real value of this study is pointing out diagnostic and treatment strategies.

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The paper analyzes one of the known complications of CSF shunt devices draining into the peritoneal cavity, which is the perforation of a viscus. Because of its usually "impressive" clinical presentation, the complication was quite often reported as an anecdotal case. The present paper represents the first attempt to evaluate the incidence and the predisposing factors in a large series of patients followed for a sufficiently long period of time. The new information provided is the higher incidence of the complication as compared to previous figures, mainly due to the detection of cases in which the catheter was not extruded through the anus, the bladder, the vagina or the abdominal wall. A further important point relates to the pathogenesis of the complications, as the authors' experience appears to rule out the use of spring-loaded catheters and of a trocar for the shunt implantation. Both maneuvers were in fact often blamed for the complications.

Actually, we observed only two cases of viscus perforation, one by a spring-loaded catheter and the second by a "standard" silastic peritoneal catheter in a series of more than a thousand patients operated on in the last 30 years. In particular, we utilized the spring-loaded catheter in a large proportion of these children and we penetrated the abdominal wall by using the trocar in all the cases. Consequently, our experience appears to confirm that spring-loaded catheters and trocar penetration of the abdominal wall (with the possible exclusion of children with myelodysplasia and distended bladder) are innocent in inducing a viscus perforation, or at least as guilty as other types of shunt and surgical maneuvers to access the peritoneal catheter.

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FUTURE MEETINGS—CONGRESS OF NEUROLOGICAL SURGEONS

The following are the planned sites and dates for future annual meetings of the Congress of Neurological Surgeons:

2006	Chicago, IL	October 7–12
2007	San Diego, CA	September 15–20
2008	Orlando, FL	September 20–25

FUTURE MEETINGS—AMERICAN ASSOCIATION OF NEUROLOGICAL SURGEONS

The following are the planned sites and dates for future annual meetings of the American Association of Neurological Surgeons:

2006	San Francisco, CA	April 22–27
2007	Washington, DC	April 14–19
2008	Chicago, IL	March 29–April 3
2009	San Diego, CA	May 2–7