8 Mechanical Complications of Shunts

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Contents

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8.1 Fracture

8.1.1 Epidemiology

Fractures or ruptures of the shunt catheters are the causative events of 4.5–13.6 % of shunt revision surgeries [46, 63]. Besides the contribution to patients' morbidity, there is an increased impact to health care costs, considering the large numbers of patients with CSF shunts and the cumulative long time that these shunts are implanted for. An accurate conclusion regarding the exact impact of mechanical complications to shunt malfunction is difficult to draw because these problems usually present a few years after the implantation and many patients are lost to follow-up. In addition, in a group of patients with such fractures or disconnections the clinical impact is minimal or unnoticed because either the patient has become shunt-independent or the CSF diversion is still patent through a fibrous reactive sleeve over the damaged catheter [75]. Furthermore, although a great amount of research focuses on shunt infection issues, mechanical dysfunction has not gained proportionate attention in the literature over the years.

A large series of 1,719 patients shunted for hydrocephalus and followed for 10 years showed that the overall risk for shunt malfunction at the end of the study was 70 % [63]. The most frequent cause was obstruction (56 %), followed by fracture, disconnection, and late migration which accounted for 19 % of mechanical complications

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occurring in 13 % of the children. It was similarly noticed that such complications occurred mostly in children, treated in their early age for hydrocephalus and these catheters were placed for longer periods (>5 years) [9].

8.1.2 The Implanted Material

Materials have a wide spectrum of properties that are used to describe and quantify their features. These properties can be thermal, electrical, chemical, mechanical, etc. For materials that are used in medicine and more specifically for implantable human purposes, all these properties are important and this section will focus on the mechanical characteristics of silicone rubbers and their correlation with the host's reaction (humans) in their presence.

Polysiloxanes are mixed inorganic and organic polymers mostly known as *silicon rubber* [49]. These are based on a silicon and oxygen skeleton, instead of an organic carbon chain skeleton. Silicon as an element was first discovered in 1824 by J.J. Berzelius, but it was in the 1940s that F.S. Kipping (the father of silicon science) achieved extensive synthesis of silicone compounds and allowed the industrial production of the new material. The high resistance to heat made it superior for medical use because its physical properties were not affected by thermal sterilization. Its high flexibility and biocompatibility made silicon well suited for in vivo implantation use [23]. In the early 1950s, the silicon rubber marketed for medical use was Silastic® (Dow Corning, Midland, MI). In neurosurgery, it was introduced as the tubing material of the newly presented CSF diversion systems for the treatment of hydrocephalus, at the end of the same decade. Since then, silicon rubber has been the material that was used almost exclusively by the manufacturers of CSF shunt systems.

Calcification

It is the first step of the process that leads to: (1) degradation and weakness of the catheter and (2) cell aggregation and tethering of the tube to the surrounding tissue. The phenomenon was well studied after the first malfunctions of several silicon-made implants appeared (cardiac prostheses, breast implants) [17, 32, 34]. When the local concentration of an insoluble compound into a solution exceeds the limit of solubility, then *precipitation* of the compound from the solution occurs [9, 65, 67]. The following stages are the formation of a small *nucleus* of precipitate and then the growth of it by continuous precipitation (Fig. 8.1). This nucleation process is enhanced by the presence of solid interfaces and, especially for the precipitation of *calcium phosphate,* is accelerated in an alkaline environment [33]. Two mechanisms of calcification are known [65, 84]: *Metastatic calcification* is associated with increased calcium and phosphorus blood concentrations and is described in renal insufficiency; and *dystrophic calcification* has normal calcium and phosphorus blood concentrations but altered cellular metabolism and is the one encountered in silicone implants. An accelerated formation of nuclei from the cellular debris that accumulate in proximity to the implant has been demonstrated [9, 32]. Further formation of initial nuclei results from cracks and surface irregularities of the material which allow element extrusion and interaction with the surrounding cells.

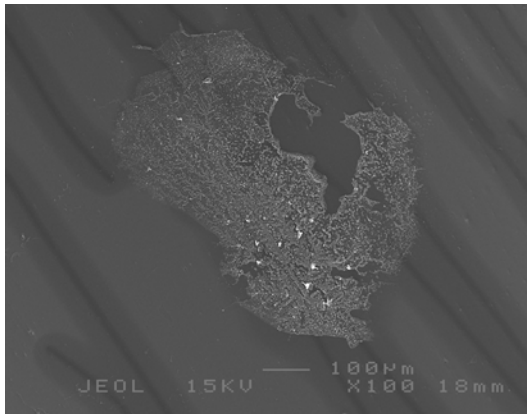

Fig. 8.1 Scanning Electron Microscopy of a shunt valve of diaphragm type, removed from a patient due to shunt malfunction. There is precipitation of crystals on the diaphragm, which with time deteriorates and interferes with the physical properties of the membrane and can block the gap between diaphragm and case (magnification ×100)

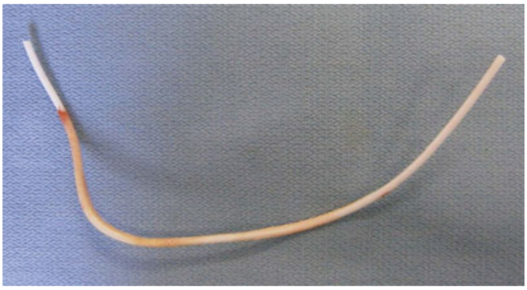

Fig. 8.2 Distal catheter removed during shunt revision. There is obvious calcification on its outer surface at the area corresponding to the neck region, where the shunt was implanted

Immune Response

Investigation of previously implanted shunts revealed eosinophils and giant cells as parts of the fibrous sleeve, indicating that a hypersensitivity reaction occurs [74]. Most of these studies applied plain radiographs, scanning electron microscopy (SEM), spectral analysis, and simple histology. The cell-mediated immunity was supported from the presence of T-cell granulomas. Others have found immunoglobulin G antibodies specifically directed against silicon, as a part of humoral immunity involvement [30]. On the other hand, some investigators only found dense hyalinized connective tissue with fibroblasts and calcific deposits [85]. Heggers et al. supported a chronic inflammatory reaction – predominantly a foreign body giant cell granuloma type – that was initiated from the release of silicon particles from the aging material [34, 38]. Some authors support that different kind of particles are responsible for the foreign body reaction such as cotton fibers, talc granules, and hair that inadvertently enter the host during surgery [66]. The most profound tissue reaction over the ventriculoperitoneal (VP) shunt tubing was noticed at the subcutaneous course of the catheter and especially at the neck area [9, 75] (Fig. 8.2). Calcification was present only in catheters placed in subcutaneous or vascular areas [46]. It is supported that the cerebral parenchyma and the peritoneal cavity are far less reactive for the silicon catheters [42]. This suits well with the "cellular reaction" theory since the subcutaneous tissue and the intravascular space are areas where migration of immunocompetent cells can occur but it does not explain the lack of calcification in the peritoneal cavity. Furthermore, some case reports of distal catheter insertion into abdominal hollow viscera suggest that a key point for that is the anchoring of the catheter to the organ serosa, which appears to be initiated by a local inflammatory reaction around the distal tip that facilitates erosion and perforation [41, 53, 64]. The clear mechanism is not yet fully understood. Regarding the most proximal compartment of the host involved, there is no cellular immunity in the cerebral parenchyma (similar to the one described for the rest of the body) and ventricular catheters do not induce a gliotic reaction inside the brain tissue as noticed by the absence of adherent cells on the silicone [9, 19].

Degradation and Altered Physical Properties

The interaction of the silicone catheter with the surrounding tissue leads to the calcification of tube. The implant becomes rigid and fragile. Besides the calcified covering sheet, the degradation process evolves. It has been shown that the dynamic properties of the silicon rubber begin to deteriorate about 6 months after the implantation; the ultimate tensile strength and extensibility gradually decreases over the first 3 years and becomes remarkably altered after 5 years [23, 76]. Different studies have found reticulation damage as an important factor [9, 23]. The four ways that biopolymers deteriorate were described by Kronenthal [43]: (a) the structure is altered by hydration; (b) some covalent bonds of the chain are weakened; (c) these bonds are broken; and (c) soluble fragments are digested by the macrophages. In the human body, the silicones are not subjected to extreme temperatures or irradiation, which are known to contribute to degradation, but the subsequent chemical and mechanical insults (especially in prolonged time periods) can have the same result. This is more evident in prosthetic heart valves that are exposed to strong biological activity (blood cells) and intense mechanical stress (heart beats and blood flow) [65, 78]. In VP (and ventriculoatrial – VA) shunts, the more mobile segment is in the neck [23, 46]. Repeatable head movements – especially extension and contralateral rotation – increase the tension of the mobile neck segment over the fixed parts. The three more vulnerable zones were found to be: (a) the connection between the distal catheter and the reservoir or the valve, (b) the point of traversing the galea, and (c) the crossing of the clavicle [24]. Shunts that are inserted in young children and remain for long time periods are exposed to additional mechanical stress due to the increasing height of the child. The vulnerable part is this case is from the occiput to the peritoneal insertion [24] (Fig. 8.3). There are reports that even a minimal-to-moderate external tension over the tethered and weakened catheter led to fracture as it was the case in two boys where the shunt was fractured over the occipital area after a haircut with clippers [45]. In addition, children have a greater tendency for shunt calcification from a physiological point of view and this maybe related to the increased serum phosphorus levels, compared to adults [9, 18]. A reduction in catheter tubing tensile strength and extensibility was demonstrated when the apparatus had been implanted for more than 6 years [23]. These changes were related to mineralization of the catheter, which resulted in a 30–40 % reduction in tubing thickness. Similar findings were reported from Tomes et al. who showed that catheters become weaker the longer they are implanted and that tubing with a greater cross-sectional area requires greater force to fracture [79].

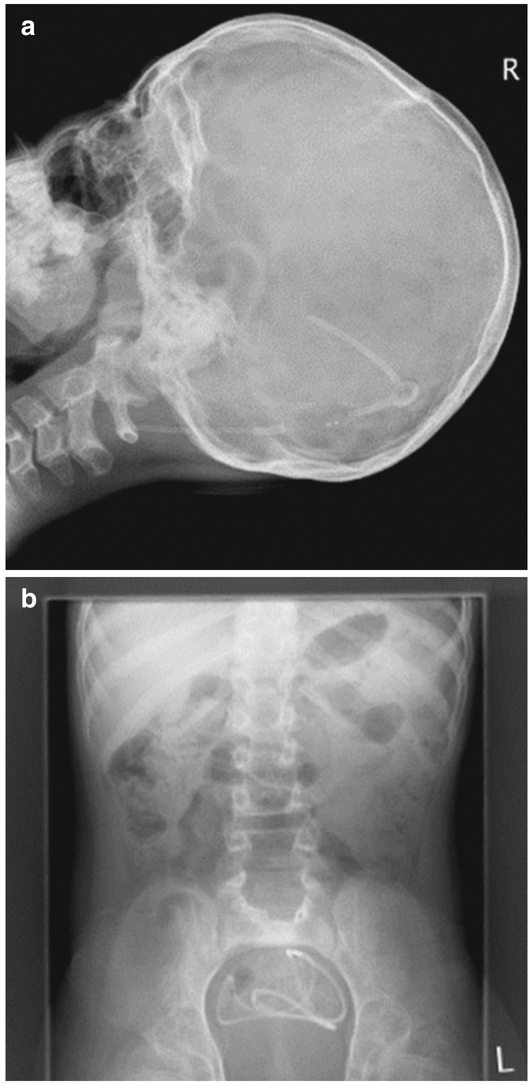

Fig. 8.3 Distal catheter fracture in the region of the neck. Plain radiographs of a 10-year-old boy, who had a shunt inserted when he was 6 months old, and he presented on this occasion with symptoms of raised intracranial pressure. (**a**) Lateral skull radiograph. The distal shunt tube is abruptly discontinued in the neck region, a few centimeters below the valve. (**b**) Abdominal plain radiograph. The remaining distal tube has "fallen" in the peritoneal cavity and coiled up at the region of Douglas pouch

8.1.3 Material Selection and Importance of the Hardware

As previously mentioned, there are reports of allergy to the implanted silicone catheters [35, 36]. In these cases, the presentation was similar to shunt infection or obstruction, but the importance is that the authors used as an alternative catheters that were made from *polyurethane*. Until recently, such choice was not available. Polyurethane is a polymer composed of a chain of organic units joined by carbamate (urethane) links. It shares many properties with silicone such as heat resistance, which allows it to be thermally sterilized, high biocompatibility, low toxicity, and high tensile strength. On the other hand, it is more rigid, making such systems less easy to manufacture than silicone-based ones [35]. Polyurethane breast implants tended to produce thicker and longer-lasting surrounding capsules compared to the silicon-based ones [70].

Fig. 8.4 Shunt valve of Holter type, removed during shunt revision, 20 years after implantation. The valve case has completely disintegrated due to material distruction. Extensive calcification is seen on the outer surface

On the field of CSF diversion shunt systems, the two materials have not yet been properly compared on a clinical basis.

In order to be radiographically visualized after implantation, shunt catheters must be either homogeneously *impregnated with barium* or only have a stripe of it. It has been demonstrated that barium precipitates locally as barium salts, enhancing the local immune response and increasing the focal tethering effect and subsequently the risk for catheter fracture [9]. Furthermore, catheters with barium that were tested for their tensile strength without being previously implanted, showed significantly lower values in terms of both ultimate strain and ultimate stress [79]. Similar reports were not found regarding *antibiotic-impregnated* shunt catheters. It is not mentioned if their physical properties are altered in vitro due to the presence of the antibiotics. Aryan et al. did not find significant local reaction in such externalized catheters [6]. It is unknown if their long-term fracture rates are different from the conventional type of catheters.

The valve type may influence the mechanical features and durability of the shunt system. The valve *profile* has a major impact [9]. A tubular valve in the catheter line may have a tendency to migrate especially in a growing child as it is pulled by a fixed calcified catheter. Round-bodied valves are more resistant to migration distally. If such valves are placed in the hypochondriac region (to prevent cranial MRI artifacts and skin erosion issues), there is a tendency to fracture at the side of the outlet connector as this is the point of maximal stress force due to the continuous abdominal movements over the fixed valve [2]. Shunt failure caused by valve collapse has been reported in the past [50] (Fig. 8.4) and regarded 11 patients with the Mini-Holter valve where intussusception of the valve ends into the valve tubing was noticed, possibly due to the siphoning effect. The same type of valve was mechanically disrupted in a case report in which the proximal metallic portion was "dislocated" out of its silicone sheath, possibly due to the growth-related traction applied to the fixed valve [86]. In a recent retrospective study, the fixed pressure–gravity-assisted valve paediGAV (Aesculap AG & Co. KG, Tuttligen, Germany) and the programmable differential pressure Codman Hakim (Codman &Shurtlef, Inc., Raynham, USA) were compared and there was no significant difference regarding shunt failure of mechanical etiology [7].

A critical point in mechanical complications of shunts is *the number of shunt components*. The more the pieces connected to each other, the higher the failure rates [3]. A typical shunt includes one proximal catheter, connected to a reservoir or a burr-hole-type valve and a distal catheter connected to the valve. This means two components with one connection at best, but frequently three components with two connections. In a review of 275 shunt revisions, disconnection of the system accounted for 15 % of the malfunctions [3]. It was also found that the more distal the connection was from the ventricle, the higher the likelihood of disconnection. One-piece shunt systems are described to decrease early fractures and disconnections by avoiding distal connectors [31, 59].

8.1.4 Surgical Technique

Although significant amount of data have highlighted the contribution of the surgical technique in the prevention of shunt infection and obstruction, too little evidence exists regarding the correlation of mechanical complications and surgical choices or maneuvers [22]. When shunts other than one piece are utilized, an important determinant is the competence of the connection between two parts. Most times this requires reinforcement with surgical knots. The knots have to be strong enough to secure the connection but not too tight to prevent rupture of the catheter or the suture. The choice of the suture material may have an impact. Multifilament nonabsorbable sutures are mostly preferred (Silk). They are better manipulated with minimal memory effect. Monofilament sutures are regarded safer for the prevention of infection (bacteria are attached less easily compared to multifilament material) but may tear the silicon tube. For the same reason, the size of the suture is also important. Sizes 2-0 or 3-0 are preferred. The knot has to be efficient but not too big and preferably should face downward so that it is hidden in cases of thin-skinned patients [37, 72].

Several studies and reviews highlight the significance of handling the shunt components only with instruments, avoiding any contact with gloved hands, to reduce the infection rates [10, 11, 71, 81]. If these instruments are sharp and improperly used, they may tear the shunt catheters leading to shunt malfunction if the damage is not noticed. Similarly, when passing the catheter through the tunneler, an acute angle of insertion should be avoided in order to prevent abrasions to the tube caused by the sharp end of the tunneler. Lubrication of the catheter with sterile saline is a useful maneuver. When performing a shunt revision (i.e., for proximal obstruction) many surgeons use a scalpel blade to cut the knot and disconnect the shunt components. This is hazardous for the catheter's integrity and should be performed with great caution if there is no option for insertion of new hardware. The surgeon must target the knot – not the thread – and the direction of the movement should be away from the catheter. After that, the tube is inspected for unnoticed tears. If such exist, a small piece of the catheter – containing the tear – is cut away and the rest is reconnected as usual.

The choice of the burr-hole site may influence the mechanical complications rate. As previously explained, a shunt with a frontal burr hole usually needs an additional connection and this adds to the failure likelihood. On the other hand, Aldrich et al. found that occipitally placed shunts have a significantly higher rate of dislocation than frontally placed shunts [3]. Langmoen et al. studied the differences between ventriculoperitoneal and ventriculoatrial shunts with respect to the problem of mechanical failures and they found that the fracture rates of the distal catheter were almost the same (2.9–3 %) [46].

8.2 Dislocation

Dislocation of a shunt component refers to the migration of a previously correctly placed catheter or valve. The component could be disconnected from the shunt system or not. There is a lack of uniformly applied definitions regarding mechanical complications of shunts. In addition, there is paucity in the recent literature about this specific type of shunt malfunction. Most authors focus on the rheological properties of these systems.

The prevalence of pediatric shunt malfunction was reported to be 39 % at the first year and 53 % at 2 years [21]. The rate of shunt discontinuity (fracture or disconnection) is up to 10 % and refers to either ventriculoperitoneal or subduroperitoneal shunts [48]. Migrations of proximal or distal catheters have been reported on case reports and accurate frequencies extracted from efficient series or reviews are missing.

8.2.1 Proximal Catheter

The dislocated proximal catheter could enter deeper into the cranial cavity or retract distally from the previously desired location. Most systems include an angled port where the distal part of the ventricular catheter is secured in a 90° position before the connection to the distal part. Others prefer to use burr-hole reservoirs or burr-hole-type valves which are connected to the ventricular catheter. The connection should be secured with a suture properly knotted. This connection is not always straightforward, especially in cases of slitlike ventricles where even minimal manipulation of the proximal catheter can lead to improper placement. A loose connection of the ventricular catheter may result in spontaneous proximal migration. This can also happen iatrogenically in future shunt revisions when one tries to lift the burr-hole reservoir or valve and misses the disconnected catheter. Besides the surgical aspect, recently the material of the catheter was found to be responsible for the disconnection and intracranial migration [15]. The Bioglide® catheter (Medtronic, Minneapolis, US-MN) designed to potentially decrease cell adhesion and thereby reduce shunt obstruction or infection by becoming hydrophilic and lubricious when hydrated, was prone to disconnection because of this slippery surface. It was eventually recalled by Medtronic in 2009.

8.2.2 Distal Catheter

The most case reports in the literature concerning catheter migration are about the distal ventriculoperitoneal components. The distal catheter in the majority of VP shunts starts from the distal end of the valve – usually located occipitally or frontally – and ends into the peritoneal cavity in a variety of lengths, related to the manufacturer's guidelines and the surgeon's preferences. The length varies from 15 to 120 cm. Some surgeons prefer to cut short the catheter especially in newborns. The long tube and the intestinal peristaltic movements were considered responsible in rare cases of spontaneous knot formation [14, 41]. On the other hand, short catheters may add to overdrainage as explained by the Poisseuille law. A intra-abdominal length of 30–40 cm is sufficient.

The distal catheter can migrate either distally from its previous position or proximally in a retrograde direction. As already mentioned, it can be disconnected from the shunt system or not. Usually it remains coiled into the peritoneal cavity (if disconnected) or a herniated loop protrudes through the peritoneal incision and part of it is fixed subcutaneously. There are case reports of distal catheter dislocation in almost every compartment of the human body. In the peritoneal cavity, the catheter can entry a hollow [40] or (much more rarely) a solid viscus [80]. The large intestine is the more probable entry site [28] and from there it protrudes through the anus. In cases of stomach perforation, transoral protrusions have been reported [8]. Additional reports include urinary bladder perforation and transurethreal protrusion [13], dislocation through the vagina [55], into the gallbladder [58], the scrotum [60], and extrusion through the abdominal wall [82], the umbilicus [1] and the lumbar region [39].

Fig. 8.5 Extrusion of distal shunt catheter through the anus, on a 28-month-old girl. The shunt was inserted in the peritoneal cavity 12 months before, through open mini-laparotomy (not with trocar)

Several theories have been presented in order to explain the "transperitoneal" dislocations of the distal catheters. One is the iatrogenic perforation of the intestine during the insertion of the shunt. Some surgeons use the trocar technique, which may predispose to this complication in inexperienced hands [29]. Studies that compared mini-laparotomy and laparoscopic-assisted insertion techniques, failed to show an advantage of one against the other [44]. In obese patients and in multiple peritoneal reoperations, the laparoscopic-assisted technique should be strongly considered [29]. In general, anal protrusion of the catheter can have an early (a few weeks) or delayed presentation (from months to years) (Fig. 8.5). The "iatrogenic" theory applies mostly in the early presentation cases. For delayed presentations different explanations are given. Spring-coiled catheters had a higher tendency to relate with bowel perforation. The plain silicone catheters can initiate an allergic reaction and become adherent to the intestinal serosa with subsequent erosion into the lumen [12]. Local infective adhesions were also mentioned [53]. Some authors recommend the hydration of the silicon tube prior to insertion based on the tendency of the silicon rubber to become sticky when it remains in a dry state [41]. Another theory focuses on children with myelomeningoceles (MMC), which are reported to be more susceptible to bowel perforation due to their weaker intestinal musculature [25]. Glatstein et al. in 2011 reviewed the reported pediatric cases with catheter protrusions through the anus and showed 23 patients with ages ranging from 0.9 to 72 months (6 years) [29]. Sathyanarayana et al. in 2000 reviewed 45 adult patients and reported a bowel perforation rate of less than 0.1 % in VP shunt insertions [64].

A retrograde migration of the peritoneal catheter is also well described. In these cases, the tube can end at the thoracoabdominal subcutaneous tissue (Fig. 8.6), the breast [69], the heart [57, 61], the lateral ventricle [4, 52], the subdural space [77], or extruded through the neck incision [83]. The mechanism here is more complex and multifactorial. The migration may be the result of abdominal wall contractions that can expel the shunt catheter into the fibrous tract surrounding the catheter [69]. Increased intraabdominal pressure due to obesity, constipation, or pseudocyst formation may contribute. Anchoring of the shunt tube to a more proximal calcified point may act like a “windlass” resulting in traction of the tube. The retained memory effect of the catheters that are coiled into the manufacturer’s package could be another possible explanation [20]. Forceful rotation or flexion–extension movements of the head and neck can pull out the distal catheter from the peritoneal cavity [69]. In girls the distal tube that is tunneled closely to the breast may become adherent to it and eventually, when the breast grows, can – theoretically – facilitate the upward migration of the catheter. Similar result can have the deterioration of severe scoliosis in the calcified shunt.

Fig. 8.6 Lateral abdominal radiograph of a 5-year-old girl who presented a few months after shunt insertion with symptoms of raised intracranial pressure. The distal catheter has coiled up in the region of the abdominal wound, outside the peritoneal cavity

The migration of the distal tube into the heart or more distally in the pulmonary vasculature is also described. Until 2011, there were 13 reported cases [57]. One mechanism proposed transvenous placement of the catheter, followed by proximal migration into the pulmonary artery. The transvenous placement must have occurred during the initial subcutaneous tunneling and further migration due to negative intrathoracic pressure and venous flow [26]. A different mechanism involved chronic erosion of a neck vein by the adherent shunt tube with subsequent proximal migration into the pulmonary artery [54].

The migration of a lumboperitoneal shunt catheter intracranially has also been described.

In this case report [5], it occurred 10 months after the insertion and the tip of it was found in the deep parenchymal structures. Possibly it was caused by spinocerebral CSF flow.

8.2.3 Presentation

Fractures, disconnections, and dislocations of shunt components usually present as shunt malfunctions. But they do not always become clinically evident. In a retrospective study it was found that from the 25 cases of disconnections in 22 patients, only 9 (40.9 %) had nonfunctioning shunts and these shunts were removed in 8 patients [48]. In the above mentioned study, only 3 out of 9 patients with nonfunctioning shunts had no symptoms [48]. It should be kept in mind that shunt failure is a neurosurgical emergency until proven otherwise and that a high index of suspicion must be maintained when receiving a shunted patient with symptoms [56]. A patient shunted in his early childhood for posthemorrhagic hydrocephalus or after MMC closure and presents after a few years with evidence of shunt disconnection is extremely likely to have signs and symptoms of increased intracranial pressure. All these patients should have proper clinical evaluation, including fundoscopy. On the other hand, many disconnections are found incidentally, usually in scheduled follow-up. The typical finding is the discontinuation of the radiolucent catheter strip, between the retroauricular area and the clavicle, in a formal shunt series (plain X-ray films). Less frequently, the ventricular catheter is found disconnected from its burr-hole attachment.

In specific cases of migration, the clinical findings are related to the organ or body compartment that is involved. External protrusion or extrusion is evident by the caregivers and often has minimal clinical sequelae. Worsening respiratory distress and “butterflies in the chest” were findings in patients with migration to the pulmonary vasculature [57]. Recurrent pneumonia was the result of a dislocated distal catheter into the lung [62] and several authors have reported CSF galactorrhea because of a distal catheter that migrated into the breast [47, 51].

8.2.4 Management

As already mentioned, a patient with a disconnected shunt should be treated with great caution, even if he/she remains asymptomatic. Besides the shunt series, Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) are crucial in order to compare the findings with previous scan and appreciate any changes in the ventricular size and the subarachnoid spaces. The imaging features of the third ventricle morphology should be reviewed, in case an endoscopic third ventriculostomy (ETV) becomes an alternative option. Radionuclide shuntograms are reported to provide additional information, but are rarely performed in practice. Shunt taps are used so that the functional status of the system is assessed. Attempts to use ultrasound to verify shunt function have not been established in routine clinical practice [68]. The most reliable technique to diagnose shunt malfunction is the intraoperative exploration. A disconnected system can be replaced as a whole or in parts. Additional connections should be avoided. In all the cases, the patient is prepared and draped as if a revision of the whole system is planned.

The finding of a disconnected shunt can be a chance for the patient to become shunt free. Of course, this should be attempted with caution in selected cases. Iannelli et al. [36] showed that in 17 out of 27 children (63 %), they detected CSF shunt independency at the time of a scheduled catheter lengthening procedure. The spectrum of candidates for an ETV is becoming wider and should always be kept in mind as an alternative. Neuroendoscopy can be applied in cases of intraventricular migration of proximal catheters. Usually, the burr hole is enlarged so that the endoscope can be inserted and with a grasping forceps the catheter can be grabbed and retrieved. If that becomes difficult it should be left behind and followed.

In the specific cases of externally protruded catheters, the decision making is more straightforward. CSF samples are sent for analysis, microbiology, and cultures. If there is clinical or laboratory evidence of infection the whole system is removed, an external drain is inserted, and antibiotics are administrated intravenously and possibly intrathecally, based on the local Infectious Diseases (ID) team guidelines. If the CSF is clear, then only the distal part can be removed *outward* in a fashion that prevents spread of the infection closely to the remaining shunt. If there is no clinical or radiological evidence of recent bowel perforation, then a new catheter can be inserted into the peritoneal cavity. A general surgeon can provide his assistance in such cases or – more importantly – if there are signs of intraperitoneal perforation and infection. General surgeons are also helpful in cases of laparoscopic retrieval of disconnected catheters, even as a day-case procedures [73].

Retrieval of migrated catheters into the heart or the pulmonary artery has several options. Some authors reported a smooth removal through a retroauricular incision without any complications [61]. Others utilized fluoroscopic guidance successfully [26] or requested assistance from cardiothoracic surgeons due to potential hazards threatening the cardiac valves or chambers [27]. Endovascular methods are another vital option [16] and nevertheless, whenever the surgeon feels unfamiliar with less-known territories, asking for help from specialized expertise is always wise.

References

1. Adeloye A (1973) Spontaneous extrusion of the abdominal tube through the umbilicus complicating peritoneal shunt for hydrocephalus. Case report. J Neurosurg 38(6):758–760
2. Aihara N, Takagi T, Hashimoto N et al (1997) Breakage of shunt devices (Sophy programmable pressure valve) following implantation in the hypochondriac region. Childs Nerv Syst 13(11–12): 636–638
3. Aldrich EF, Harmann P (1990) Disconnection as a cause of ventriculoperitoneal shunt malfunction in multicomponent shunt systems. Pediatr Neurosurg 16(6):309–311; discussion 312
4. Ammar A, Nasser M (1995) Intraventricular migration of VP shunt. Neurosurg Rev 18(4):293–295
5. Anthogalidis EI, Sure U, Hellwig D, Bertalanffy H (1999) Intracranial dislocation of a lumbo-peritoneal shunt-catheter: case report and review of the literature. Clin Neurol Neurosurg 101(3):203–206
6. Aryan HE, Meltzer HS, Park MS et al (2005) Initial experience with antibiotic-impregnated silicone catheters for shunting of cerebrospinal fluid in children. Childs Nerv Syst 21(1):56–61
7. Beez T, Sarikaya-Seiwert S, Bellstadt L, Muhmer M, Steiger HJ (2014) Role of ventriculoperitoneal shunt valve design in the treatment of pediatric hydrocephalus-a single center study of valve performance in the clinical setting. Childs Nerv Syst 30(2):293–297
8. Berhouma M, Messerer M, Houissa S, Khaldi M (2008) Transoral protrusion of a peritoneal catheter: a rare complication of ventriculoperitoneal shunt. Pediatr Neurosurg 44(2):169–171
9. Boch AL, Hermelin E, Sainte-Rose C, Sgouros S (1998) Mechanical dysfunction of ventriculoperitoneal shunts caused by calcification of the silicone rubber catheter. J Neurosurg 88(6):975–982
10. Browd SR, Gottfried ON, Ragel BT, Kestle JR (2006) Failure of cerebrospinal fluid shunts: part II: overdrainage, loculation, and abdominal complications. Pediatr Neurol 34(3):171–176
11. Browd SR, Ragel BT, Gottfried ON, Kestle JR (2006) Failure of cerebrospinal fluid shunts: part I: obstruction and mechanical failure. Pediatr Neurol 34(2): 83–92
12. Brownlee JD, Brodkey JS, Schaefer IK (1998) Colonic perforation by ventriculoperitoneal shunt tubing: a case of suspected silicone allergy. Surg Neurol 49(1): 21–24
13. Burnette DG Jr (1982) Bladder perforation and urethral catheter extrusion: an unusual complication of cerebrospinal fluid-peritoneal shunting. J Urol 127(3): 543–544
14. Charalambides C, Sgouros S (2012) Spontaneous knot formation in the peritoneal catheter: a rare cause of ventriculoperitoneal shunt malfunction. Pediatr Neurosurg 48:310–312
15. Chen HH, Riva-Cambrin J, Brockmeyer DL, Walker ML, Kestle JR (2011) Shunt failure due to intracranial migration of BioGlide ventricular catheters. J Neurosurg Pediatr 7(4):408–412
16. Chong JY, Kim JM, Cho DC, Kim CH (2008) Upward migration of distal ventriculoperitoneal shunt catheter into the heart: case report. J Korean Neurosurg Soc 44(3):170–173
17. Coleman DL, Lim D, Kessler T, Andrade JD (1981) Calcification of nontextured implantable blood pumps. Trans Am Soc Artif Intern Organs 27:97–104
18. Decq P, Barat JL, Duplessis E et al (1995) Shunt failure in adult hydrocephalus: flow-controlled shunt versus differential pressure shunts–a cooperative study in 289 patients. Surg Neurol 43(4):333–339
19. Del Bigio MR, Fedoroff S (1992) Short-term response of brain tissue to cerebrospinal fluid shunts in vivo and in vitro. J Biomed Mater Res 26(8):979–987
20. Dominguez CJ, Tyagi A, Hall G, Timothy J, Chumas PD (2000) Sub-galeal coiling of the proximal and distal components of a ventriculo-peritoneal shunt. An unusual complication and proposed mechanism. Childs Nerv Syst 16(8):493–495
21. Drake JM, Kestle JR, Milner R et al (1998) Randomized trial of cerebrospinal fluid shunt valve

design in pediatric hydrocephalus. Neurosurgery 43(2):294–303; discussion 303-295
22. Drake JM, Kestle JR, Tuli S (2000) CSF shunts 50 years on–past, present and future. Childs Nerv Syst 16(10–11):800–804
23. Echizenya K, Satoh M, Murai H et al (1987) Mineralization and biodegradation of CSF shunting systems. J Neurosurg 67(4):584–591
24. Elisevich K, Mattar AG, Cheeseman F (1994) Biodegradation of distal shunt catheters. Pediatr Neurosurg 21(1):71–76
25. Etus V (2011) Ventriculoperitoneal shunt catheter protrusion through the anus: case report of an uncommon complication and literature review. Commentary. Childs Nerv Syst 27(11):2015
26. Fewel ME, Garton HJ (2004) Migration of distal ventriculoperitoneal shunt catheter into the heart. Case report and review of the literature. J Neurosurg 100(2 Suppl Pediatrics):206–211
27. Frazier JL, Wang PP, Patel SH et al (2002) Unusual migration of the distal catheter of a ventriculoperitoneal shunt into the heart: case report. Neurosurgery 51(3): 819–822; discussion 822
28. Ghritlaharey RK, Budhwani KS, Shrivastava DK et al (2007) Trans-anal protrusion of ventriculo-peritoneal shunt catheter with silent bowel perforation: report of ten cases in children. Pediatr Surg Int 23(6): 575–580
29. Glatstein M, Constantini S, Scolnik D, Shimoni N, Roth J (2011) Ventriculoperitoneal shunt catheter protrusion through the anus: case report of an uncommon complication and literature review. Childs Nerv Syst 27(11):2011–2014
30. Goldblum RM, Pelley RP, O'Donell AA, Pyron D, Heggers JP (1992) Antibodies to silicone elastomers and reactions to ventriculoperitoneal shunts. Lancet 340(8818):510–513
31. Griebel R, Khan M, Tan L (1985) CSF shunt complications: an analysis of contributory factors. Childs Nerv Syst 1(2):77–80
32. Harasaki H, McMahon J, Richards T et al (1985) Calcification in cardiovascular implants: degraded cell related phenomena. Trans Am Soc Artif Intern Organs 31:489–494
33. Harasaki H, Moritz A, Uchida N et al (1987) Initiation and growth of calcification in a polyurethane-coated blood pump. ASAIO Trans 33(3):643–649
34. Heggers JP, Kossovsky N, Parsons RW et al (1983) Biocompatibility of silicone implants. Ann Plast Surg 11(1):38–45
35. Hussain NS, Wang PP, James C, Carson BS, Avellino AM (2005) Distal ventriculoperitoneal shunt failure caused by silicone allergy. Case report. J Neurosurg 102(3):536–539
36. Iannelli A, Rea G, Di Rocco C (2005) CSF shunt removal in children with hydrocephalus. Acta Neurochir (Wien) 147(5):503–507; discussion 507
37. Israelsson LA, Jonsson T (1994) Physical properties of self locking and conventional surgical knots. Eur J Surg 160(6–7):323–327
38. Jimenez DF, Keating R, Goodrich JT (1994) Silicone allergy in ventriculoperitoneal shunts. Childs Nerv Syst 10(1):59–63
39. Joubert MJ, Stephanov S (1983) Extrusion of peritoneal catheter through the mid-lumbar region. An unusual complication of ventriculo-peritoneal shunt. Surg Neurol 19(2):120–121
40. Kang JK, Lee IW (1999) Long-term follow-up of shunting therapy. Childs Nerv Syst 15(11–12):711–717
41. Kataria R, Sinha VD, Chopra S, Gupta A, Vyas N (2013) Urinary bladder perforation, intra-corporeal knotting, and per-urethral extrusion of ventriculoperitoneal shunt in a single patient: case report and review of literature. Childs Nerv Syst 29(4):693–697
42. Koga H, Mukawa J, Nakata M, Sakuta O, Higa Y (1992) Analysis of retained ventricular shunt catheters. Neurol Med Chir (Tokyo) 32(11):824–828
43. Kronenthal RL (1975) Biodegradable polymers in medicine and surgery. In: Kronenthal RL, Oser Z, Martin E (eds) Polymers in medicine and surgery. Plenum Press, New York, pp 119–137
44. Kumar R, Singh V, Kumar MV (2005) Shunt revision in hydrocephalus. Indian J Pediatr 72(10):843–847
45. Kuo MF, Wang HS, Yang SH (2009) Ventriculoperitoneal shunt dislodgement after a haircut with hair clippers in two shunted boys. Childs Nerv Syst 25(11):1491–1493
46. Langmoen IA, Lundar T, Vatne K, Hovind KH (1992) Occurrence and management of fractured peripheral catheters in CSF shunts. Childs Nerv Syst 8(4):222–225
47. Lee SC, Chen JF, Tu PH, Lee ST (2008) Cerebrospinal fluid galactorrhea: a rare complication of ventriculoperitoneal shunting. J Clin Neurosci 15(6):698–700
48. Lee YH, Park EK, Kim DS, Choi JU, Shim KW (2010) What should we do with a discontinued shunt? Childs Nerv Syst 26(6):791–796
49. Lewis FM (1969) Elastomers by condensation polymerization. In: Kennedy JP, Tronqvist E (eds) Polymer chemistry of synthetic elastomers, part 2. Wiley, New York, pp 767–804
50. Lundar T, Langmoen IA, Hovind KH (1991) Shunt failure caused by valve collapse. J Neurol Neurosurg Psychiatry 54(6):559–560
51. Maknojia A, Caron JL (2014) Proximal subcutaneous migration of the distal end of a ventriculoperitoneal shunt presenting with recurrent cerebrospinal fluid galactorrhea. J Neurosurg 120(1):164–166
52. Mazza CA, Bricolo A (1975) Upward dislocation of peritoneal catheter into the ventricular cavity. A rare complication of the ventriculo-peritoneal shunt. Report of a case. Neuropadiatrie 6(3):313–316
53. Miserocchi G, Sironi VA, Ravagnati L (1984) Anal protrusion as a complication of ventriculo-peritoneal shunt. Case report and review of the literature. J Neurosurg Sci 28(1):43–46
54. Morell RC, Bell WO, Hertz GE, D'Souza V (1994) Migration of a ventriculoperitoneal shunt into the pulmonary artery. J Neurosurg Anesthesiol 6(2):132–134
55. Nagulic M, Djordjevic M, Samardzic M (1996) Peritoneo-vulvar catheter extrusion after shunt operation. Childs Nerv Syst 12(4):222–223

56. Neuren A, Ellison PH (1979) Acute hydrocephalus and death following V-P shunt disconnection. Pediatrics 64(1):90–93
57. Nguyen HS, Turner M, Butty SD, Cohen-Gadol AA (2010) Migration of a distal shunt catheter into the heart and pulmonary artery: report of a case and review of the literature. Childs Nerv Syst 26(8):1113–1116
58. Portnoy HD, Croissant PD (1973) Two unusual complications of a ventriculoperitoneal shunt. Case report. J Neurosurg 39(6):775–776
59. Raimondi AJ, Robinson JS, Kuwawura K (1977) Complications of ventriculo-peritoneal shunting and a critical comparison of the three-piece and one-piece systems. Childs Brain 3(6):321–342
60. Ramani PS (1974) Extrusion of abdominal catheter of ventriculoperitoneal shunt into the scrotum. Case report. J Neurosurg 40(6):772–773
61. Rizk E, Dias MS, Verbrugge J, Boop FA (2009) Intracardiac migration of a distal shunt catheter: an unusual complication of ventricular shunts. Report of 2 cases. J Neurosurg Pediatr 3(6):525–528
62. Sahin S, Shaaban AF, Iskandar BJ (2007) Recurrent pneumonia caused by transdiaphragmatic erosion of a ventriculoperitoneal shunt into the lung. Case report. J Neurosurg 107(2 Suppl):156–158
63. Sainte-Rose C, Piatt JH, Renier D et al (1991) Mechanical complications in shunts. Pediatr Neurosurg 17(1):2–9
64. Sathyanarayana S, Wylen EL, Baskaya MK, Nanda A (2000) Spontaneous bowel perforation after ventriculoperitoneal shunt surgery: case report and a review of 45 cases. Surg Neurol 54(5):388–396
65. Schoen FJ, Harasaki H, Kim KM, Anderson HC, Levy RJ (1988) Biomaterial-associated calcification: pathology, mechanisms, and strategies for prevention. J Biomed Mater Res 22(A1 Suppl):11–36
66. Sekhar LN, Moossy J, Guthkelch AN (1982) Malfunctioning ventriculoperitoneal shunts. Clinical and pathological features. J Neurosurg 56(3):411–416
67. Sgouros S, Dipple SJ (2004) An investigation of structural degradation of cerebrospinal fluid shunt valves performed using scanning electron microscopy and energy-dispersive x-ray microanalysis. J Neurosurg 100:534–540
68. Sgouros S, John P, Walsh AR, Hockley AD (1996) The value of Colour Doppler Imaging in assessing flow through ventricular shunts. Childs Nerv Syst 12:454–459
69. Shafiee S, Nejat F, Raouf SM, Mehdizadeh M, El Khashab M (2011) Coiling and migration of peritoneal catheter into the breast: a very rare complication of ventriculoperitoneal shunt. Childs Nerv Syst 27(9): 1499–1501
70. Shanklin DR, Smalley DL (1999) Dynamics of wound healing after silicone device implantation. Exp Mol Pathol 67(1):26–39
71. Shannon CN, Acakpo-Satchivi L, Kirby RS, Franklin FA, Wellons JC (2012) Ventriculoperitoneal shunt failure: an institutional review of 2-year survival rates. Childs Nerv Syst 28(12):2093–2099
72. Shaw AD, Duthie GS (1995) A simple assessment of surgical sutures and knots. J R Coll Surg Edinb 40(6):388–391
73. Short M, Solanki G, Jawaheer G (2010) Laparoscopic retrieval of disconnected shunt catheters from the peritoneal cavity as a day-case procedure in children–early feasibility report. Childs Nerv Syst 26(6):797–800
74. Snow RB, Kossovsky N (1989) Hypersensitivity reaction associated with sterile ventriculoperitoneal shunt malfunction. Surg Neurol 31(3):209–214
75. Stannard MW, Rollins NK (1995) Subcutaneous catheter calcification in ventriculoperitoneal shunts. AJNR Am J Neuroradiol 16(6):1276–1278
76. Swanson JW, Lebeau JE (1974) The effect of implantation on the physical properties of silicone rubber. J Biomed Mater Res 8(6):357–367
77. Thauvoy C (1989) Unusual complication of peritoneal drainage: migration of a shunt in the subdural space. Childs Nerv Syst 5(5):279
78. Thubrikar MJ, Deck JD, Aouad J, Nolan SP (1983) Role of mechanical stress in calcification of aortic bioprosthetic valves. J Thorac Cardiovasc Surg 86(1): 115–125
79. Tomes DJ, Hellbusch LC, Alberts LR (2003) Stretching and breaking characteristics of cerebrospinal fluid shunt tubing. J Neurosurg 98(3):578–583
80. Touho H, Nakauchi M, Tasawa T, Nakagawa J, Karasawa J (1987) Intrahepatic migration of a peritoneal shunt catheter: case report. Neurosurgery 21(2): 258–259
81. Vinchon M, Rekate H, Kulkarni AV (2012) Pediatric hydrocephalus outcomes: a review. Fluids Barriers CNS 9(1):18
82. Wakai S (1982) Extrusion of a peritoneal catheter through the abdominal wall in an infant. Case report. J Neurosurg 57(1):148–149
83. Whittle IR, Johnston IH (1983) Extrusion of peritoneal catheter through neck incision: a rare complication of ventriculoperitoneal shunting. Aust N Z J Surg 53(2):177–178
84. Woodward SC (1981) Mineralization of connective tissue surrounding implanted devices. Trans Am Soc Artif Intern Organs 27:697–702
85. Yamamoto S, Ohno K, Aoyagi M, Ichinose S, Hirakawa K (2002) Calcific deposits on degraded shunt catheters: long-term follow-up of V-P shunts and late complications in three cases. Childs Nerv Syst 18(1–2):19–25
86. Yoshida M, Ohtsuru K, Kuramoto S (1991) External dislocation of the metallic device of a shunt valve, an unusual long-term complication of the Holter valve–case report. Kurume Med J 38(2):109–112