Journal of Visceral Surgery (2019) 156, 423—431

Available online at
ScienceDirect
www.sciencedirect.com

Elsevier Masson France
EM|consulte
www.em-consulte.com/en

REVIEW

Update about ventriculoperitoneal shunts: When to combine visceral and neurosurgical management?☆

A. Sellier[a,*], T. Monchal[b], C. Joubert[a], S. Bourgouin[b], N. Desse[a], C. Bernard[a], P. Balandraud[b,c], A. Dagain[a,c]

[a] *Service de neurochirurgie, hôpital d'instruction des armées Sainte-Anne, 83000 Toulon, France*
[b] *Service de chirurgie viscérale, hôpital d'instruction des armées Sainte-Anne, 83000 Toulon, France*
[c] *Service de santé des Armées, École du Val-de-Grâce, 75005 Paris, France*

Available online 7 May 2019

KEYWORDS
Ventriculoperitoneal shunt;
Hydrocephalus;
Pseudo-cyst;
Peritonitis;
Laparoscopy

Summary Ventriculoperitoneal shunts (VPS) are the treatment of choice for chronic hydrocephalus. However, the rate of abdominal complications is far from negligible. Combined abdominal and neurological surgical management is often necessary. The goal of this study was to describe the abdominal complications related to VPS and their management. This update overviews: (1) acute or chronic abdominal complications after insertion of a VPS, especially those that call for involvement of visceral surgeons; and (2) the particular precautions necessary when neurosurgeons and visceral surgeons have to collaborate in case an abdominal operation is necessary in patients with a VPS.

Introduction

Hydrocephalus involves approximately 40 out of 100,000 persons [1]. The neurosurgical treatment recommended for chronic hydrocephalus in the adult as well as in the child is insertion of a ventriculoperitoneal shunt (VPS), which transfers the cerebrospinal fluid (CSF) from the ventricular system of the brain to the peritoneal cavity, in order to prevent increased intracranial hyper-pressure. Because of their localization, VPS are associated with a non-negligible rate of abdominal complications, ranging from 5 to 47% [2]. Consequently, visceral surgeons may be called upon to manage these complications conjointly with neurosurgeons. Moreover, the presence of a VPS in patients that need to undergo an abdominal surgical procedure is frequent and this warrants specific precautions. This update covers:

- the acute or chronic abdominal complications after insertion of a VPS, and in particular, those that require management by a visceral surgeon;
- the particular precautions that must be taken conjointly by two specialties, neurosurgeon and visceral surgeons, when dealing with abdominal surgery in patients with a VPS.

Generalities

Hydrocephalus is defined as disorder of CSF circulation, with accumulation that can lead to increased fluid volume

☆ Opinions or affirmations expressed herein are the personal opinions of the authors and should not be considered official or reflecting the views of the French military health service.

* Corresponding author. Hôpital d'instruction des armées Sainte-Anne, 1, boulevard Sainte-Anne, BP 20545, 83041 Toulon cedex 9, France.
E-mail address: aurore.sellier@live.fr (A. Sellier).

https://doi.org/10.1016/j.jviscsurg.2019.03.003
1878-7886/

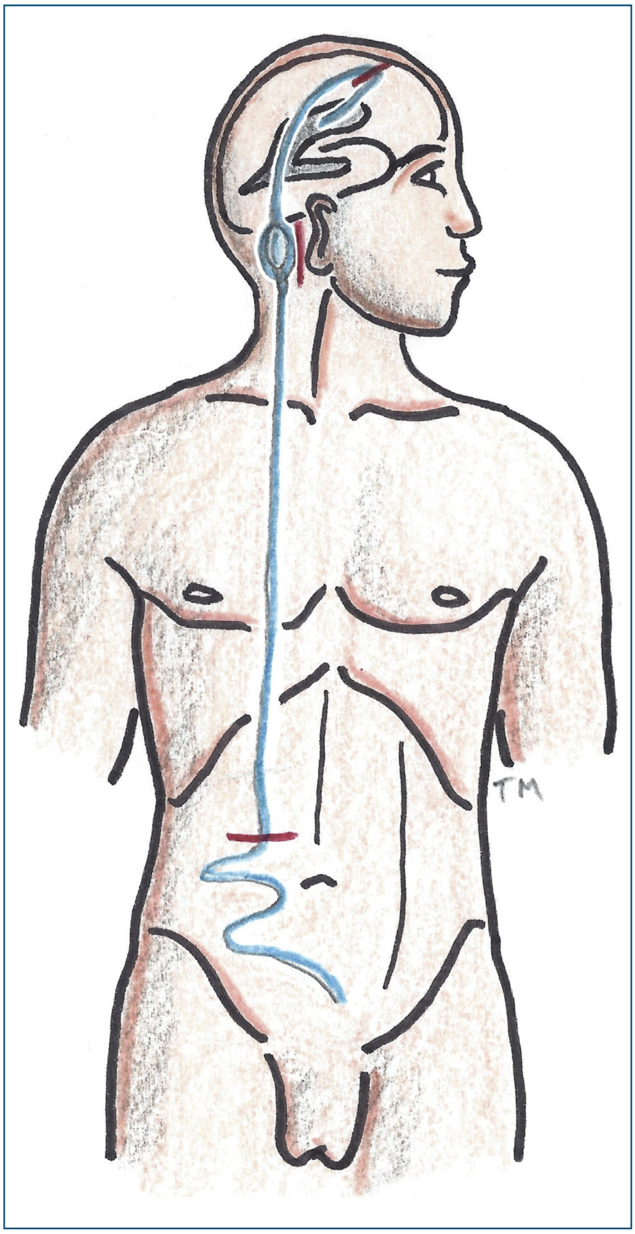

Figure 1. Schema of ventriculoperitoneal shunts (VPS) (drain in blue, skin incisions in red).

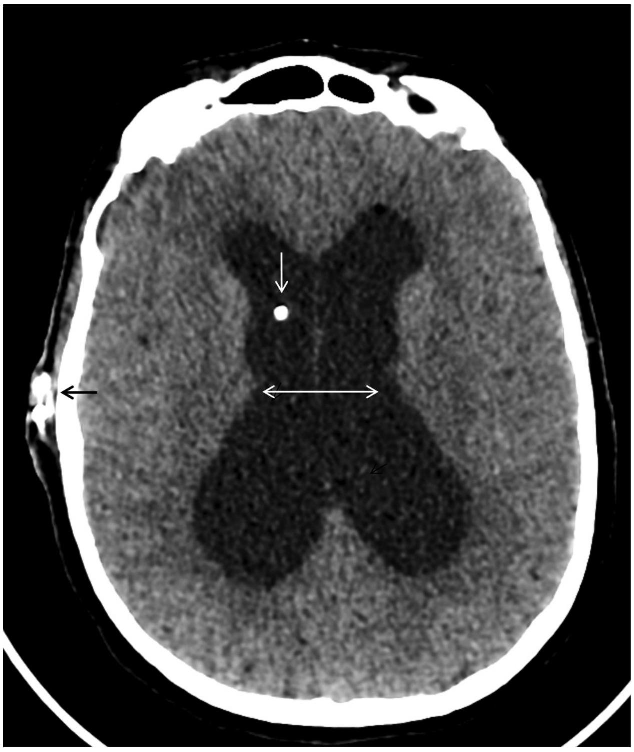

Figure 2. Axial slice CT scan centered on the ventricles, after insertion of a ventriculoperitoneal shunt: ventricular dilatation (double white arrow), the drain is correctly in place in the frontal horn of the right lateral ventricle (single white arrow), and body of the subcutaneous valve (black arrow).

in the cerebral ventricles. First used by Kausch in 1905 [3], VPS are used to decompress the CSF by transferring the fluid to the intra-abdominal space (Fig. 1). The peritoneum is the ideal space for the shunt because of its rapid and efficient reabsorptive capacity. VPS can treat all causes of hydrocephalus, whether due to overproduction of CSF, or obstruction between the ventricles and the sub-arachnoid spaces. The causes are variable: infectious, neoplastic, traumatic, vascular, congenital or idiopathic (when the intracranial pressure of the hydrocephalus is normal).

VPS are created under general anesthesia, the patient supine, head rotated to the side opposite from the derivation. A frontal circular incision is made, then a burr-hole is performed 3 cm from the midline, in front of the coronal suture line. A ventricular drain is inserted into the burr-hole perpendicular to the cranial vault, into the anterior horn of the lateral ventricle (Fig. 2). A 4 cm horizontal incision is made in the homolateral para-umbilical area. A subcutaneous guide-wire is used to connect the two approaches from below to above, after performing a counter-incision behind the ear. The catheter is inserted over the guide-wire and runs from the abdominal to the frontal scalp incisions. Connectors are used to attach the catheters and the pressure valve. A valve is necessary for regulation of the quantity of fluid flow. Two types of valves are available: adjustable pressure valves, that allow flow as needed, or fixed pressure valves that work when a prefixed threshold value is reached, and above which the valve diverts the CST. The latter is currently the most widely used. The valves can be programmed using a handheld magnetized or ultrasonic device applied trans-cutaneously (similar to modern pacemakers). Some devices also have an integrated anti-siphon or anti-gravitational system that limit the siphoning effect related to changing positions from decubitus to the upright position [4]. A reservoir can be inserted between the valve and catheter, allowing the surgeon to test the shunt function or to withdraw fluid for lab tests.

Finally, the peritoneum is opened over a half centimeter after opening the anterior and posterior fascia of the rectus muscle. The catheter is inserted 30 cm (or more in pediatric patients, to compensate for future growth of the child). The length of the catheter should be enough to limit the number of surgical revisions during the growth period and the length is usually calculated according to available growth curves [5]. The direction and placement of the drainage catheter in the intraperitoneal space is difficult to foresee, as it is introduced blindly through a mini-laparotomy. Several recent studies are in favor of introducing the distal end of the VPS via laparoscopy, and to control visually the placement and position of the distal end [6—9]. In this setting, some authors recommend placing the catheter behind the liver [10].

If the patient presents any contraindication to the peritoneal location such as adhesions or a history of extensive abdominal surgery, alternative drainage sites such as ventriculo-atrial or ventriculo-pleural shunts may be envisioned. Lumbo-peritoneal shunts are another alternative that avoids contact with the cerebral parenchyma. These are used more and more often today in Japan [11]. Other rarely-used techniques have been described, including derivation into the gallbladder, the bladder or the superior sagittal sinus [12—14]. Endoscopic ventriculocisternostomy allows treatment of non-communicating hydrocephali when an obstacle is identified in the outflow channel between the third and fourth ventricles. This technique does not require insertion of any foreign material [15] and can be useful to resolve problems related to dysfunction or infection of a VPS.

The two main neurological complications of VPS are excessive-drainage and acute hydrocephalus. Excessive-drainage leads to intracranial hypotension, potentially responsible for chronic or acute subdural hematoma that can be life-threatening. Conversely, VPS dysfunction can lead to acute hydrocephalus when the CSF is insufficiently drained. Signs of intracranial hypertension (ICHT) such as headache, nausea, vomiting, impaired consciousness or even coma (potentially leading to death) may occur.

The rate of abdominal complications related to VPS is not negligible, ranging from 5 to 47% [2], making VPS the most complicated medical device in modern medicine [4]. In the postoperative period, complications such as peritonitis, intestinal perforation, misplacement, parietal hematoma, cutaneous necrosis, or intestinal obstruction with occasional volvulus have been observed. Other late complications are possible including cutaneous fistula at the level of the anterior abdominal wall, inguinal hernia, intraperitoneal pseudo-cyst, ascites, catheter migration or incisional hernia [16,17]. Other rare complications include hydrocele, diffusion of cerebral metastases to the abdomen via the VPS, omental impaction, migration of the distal end of the catheter through the anus or into the urethra, bladder or other organs, or even migration of the entire catheter into the abdomen [18,19].

Visceral surgeons are often called in to treat some of these complications that comprise true neurological or visceral emergencies. In these situations, close formal concertation between the neurosurgeon and visceral surgeon is necessary. The evolutive profile of patients with hydrocephalus is variable and can be life-threatening or endanger the neurologic functional prognosis in the short-term. Schematically, VPS inserted in adults for slowly-evolving communicating hydrocephalus can be withdrawn temporarily because symptoms reappear slowly and progressively. Conversely, in patients with non-communicating hydrocephalus, or patients with a valve inserted in childhood, degradation can occur within a few hours, with altered mental status and death, and in these patients, the VPS cannot be removed without major risks. Contact with the neurosurgeon in charge of the patient should be systematic in this setting.

Acute abdominal complications

Infections

Catheter infections represent the principal cause of malfunction of VPS. Notwithstanding the numerous potential preventive measures to decrease the infective risk, the incidence of postoperative infections after insertion of VPS remains high, ranging from 0 to 35% according to the studies [20]. Seventy percent of these infections are symptomatic within one month of catheter insertion while 90% manifest during the first nine months, implying that most of these infections originate during the surgical procedure [16]. The main infective source is the cutaneous flora, with 2/3 of infections stemming from *Staphylococcus epidermidis*, mainly coagulase-negative (followed by *Staphylococcus aureus*), most often introduced during surgery. Six to 20% of cultures contain gram-negative bacilli (above all *Pseudomonas*, *Escherichia coli* and *Klebsiella*), originating from the gastrointestinal tract [21]. Patients have fever in 14 to 90% of cases and an inflammatory syndrome is present in 25% of infections [1].

Figure 3. Septic complication one month after ventriculoperitoneal shunt revision with inflammatory abdominal and retro-auricular scars. 60 mm intraperitoneal fluid collection (white arrow) in contact with the ventriculoperitoneal shunt (black arrows).

Clinical manifestations related to the proximal infection of the catheter include easily recognized symptoms of meningitis or ICHT, but these are inconsistently present. Distal infections are responsible for abdominal symptoms including signs of peritoneal irritation upon examination (localized abdominal guarding or rigidity). Gastrointestinal signs (vomiting or even intestinal obstruction) can also be associated [22].

Ventricular dilatation found on cerebral CT scan is indicative of shunt dysfunction. Abdominal CT scan should be scrutinized for abnormalities along the course of the shunt, specifically at the level of the distal extremity (intra- or pre-peritoneal collection near or around the catheter or its free end in the peritoneal cavity), or the presence of associated entities such as pseudo-cystic loculation or ascites, or signs of localized peritonitis: infiltration of intraperitoneal fatty tissues, thickened small intestinal loops or adynamic ileus, collected abscess (Figs. 3 and 4). In the case of hollow viscus perforation, aside from the above-described signs and symptoms, pneumoperitoneum is inconsistently found [23].

Blood cultures should be drawn routinely but cultures are positive in only 23% of cases in patients with VPS, whereas their etiologic value is much higher in ventriculo-atrial shunts where 95% of cultures are positive [24]. Lumbar puncture, direct transcutaneous puncture of the reservoir or cultures when fluid is withdrawn during external ventricular shunt are the main diagnostic tools. Blood samples often show pleocytosis, with predominant neutrophil leukocytosis, associated with CSF showing high protein and low glucose levels. However, these signs can be lacking,

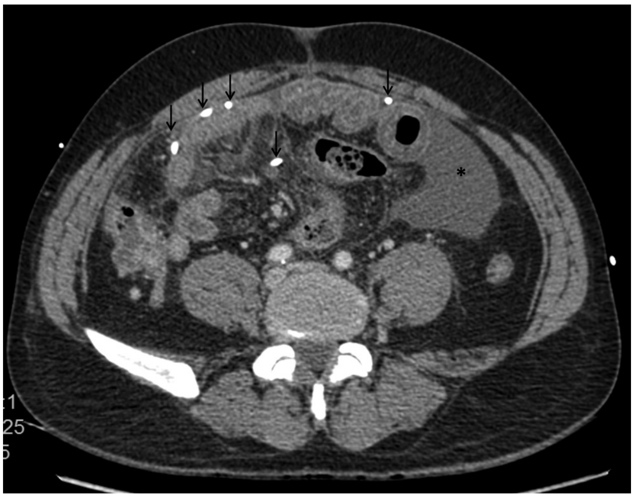

Figure 4. Obstruction syndrome two months after ventriculoperitoneal shunt insertion with signs of peritoneal irritation on physical examination. CT scan shows signs of peritonitis with free fluid (black asterisk), infiltration of mesenteric fatty tissues in contact with the ventriculoperitoneal shunt (black arrows) and adynamic distension of small bowel loops.

especially when infection is distal. Likewise, CSF cultures can be negative if retrograde colonization has not taken place. Thus, if the patient presents an acute abdominal syndrome, treatment should not be delayed waiting for the results of these cultures, and symptoms disappear six to 12 hours after removal of the catheter [25]. Identification of the germ (in the CSF or in blood cultures) can direct the research of the portal of entry, cutaneous or gastrointestinal (cf. supra) and help in the choice of an adapted antibiotic therapy. Emergency treatment is early ablation of the shunt and immediate intravenous antibiotic therapy. The shunt can be removed under local anesthesia. However, if there is associated disease, such as pseudo-cyst or abscess, laparoscopy is recommended to perform peritoneal lavage and establish intraperitoneal drainage. Indeed, whenever possible, laparoscopy has become the reference for abdominal complications related to VPS [16,19]. A new internal shunt can be inserted at a distance from the site of infection. During this interval, a temporary solution is to insert an external shunt or to create a ventriculostomy, or even divide and remove the abdominal catheter and exteriorize the distal portion, if there are no associated signs of meningitis and the patient is stable. First-line antibiotics should have a good meningeal diffusion and be active against *staphylococcus* and gram-negative bacteria. An alternative is to introduce antibiotics (most often vancomycin) directly into the ventricles, especially when systemic treatment has failed [26]. The shunt should be closed off for 30 to 60 min during the ventricular perfusion.

Visceral wounds and perforations

Organ perforations after insertion of VPS, while very rare, can occur up to 10 years after surgery, with an attendant mortality rate of 15% [27]. Perforation can be chronic or acute. Chronic perforation occurs at distance from surgery and will be dealt with later in the chapter on chronic complications.

When perforation occurs during the operation, treatment must be immediate. Most often the organ involved is the small intestine. In the case described by Vinchon et al. [23], the intraoperative perforation occurred in a patient who had already presented signs of enteritis before surgery, and had a long history of multiple antecedent abdominal surgeries, which could have facilitated the perforation. Management of this patient included successively, conversion to laparotomy to suture the visceral perforation and creation of a ventriculo-atrial shunt. Maintaining a peritoneal drain during the same operation is not recommended because of the very high septic risk in this setting.

Since the first case published in 1993 [7], laparoscopic insertion of the peritoneal catheter of VPS has developed, thus avoiding blind introduction and reducing the risk of perforation. In a systematic review published by Phan et al. [28], identifying 10 series of VPS inserted via laparoscopy or mini-laparotomy published before February 2015, no statistically significant difference was found between the two approaches with regard to the rate of complications, or infections, or the duration of hospital stay. Laparoscopy was associated with a slight reduction in the duration of operation, fewer abdominal VPS misplacements, distal obstructions and dysfunction. Naftel et al. [29] compared the complication rates after VPS in a cohort of 810 patients who had undergone either laparoscopy or traditional mini-laparotomy. There was no statistically significant difference found in terms of VPS dysfunction or postoperative infection rate. Conversely, duration of operation was statistically significantly shorter with laparoscopy and the risk of distal failure was 7.5 times higher after mini-laparotomy compared to laparoscopy ($P < 0.0005$ and $P = 0.014$, respectively). However, the rate of hollow organ perforation was not specifically analyzed in these studies. Schucht et al. compared these two surgical approaches in their randomized clinical study of 120 patients published in 2016 [9]. Again, no statistically significant difference was found with regard to the rate of complications but there were statistically significantly more misplacements or secondary distal catheter migrations in the mini-laparotomy group (8% vs. 0%) than in the laparoscopy group ($P = 0.029$). There were, however, two intestinal perforations in the laparoscopy group, owing to peritoneal adhesions in patients with antecedent surgery, and requiring conversion to an open procedure. No statistically significant difference in duration of operation or hospital stay was found between both groups.

Overall, these different studies reported a decreased rate of distal catheter misplacement after laparoscopically guided placement with similar or shorter operative durations compared to traditional mini-laparotomy, without any statistically significant difference in the rate of other complications. Laparoscopy could become the technique of reference for VPS placement, especially if experienced laparoscopic surgeons are available. In patients having undergone previous abdominal surgery, it seems preferable to place the catheter at distance from scars and previous operative sites to avoid potential adhesions.

Chronic complications

Pseudo-cyst (PC)

Development of a PC is a rare complication of VPS, observed mainly in children, and for which the prevalence ranges from 0.7 to 12.5% [10,30]. Two hundred and four cases have been reported between 1954 and 2001 [3]. They occur principally during the first six months after the creation of the VPS [2], and are enhanced by the presence of factors decreasing the absorption of the CSF: intraperitoneal adhesions after

Figure 5. Pseudo-cyst. Clinical picture of intracranial hypertension and impaired conscience one year after insertion of the ventriculoperitoneal shunt with recurrence of acute hydrocephalus on cerebral CT scan. Abdominal CT scan shows a small fluid collection (black asterisk) with thickened walls corresponding to the pseudo-cyst, with infiltration of fatty tissue in right flank in contact with the distal loop of the catheter (black arrow).

abdominal surgery, or multiple revisions, increased protein count or peritoneal inflammation [3]. Clinical signs include pain and abdominal distention associated with a palpable mass. The cyst is hypoechogenic on ultrasonic examination. CT scan is the most reliable investigation: the cystic formation is homogeneous and well-delineated at the distal end of the catheter [31] (Figs. 5 and 6). Extra-peritoneal localizations have also been reported: breast (after rupture or migration of the catheter), liver, and neck [32—34]. The exact cause of PC remains unknown; the most probable explanation is the creation of an impermeable resorption surface by the inflammatory process [30]. Low profile infections can also be at the origin of their formation. In a review by Mobley et al. of 128 PC, the infection rate was 42% [35].

Aparici-Robles and Molina-Fabrega [31] reported their experience with six patients with PC after VPS. Cerebral CT was normal and intra-cystic fluid was sterile in all six cases. Five patients were treated by fenestration of the PC via laparotomy and replacement of the catheter while the sixth patient was treated by percutaneous drainage. In late follow-up, PC recurred in two patients, including the patient treated by percutaneous puncture. Gmeiner et al. reported an elevated incidence of PC in their cohort (12.5%, $n = 14$) of 112 patients [30]. Surgical strategies varied with assorted combinations of fenestration, repositioning of the catheter in the peritoneal cavity, externalization of the catheter, or changing the ventriculoperitoneal shunt. The PC occurred more often in patients for whom the VPS was neither completely nor partially replaced. These authors proposed a management algorithm for PC [30]:

- routine cultures of the CSF and the extremities of the VPS during surgical revision;
- if infection is diagnosed preoperatively, the VPS must be routinely removed, and an external drainage can be envisioned if there are no preoperative signs or intraoperative suspicion of infection, but the VPS must be entirely removed if the intraoperative cultures are positive;
- a probabilistic antibiotic therapy should then be instituted, secondarily adapted to bacterial sensitivity if the cultures are positive;
- after elimination or effective treatment of infection, the VPS (or its distal portion) should be repositioned in the peritoneal space, whereas for patients who are not infected, the upper and intraventricular portions of the VPS can be left in place, thus reducing the risk of complications; whereas
- if the PC recurs, the authors advise insertion of ventriculo-atrial shunts.

Lastly, if the PC is symptomatic, which occurs most often when the PC are voluminous, a local approach with fenestration of the wall is advised. Since the beginning of the 21st century, the laparoscopic approach has been used to drain the PC and perform adhesiolysis of any intraperitoneal bands [2,16,36]. Fig. 7 shows the laparoscopic approach of a PC, fenestration and repositioning of the catheter.

Migration and disconnection of the catheter

Migration of the intra-abdominal portion of the catheter occurs in 0.8 to 3% of cases [37]. The large mobility of the catheter and organs in the abdominal cavity favorize this complication. The migrated catheter can be found in the abdominal cavity, abdominal wall, scrotum, umbilicus, pleura, mediastinum, heart, and greater vessels [16]. Catheters have been known to migrate to umbilical or inguinal hernias, especially when the processus vaginalis canal has not been closed or when the umbilical orifice is re-permeabilized due to increased intra-abdominal pressure [38]. Migrations can also occur when the catheter disconnects, most often between the silicon and metallic portions. Treatment consists of revision, VPS removal and replacement. If the distal catheter disconnects or migrates, simple revision of the VPS is impossible, because its distal portion cannot be removed. Treatment consists of exploration of the abdominal cavity, preferably via laparoscopy and extraction of the distal portion of the catheter [2].

Ascites

CSF ascites can occur in patients with VPS. Most often infection and then peritonitis are the cause of the CSF effusion, frequently associated with an inflammatory pseudo-cyst. Rarely, the ascites fluid is sterile, as reported in some 30 patients of the literature [39]. Ascites in these cases was due to tumors (particularly choroid plexus tumors, suprasellar gliomas or VPS-disseminated metastases), peritoneal inflammation, or sometimes a massive allergic reaction against the catheter [39]. Ascites can regress once the catheter is replaced by a ventriculo-atrial shunt. This diagnosis is the last to evoke, after elimination of all other causes, malignant or infective. A diagnostic test consists of externalization of the distal portion of the catheter, which should lead to disappearance of the ascites.

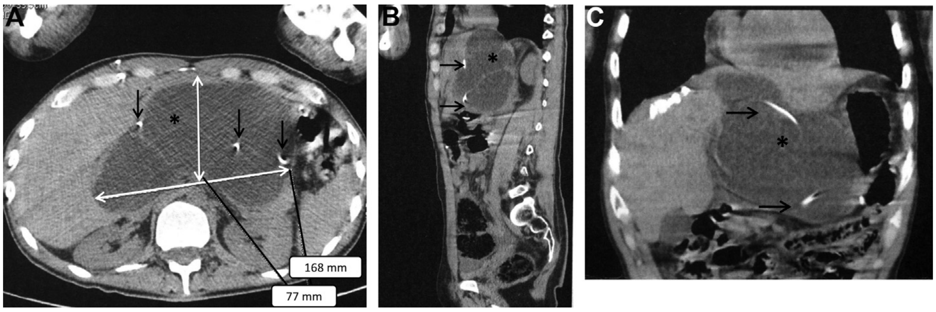

Figure 6. Voluminous epigastric pseudo-cyst (black asterisk) (17 cm diameter) developed around the catheter (black arrows), presenting as abdominal pain several years after insertion of ventriculoperitoneal shunt in childhood [axial (A), sagittal (B) and coronal (C) slices].

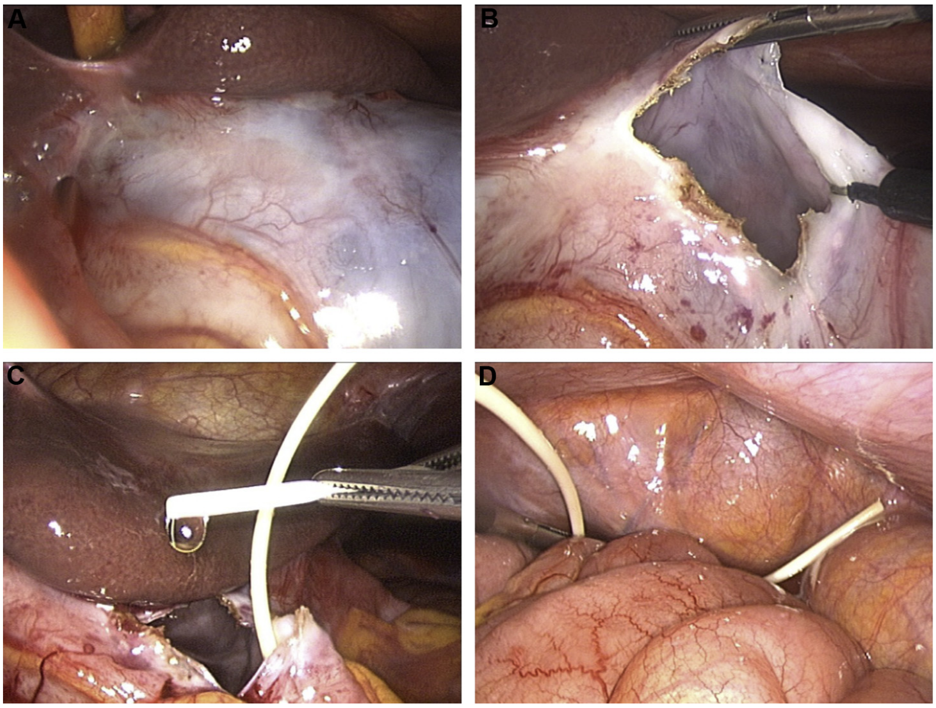

Figure 7. Laparoscopic treatment of pseudo-cyst. A. Operative view of pseudo-cyst under the left liver lobe. B. Fenestration and drainage. C. Recuperation of the distal end of the catheter. Note the flow of cerebrospinal fluid attesting to the persistent functional characteristic of the ventriculoperitoneal shunt. D. Replacement of the catheter in the Douglas pouch.

Late visceral perforation

The mechanisms of visceral perforation secondary to VPS are not well known. They most likely are due to a foreign-body reaction giving rise to peritoneal and visceral inflammation, which, combined with the pulsatile pressure of the CSF can lead to erosion through the viscus walls [40]. Its incidence is much lower since the inception of silicone catheters. It is at the level of the bowel, and more precisely, the colon, that most perforations are seen with a prevalence ranging from 0.1 to 0.7%, and a mean interval of 6.1 months between surgery and onset of perforation [41]. Once this happens, the catheter can migrate along the digestive tract due to intestinal peristalsis (Fig. 8, [42]), and eventually exit through the anus. More than 100 such cases have been reported over the last 50 years, within a mean delay of 6.1 months [43]. In the study by Vinchon et al. [23], analyzing 19 cases of intestinal perforations, three quarters of patients complained of fever, and less than half had abdominal symptoms. Signs of ICHT or meningeal symptoms can also exist, although not routinely, and the abdominal and cerebral CT scans can be normal. The most common localization described in the literature are the gallbladder, stomach, bladder, scrotum and vagina [16].

Treatment consists of ablation of the catheter: the catheter is divided just under the subcutaneous portion and extracted from below, in order not to contaminate the trajectory in a retrograde fashion, then the rest is pulled out through the retro-auricular incision. Simple catheter ablation is all that is needed in most cases to allow the intestinal perforation to heal spontaneously, through local fibrosis created at the perforation site [44]. The patient remains NPO for two days and simple surveillance is often proposed [23]. Conversely, laparotomy or laparoscopy become necessary if there are signs of diffuse peritonitis, but this occurs rarely.

Visceral surgery in patients with VPS

Not infrequently, patients with a VPS need abdominal or urological transperitoneal operations. There are no clear recommendations in the literature concerning the management of such patients.

Visceral surgeons must pay particular attention to avoid creating any obstruction or infection of the catheter, with the risk of very severe consequences. This risk is greater when the time interval between VPS insertion and surgery is short. In the study by Li et al., mean survival of the VPS was six weeks if surgery took place during the same year

Figure 8. Intestinal migration of the ventriculoperitoneal shunt: antero-posterior radiography of the pelvis (A) showing the distal end of the catheter in the pelvis. Axial, coronal and sagittal (B, C, D) slices of the CT scan showing the migration of the distal portion of the catheter into sigmoid colon and rectum (figure from Maller et al. [42]).

after insertion ($P < 0.001$), with a risk of VPS dysfunction that decreased by 29% every year afterwards (odds ratio: 0.71, 95% CI: 0.66—0.89, $P = 0.005$) [45]. Li et al. [46] analyzed the data from 39 abdominal operations in patients with a shunt, 35 of whom required an operation with entry into the intestinal or urological tract. Only two patients had to undergo VP externalization for infection. The risk can therefore be considered to be low for such patients who require an operation on their intestinal or urological tract, whether the operation is elective or in a contaminated field, as underscored by Pittman et al. in 1992 [47].

With regard to laparoscopy, abdominal CO_2 insufflation increases the intracranial pressure. One study of a series of 10 patients undergoing insertion of a VPS via laparoscopy showed that an increase of 15 mmHg intra-abdominal pressure was accompanied by an increase of intracranial pressure up to 32 cm H_2O (for a normal pressure less than 20 cm H_2O) [48]. However, an experimental study by Neale and Falk [49] showed that the VPS check-valve system did not fail when intraperitoneal pressure increased. Moreover, several studies have reported the use of laparoscopy in patients with a shunt [2,16,19,36]. There is therefore no absolute contraindication to laparoscopy in this setting, but postoperative surveillance is necessary because some rare cases of VPS dysfunction after laparoscopy have been reported [50].

With regard to perioperative antibiotic prophylaxis during abdominal operations in patients with VPS, there are no well-established protocols. Li et al. proposed administration of preoperative cephalosporin's, continued for 24 h postoperatively, if the digestive or urological tracts are not opened. If they are, antibiotic prophylaxis should cover the intestinal tract germs and comprise either cefepime or metronidazole preoperatively continued during three days postoperatively if there is no concomitant digestive tract infection [46].

For abdominal diseases that do not require surgical management such as diverticulitis, ileitis or certain cases of cholecystitis, there are no formal management plans for patients with a VPS. According to our experience, treatment should not differ from that for patients without any shunt with regard to the type or duration of antibiotics, but patient surveillance should be increased during the initial routine postoperative hospital stay. One must unrelentingly search for signs of infection or VPS dysfunction, and foresee a close neurological follow-up during the following months with clinical and radiological monitoring.

Conclusion

There are several scenarios in which the visceral surgeon can become involved in management of patients with a VPS. VPS insertion is performed more and more often via laparoscopy, which can decrease the rate of several postoperative complications without undue lengthening of operation [9—11]. Several reports of laparoscopic management of abdominal complications related to VPS have been published in the literature [2,18,28,30]. It is therefore important for all visceral surgeons to know the principles and potential complications of VPS and for neurosurgeons to familiarize themselves with laparoscopy. The combined competences of the two specialties are often necessary during the course of events for patients with VPS.

Essential points

- Insertion of the peritoneal catheter for VPS is performed laparoscopically more and more often, thus avoiding blind introduction of the catheter into the peritoneal cavity, limiting the risk of gastrointestinal perforation, while reducing operative times and decreasing the rate of distal dysfunctions of VPS.
    - In case of catheter dysfunction:
        - Signs of meningitis associated with peritonitis can be present.
        - CSF samples are essential and must be drawn before starting any antibiotic regimen via lumbar tap, reservoir tap or during any insertion of external VPS.
        - The VPS has to be changed.
        - Peritoneal lavage via laparoscopy or laparotomy is necessary only if intra-abdominal disease is suspected.
    - Intraperitoneal pseudo-cysts can manifest by abdominal signs or neurological signs of hydrocephalus. Therapy consists of:
        - Replacement of the distal portion of the catheter, the key element of treatment.
        - Elimination of infection is primordial, and its presence should lead to immediate removal of the entire VPS.
        - Or laparoscopic fenestration if the patient is symptomatic.
    - Visceral perforations can occur months or years after the initial operation and principally involve the colon. The catheter can then migrate downstream and protrude through the anus.
    - Treatment of visceral perforations:
        - Routine retrograde ablation of the catheter after upstream division.
        - There may be no need to perform any diagnostic or therapeutic abdominal exploration since the perforation can heal spontaneously.
    - VPS dysfunctions following laparotomy or laparoscopy for a gastrointestinal problem are rare, and there is no formal contraindication to operating on patients with VPS. The check-valve system should avoid any increase in intracranial pressure during the creation of the CO_2 pneumoperitoneum.

Disclosure of interest

The authors declare that they have no competing interest.
Disclosure of interest

References

[1] Gutiérrez-González R, Boto GR, Pérez-Zamarrón Á. Cerebrospinal fluid diversion devices and infection. A comprehensive review. Eur J Clin Microbiol Infect Dis 2012;31:889—97, http://dx.doi.org/10.1007/s10096-011-1420-x.

[2] Ghidirim G, Mishin I, Zastavnitsky G, Spataru V, Brinza M. Laparoscopic management of associated abdominal complications of ventriculoperitoneal shunt: case report. Eur Surg 2010;42:184—6, http://dx.doi.org/10.1007/s10353-010-0543-0.

[3] Anderson CM, Sorrells DL, Kerby JD. Intra-abdominal pseudocysts as a complication of ventriculoperitoneal shunts. J Am Coll Surg 2003;196:297—300, http://dx.doi.org/10.1016/S1072-7515(02)01751-9.

[4] Hanak BW, Bonow RH, Harris CA, Browd SR. Cerebrospinal fluid shunting complications in children. Pediatr Neurosurg 2017;52:381—400, http://dx.doi.org/10.1159/000452840.

[5] Bridges N, Rifkinson-Mann S, Handler M, Epstein F. Planning ventriculoperitoneal shunts in infants and small children. Pediatr Neurosci 1988;14:191—5.

[6] Alyeldien A, Jung S, Lienert M, Scholz M, Petridis AK. Laparoscopic insertion of the peritoneal catheter in ventriculoperitoneal shunting. Review of 405 consecutive cases. Int J Surg 2016;33:72—7, http://dx.doi.org/10.1016/j.ijsu.2016.07.052.

[7] Armbruster C, Blauensteiner J, Ammerer HP, Kriwanek S. Laparoscopically assisted implantation of ventriculoperitoneal shunts. J Laparoendosc Surg 1993;3:191—2.

[8] Dehcordi SR, Tommasi CD, Ricci A, et al. Laparoscopy-assisted ventriculoperitoneal shunt surgery: personal experience and review of the literature. Neurosurg Rev 2011;34:363—71, http://dx.doi.org/10.1007/s10143-011-0309-6.

[9] Schucht P, Banz V, Trochsler M, et al. Laparoscopically assisted ventriculoperitoneal shunt placement: a prospective randomized controlled trial. J Neurosurg 2015;122:1058—67, http://dx.doi.org/10.3171/2014.9.JNS132791.

[10] Santos de Oliveira R, Barbosa A, Vicente YAdeMVde, Rubens Machado H. An alternative approach for management of abdominal cerebrospinal fluid pseudo-cysts in children. Childs Nerv Syst 2006;23:85—90, http://dx.doi.org/10.1007/s00381-006-0183-7.

[11] Yamashiro S, Hitoshi Y, Yoshida A, Kuratsu J. Historical considerations of the predominance of lumbo-peritoneal shunt for treatment of adult communicating hydrocephalus in Kumamoto, Japan. Clin Neurol Neurosurg 2012;114:1115—6, http://dx.doi.org/10.1016/j.clineuro.2012.02.018.

[12] Demetriades AK, Haq IZ, Jarosz J, McCormick D, Bassi S. The ventriculocholecystic shunt: two case reports and a review of the literature. Br J Neurosurg 2013;27:505—8, http://dx.doi.org/10.3109/02688697.2013.771135.

[13] West CG. Ventriculovesical shunt. Technical note. J Neurosurg 1980;53:858—60, http://dx.doi.org/10.3171/jns.1980.53.6.0858.

[14] El Shafei IL, El Shafei HI. The retrograde ventriculo-sinus shunt (El Shafei RVS shunt). Rationale, evolution, surgical technique and long-term results. Pediatr Neurosurg 2005;41:305—17, http://dx.doi.org/10.1159/000088733.

[15] Cappabianca P, Cinalli G, Gangemi M, et al. Application of neuroendoscopy to intraventricular lesions. Neurosurgery 2008;62(Suppl. 2):575—98, http://dx.doi.org/10.1227/01.neu.0000316262.74843.dd.

[16] Popa F, Grigorean V, Onose G, Popescu M, Strambu V, Sandu A. Laparoscopic treatment of abdominal complications following ventriculoperitoneal shunt. J Med Life 2009;2:426—36.
[17] Coley BD, Kosnik EJ. Abdominal complications of ventriculoperitoneal shunts in children. Semin Ultrasound CT MRI 2006;27:152—60, http://dx.doi.org/10.1053/j.sult.2006.01.009.
[18] Burhan B, Serdar KB, Abdurrahman A, Edip AM, Ebuzer D. Abdominal complications of ventriculoperitoneal shunt in pediatric patients: experiences of a pediatric surgery clinic. World Neurosurg 2018;118:e129—36, http://dx.doi.org/10.1016/j.wneu.2018.06.140.
[19] Nfonsam V, Chand B, Rosenblatt S, Turner R, Luciano M. Laparoscopic management of distal ventriculoperitoneal shunt complications. Surg Endosc 2008;22:1866—70, http://dx.doi.org/10.1007/s00464-007-9728-4.
[20] Simon TD, Kronman MP, Whitlock KB, et al. Variability in management of first cerebrospinal fluid shunt infection: a prospective multi-institutional observational cohort study. J Pediatr 2016;179, http://dx.doi.org/10.1016/j.jpeds.2016.08.094 [185—191.e2].
[21] Kanangi SMR, Balasubramaniam C. Shunt infections: a review and analysis of a personal series. Childs Nerv Syst 2018;34:1915—24, http://dx.doi.org/10.1007/s00381-018-3890-y.
[22] Pelegrín I, Lora-Tamayo J, Gómez-Junyent J, et al. Management of ventriculoperitoneal shunt infections in adults: analysis of risk factors associated with treatment failure. Clin Infect Dis 2017;64:989—97, http://dx.doi.org/10.1093/cid/cix005.
[23] Vinchon M, Baroncini M, Laurent T, Patrick D. Bowel perforation caused by peritoneal shunt catheters: diagnosis and treatment. Oper Neurosurg 2006;58, http://dx.doi.org/10.1227/01.NEU.0000192683.26584.34 [ONS-76—ONS-82].
[24] Wells DL, Allen JM. Ventriculoperitoneal shunt infections in adult patients. AACN Adv Crit Care 2013;24:6—12.
[25] Gutierrez FA, Raimondi AJ. Peritoneal cysts: a complication of ventriculoperitoneal shunts. Surgery 1976;79:188—92.
[26] Wong GKC, Wong SM, Poon WS. Ventriculoperitoneal shunt infection: intravenous antibiotics, shunt removal and more aggressive treatment? ANZ J Surg 2011;81:307, http://dx.doi.org/10.1111/j.1445-2197.2011.05690.x.
[27] Riccardello GJ, Barr LK, Bassani L. Bowel perforation presenting with acute abdominal pain and subcutaneous emphysema in a 14-year-old girl with an abandoned distal peritoneal shunt catheter: case report. J Neurosurg Pediatr 2016;18:325—8, http://dx.doi.org/10.3171/2016.3.PEDS15572.
[28] Phan S, Liao J, Jia F, et al. Laparotomy vs. minimally invasive laparoscopic ventriculoperitoneal shunt placement for hydrocephalus: a systematic review and meta-analysis. Clin Neurol Neurosurg 2016;140:26—32, http://dx.doi.org/10.1016/j.clineuro.2015.10.025.
[29] Naftel RP, Argo JL, Shannon CN, et al. Laparoscopic versus open insertion of the peritoneal catheter in ventriculoperitoneal shunt placement: review of 810 consecutive cases. J Neurosurg 2011;115:151—8.
[30] Gmeiner M, Wagner H, van Ouwerkerk WJR, Senker W, Holl K, Gruber A. Abdominal pseudo-cysts and peritoneal catheter revisions: surgical long-term results in pediatric hydrocephalus. World Neurosurg 2018;111:e912—20, http://dx.doi.org/10.1016/j.wneu.2018.01.032.
[31] Aparici-Robles F, Molina-Fabrega R. Abdominal cerebrospinal fluid pseudo-cyst: a complication of ventriculoperitoneal shunts in adults. J Med Imaging Radiat Oncol 2008;52:40—3, http://dx.doi.org/10.1111/j.1440-1673.2007.01909.x.
[32] Cho KH, Yoon SH, Kim SH, Shin YS, Ahn YH, Cho KG. Neck mass after catheterization of a neck vein in a child with ventriculoperitoneal shunt. Pediatr Neurosurg 2004;40:182—5, http://dx.doi.org/10.1159/000081936.
[33] Spector JA, Culliford AT, Post NH, Weiner H, Levine JP. An unusual case of cerebrospinal fluid pseudo-cyst in a previously augmented breast. Ann Plast Surg 2005;54:85—7.
[34] Dabdoub CB, Fontoura EA, Santos EA, Romero PC, Diniz CA. Hepatic cerebrospinal fluid pseudo-cyst: a rare complication of ventriculoperitoneal shunt. Surg Neurol Int 2013;4:162, http://dx.doi.org/10.4103/2152-7806.123783 [Published online 2013 Dec 27].
[35] Mobley LW, Doran SE, Hellbusch LC. Abdominal pseudo-cyst: predisposing factors and treatment algorithm. Pediatr Neurosurg 2005;41:77—83, http://dx.doi.org/10.1159/000085160.
[36] Jain S, Bhandarkar D, Shah R, Vengsarkar U. Laparoscopic management of complicated ventriculoperitoneal shunts. Neurol India 2003;51:269.
[37] Kaplan M, Ozel SK, Dönmez O, Kazez A. Treatment approaches for abdominal migration of peritoneal catheter of ventriculoperitoneal shunt. Turk Neurosurg 2007;17:158—62.
[38] Singh S, Pant N, Kumar P, et al. Migration of ventriculoperitoneal shunt into a hernia sac: an unusual complication of ventriculoperitoneal shunt surgery in children. Pediatr Neurosurg 2016;51:154—7, http://dx.doi.org/10.1159/000442179.
[39] Kariyattil R, Steinbok P, Singhal A, Cochrane DD. Ascites and abdominal pseudo-cysts following ventriculoperitoneal shunt surgery: variations of the same theme. J Neurosurg Pediatr 2007;106:350—3.
[40] Thiong'o GM, Luzzio C, Albright AL. Ventriculoperitoneal shunt perforations of the gastrointestinal tract. J Neurosurg Pediatr 2015;16:36—41, http://dx.doi.org/10.3171/2014.11.PEDS14347.
[41] Bodeliwala S, Agrawal A, Mittal A, Singh D, Vageesh B, Singh H. Transanal protrusion of ventriculoperitoneal shunt via appendicular perforation: a rare case report. J Pediatr Neurosci 2016;11:274, http://dx.doi.org/10.4103/1817-1745.193383.
[42] Maller VV, Agarwal A, Kanekar S. Imaging of ventricular shunts. Semin Ultrasound CT MRI 2016;37:159—73, http://dx.doi.org/10.1053/j.sult.2016.01.003.
[43] Farazi MMA, Salam MA, Azim MAU. Transanal extrusion of the ventriculoperitoneal shunt tube. Med Today 2016;27:44—5.
[44] Li H-N, Tan T-C, Cheung F-C. Transanal protrusion of ventriculoperitoneal shunt. Surg Pract 2008;12:93—6, http://dx.doi.org/10.1111/j.1744-1633.2008.00407.x.
[45] Li Ching A, Humphrey G, Kamaly-Asl ID. Ventriculoperitoneal shunt survival in children who require additional abdominal surgery—are our estimations of the additional risk accurate? Br J Neurosurg 2014;28:40—8, http://dx.doi.org/10.3109/02688697.2013.815322.
[46] Li G, Dutta S. Perioperative management of ventriculoperitoneal shunts during abdominal surgery. Surg Neurol 2008;70:492—5, http://dx.doi.org/10.1016/j.surneu.2007.08.050.
[47] Pittman T, Williams D, Weber TR, Steinhardt G, Tracy T. The risk of abdominal operations in children with ventriculoperitoneal shunts. J Pediatr Surg 1992;27:1051—3.
[48] Kamine TH, Elmadhun NY, Kasper EM, Papavassiliou E, Schneider BE. Abdominal insufflation for laparoscopy increases intracranial and intrathoracic pressure in human subjects. Surg Endosc 2016;30:4029—32, http://dx.doi.org/10.1007/s00464-015-4715-7.
[49] Neale ML, Falk GL. In vitro assessment of back pressure on ventriculoperitoneal shunt valves. Surg Endosc 1999;13:512—5, http://dx.doi.org/10.1007/s004649901024.
[50] Miele VJ, Bendok B, Bloomfield SM, Ondra SL, Bailes JE. Ventriculoperitoneal shunt dysfunction in adults secondary to conditions causing a transient increase in intra-abdominal pressure: report of three cases. Neurosurgery 2004;55:434.