# Ventriculoperitoneal shunts

Ventriculoperitoneal shunt insertion for treatment of hydrocephalus is one of the most common neurosurgical procedures performed in the UK, with an estimated 3000–3500 shunt operations performed per year (Jenkinson et al, 2014). Despite widespread use of ventriculoperitoneal shunts, approximately 1 in 5 patients will return to hospital within 1 year of surgery as a result of a shunt-related complication (Merkler et al, 2017). It is therefore important to understand the basic principles of ventriculoperitoneal shunts to allow comprehensive assessment and initial management of patients presenting with shunt-related complications.

## Background

Under healthy conditions, approximately 500 ml of CSF is produced by the choroid plexus in the ventricles each day (Sakka et al, 2011). Following production, CSF circulates from the lateral ventricles through the foramen of Monro into the 3rd ventricle. It then drains via the cerebral aqueduct into the 4th ventricle before exiting into the subarachnoid space through the foramina of Magendie and Luschka. After flowing through focally-enlarged areas of subarachnoid space or 'cisterns' at the base of the brain, CSF passes over the cerebral hemispheres to be absorbed by the arachnoid villi of the dural sinuses.

Hydrocephalus refers to abnormal enlargement of the ventricles as a result of excessive accumulation of CSF and is traditionally classified as either 'obstructive' or 'communicating' in nature. Obstructive hydrocephalus occurs as a result of structural blockage of CSF flow in the ventricular system (e.g. tumours, aqueduct stenosis). The less common communicating form of hydrocephalus arises from abnormalities in CSF production, impaired CSF flow outside of the ventricular system or reduced CSF absorption by the arachnoid granulations (e.g. meningitis, subarachnoid haemorrhage).

Both forms of hydrocephalus can precipitate a significant increase in intracranial pressure. The mechanism underlying raised intracranial pressure is best understood by consideration of the Monro–Kellie doctrine. This states that the cranial compartment is a rigid cavity containing three non-compressible contents: brain, blood and CSF. A small increase in the volume of any of these components may initially be tolerated with minimal change in intracranial pressure as a result of downward displacement of CSF into the spinal subarachnoid space. Once the limit of CSF displacement is reached, any further increase in the volume of skull contents (e.g. worsening hydrocephalus, enlarging haematoma) will lead to an exponential rise in intracranial pressure and a rapid deterioration in neurological status. An important exception to this rule is during infancy when hydrocephalus can result in marked enlargement of the head because of the distensibility of the skull.

Acute onset hydrocephalus typically presents as severe headache, vomiting and/or reduced consciousness. In contrast, gradual onset hydrocephalus is associated with more subtle symptoms, such as chronic headaches, behavioural disturbance, cognitive decline or deteriorating vision. If left untreated, hydrocephalus can result in oedema of the periventricular white matter, hypoxic-ischaemic brain injury and, in severe cases, cerebral herniation (Del Bigio, 1993). Hydrocephalus can be treated by placement of a CSF shunt to divert CSF to another part of the body for absorption. Various anatomical sites have been targeted to absorb diverted CSF, but the peritoneum remains the most common.

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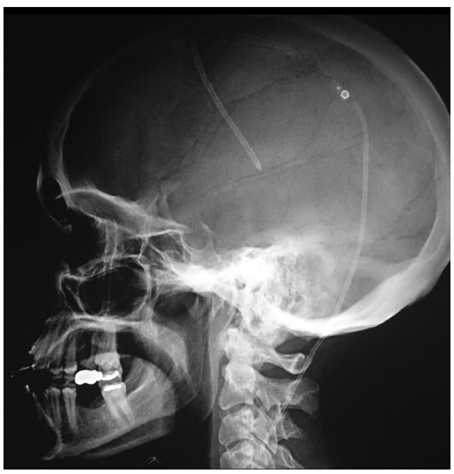
## What is a ventriculoperitoneal shunt?

A ventriculoperitoneal shunt consists of four components:

1. A proximal catheter which is placed in one of the lateral ventricles
2. A reservoir to enable CSF sampling
3. A valve to ensure unidirectional flow when a pre-determined pressure gradient is reached
4. A distal catheter tunnelled subcutaneously until it reaches the peritoneal cavity.

In addition, some modern ventriculoperitoneal shunt systems include an anti-siphon device which can help protect against overdrainage of CSF into the peritoneal cavity. The pressure setting of a ventriculoperitoneal shunt can vary widely with different valve types and manufacturers. Some modern ventriculoperitoneal shunts have programmable valves which allow the pressure threshold for CSF diversion to be adjusted non-invasively (*Figure 1*). It is important to note that these programmable valves can be inadvertently reprogrammed

Figure 1. Lateral X-ray of the skull showing a ventricular shunt catheter with a programmable valve which allows the neurosurgeon to adjust the pressure at which the shunt will open and allow drainage of CSF out of the ventricles.



by external magnetic fields. Valve settings should therefore be checked after magnetic resonance imaging according to the manufacturer's guidelines. A plain skull X-ray perpendicular to the shunt valve can reveal both the type of shunt system and current pressure setting. If found to differ from the pre-scan setting, it is important to contact the local neurosurgery team to arrange for the shunt valve to be readjusted.

## Complications of ventriculoperitoneal shunts

A retrospective cohort study revealed that 23.8% of patients developed at least one ventriculoperitoneal shunt-related complication within 4 years of surgery (Merkler et al, 2017). Complications of shunt placement can be classified broadly as problems relating to CSF flow, infection or damage to local anatomical structures.

### CSF flow complications

**Undershunting**

Obstruction is the most common cause of ventriculoperitoneal shunt malfunction and can arise at any point along the length of the shunt (Pople, 2002). The tubing can be blocked by blood or debris, entanglement with the choroid plexus, catheter fracture or disconnection, biofilm formation (usually infection related) or migration of the tubing from its desired location. The result is recurrence of the hydrocephalus symptoms with the risk of visual loss, coma and death, the severity or urgency of the situation depending on the degree to which the individual is dependent upon the shunt.

**Overshunting**

Excessive CSF drainage can occur as a result of inappropriately low CSF pressure valve settings or as a result of a siphoning effect (Pudenz and Foltz, 1991). Collapsed or 'slit' ventricles on computed tomography are observed in up to 80% of patients after ventriculoperitoneal shunt insertion (Teo and Morris, 1999), but only a minority will have symptoms suggestive of a 'low-pressure state': postural headaches and vomiting which are classically relieved by recumbency, occipital or neck pain and generalized tiredness. These symptoms typically improve with a short period of bed rest, analgesia and rehydration.

If persistent, patients may benefit from a neurosurgical review since overshunting is a common indication for shunt valve adjustment, particularly in patients whose ventriculoperitoneal shunt was inserted for treatment of normal pressure hydrocephalus (Zemack and Romner, 2008). Recurrent severe headaches unrelated to posture raise suspicion of the rare 'slit ventricle syndrome', which is thought to arise from intermittent occlusion of the proximal catheter by the ependymal lining of collapsed, non-compliant ventricles (Olson, 2004). In elderly patients with significant brain atrophy, ventricular collapse can cause tears in cerebral bridging veins and the formation of unilateral or bilateral subdural haematomas, which frequently require surgical drainage (Pudenz and Foltz, 1991).

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### Infection

Approximately 10–22% of patients with ventriculoperitoneal shunts will experience a shunt infection, usually within the first month of placement as a result of intraoperative or postoperative wound contamination with skin flora (Gutierrez-Murgas and Snowden, 2014). Consistent with this source, coagulase-negative staphylococci, *Staphyloccus aureus* and diptheroids (e.g. Propionibacterium) are the most commonly isolated pathogens in CSF cultures (Gutierrez-Murgas and Snowden, 2014).

Late onset shunt infections occur less frequently and can arise from haematogenous seeding or direct contamination of the distal catheter in the context of peritonitis (Vinchon et al, 2002). Ventriculoperitoneal shunt infections can present with few, if any, symptoms as a result of the variable inflammatory response to colonizing pathogens. While some patients will present with classic symptoms of meningism (e.g. fever, headache, neck stiffness and/or photophobia), many will remain asymptomatic until biofilm formation obstructs CSF drainage. Symptoms may also arise at the site of the distal catheter, where exposure to infected CSF can result in local or generalized peritonitis. In some cases, impaired CSF absorption by the inflamed peritoneum can result in abdominal pseudocyst formation. Following confirmation of shunt infection by CSF culture, the device is typically removed and CSF diverted via an external ventricular drain (Pople, 2002). Once parenteral antibiotics have sterilized the CSF, a new ventriculoperitoneal shunt can be inserted.

### Damage to local anatomical structures

**Proximal catheter**

Ventriculoperitoneal shunt insertion is associated with a 2.3% risk of intraparenchymal or intraventricular haemorrhage (Pople, 2002). Localized damage to the overlying cortex is also associated with an increased risk of seizures, which affect approximately 5% of patients in the first year after placement (Dan and Wade, 1986).

**Valve or catheter**

Skin irritation or breakdown along the shunt course can occur in debilitated patients who lie on the side of the ventriculoperitoneal shunt for long periods or in rare cases of silicone allergy. If left untreated, skin breakdown predisposes to an increased risk of ventriculoperitoneal shunt infection.

**Distal catheter**

Isolated abdominal pain is rarely a shunt problem, but intraperitoneal placement of the distal shunt catheter is associated with a slightly increased risk of intra-abdominal complications including peritonitis, intestinal obstruction, volvulus and strangulation (Chung et al, 2009). Low grade peritoneal catheter infection can sometimes present with 'grumbling' symptoms of abdominal pain and will usually become apparent as loculated collections around the shunt tubing on abdominal ultrasound scan or computed tomography. It may also be associated with symptoms of shunt underdrainage and ultimately tends to lead to shunt block.

## Clinical approach

Patients with shunt-related complications can present with a wide range of clinical symptoms, ranging from gradually worsening headaches to an acute, life-threatening deterioration in consciousness level. In a drowsy or unconscious patient, the airway, breathing, circulation, disability, exposure (ABCDE) approach is a systematic way to

**Table 1. Red flag symptoms of life-threatening ventriculoperitoneal shunt-related complications**

| Complication | Red flag symptoms and signs |
|---|---|
| Blocked shunt – i.e. raised intracranial pressure | Headache: progressive, worse in morning, exacerbated by recumbency or straining |
| | Nausea and/or vomiting |
| | Diplopia, squint, blurry vision, field constriction, obscurations, papilloedema |
| | Ataxia |
| | Lethargy |
| | Seizures: new onset or increased frequency if pre-existing |
| | Deteriorating school performance or disinterest in usual hobbies |
| Shunt infection | Headache |
| | Neck stiffness |
| | Photophobia |
| | Fever |
| | Rigors |
| | Night sweats |
| | Erythema or tenderness over shunt tubing |

**Table 2. Key information to obtain about a ventriculoperitoneal shunt**

| |
|---|
| First ventriculoperitoneal shunt insertion: date, indication |
| Shunt revisions: date(s), details of previous complications |
| Type of ventriculoperitoneal shunt: manufacturer, valve type (e.g. Medtronic Strata programmable valve) |
| Most recent pressure setting |
| Baseline neurological status |
| Date of last CSF sampling from shunt |
| Recent skin changes overlying shunt tubing |
| Abdominal symptoms or diagnoses |

stabilize the patient while also excluding important medical causes of reduced consciousness such as hypoxia, hypothermia, sepsis and hypoglycaemia (Thim et al, 2012). Once the patient is clinically stable, a full history should be taken, with a particular focus on 'red flag' symptoms suggestive of raised intracranial pressure, visual deterioration or CSF infection (*Table 1*) and the past medical history of the shunt (*Table 2*). In patients who are unable to communicate as a result of a reduced level of consciousness or significant neurological deficit, it is important to clarify baseline neurological function with a caregiver or historical medical records.

A full systems examination should be performed to identify possible causes for the deterioration in clinical state. On neurological examination, it is important to look for signs of elevated intracranial pressure including a reduced Glasgow Coma Scale, pupillary dilatation, bilateral ptosis, upward gaze palsy, abducens nerve palsy, reduced visual acuity or papilloedema. In babies, inspect for sun setting eyes, a bulging fontanelle and prominent scalp veins. If available, review the 'red book' to determine if the head circumference is crossing centiles. Hypertension, widened pulse pressure, bradycardia and an irregular respiratory pattern are late signs of raised intracranial pressure indicative of impending brainstem herniation. In addition to neurological signs, remember to examine the skin overlying the shunt tubing and perform an abdominal examination to identify possible extra-cranial shunt complications.

If history and examination suggest possible blockage of the ventriculoperitoneal shunt, a radiographic shunt series should be performed which comprises an anteroposterior and lateral skull X-rays, as well as anteroposterior neck, chest and abdominal X-rays. In addition to confirming the type of shunt present, these radiographs can help to identify possible sites of disconnection, fracture or tip migration. If there is suspicion of disordered CSF drainage, shunt infection or intracranial haemorrhage, an urgent non-contrast enhanced computed tomography head scan should be performed. Attempts to obtain a CSF sample by direct aspiration from the ventriculoperitoneal shunt should be avoided without prior discussion with the local neurosurgery team.

## Referral to neurosurgery

### Emergency referrals

In a patient with a ventriculoperitoneal shunt and a deteriorating level of consciousness, or clinical concern of acutely rising intracranial pressure, urgent referral to neurosurgery is advised. In the majority of cases, a referral should be made with the results of an urgent computed tomography head and X-ray shunt series, unless a significant delay in imaging is anticipated. Essential clinical information to communicate includes the nature, time of onset and rate of progression of the symptoms, along with an up-to-date Glasgow Coma Scale broken down into eye, verbal and motor scores. To facilitate planning of emergency surgery, it is important to report any medical comorbidities which may affect anaesthetic risk, the use of blood thinning medications, when the patient last ate and confirmation of nil-by-mouth status.

### Urgent referrals

Many patients with ventriculoperitoneal shunt-related complications are alert, with a subacute history of progressive symptoms. In these situations prompt discussion with neurosurgery is recommended but will not always require immediate transfer for surgical management. There is time to gather more clinical information to help the neurosurgeon on the end of the telephone draw some conclusions. When symptoms are vague, the thoughts of an experienced family member or caregiver are often the most discriminatory part of the history. Be sure to ask how quickly the patient became unwell and how closely the symptoms resemble the last time there was a shunt problem.

While it is important to ask whether new symptoms could be related to the ventriculoperitoneal shunt, it is equally important to consider what the diagnosis might be if the patient did not have a shunt. For example, a patient with a ventriculoperitoneal shunt and fever should have a septic screen performed before consideration of a shunt tap (aspiration through a needle passed into reservoir) or lumbar puncture. Alternative diagnoses should have been considered and, where appropriate, addressed before consultation with neurosurgery.

## Conclusions

Ventriculoperitoneal shunts are critical to the management of hydrocephalus but are also associated with a significant risk of both early and late postoperative complications. A comprehensive history and examination can help to identify possible ventriculoperitoneal shunt-related complications at an early stage, guide initial investigations and facilitate referral to neurosurgery colleagues for consideration of surgical management. **BJHM**

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### KEY POINTS

- Ventriculoperitoneal shunts are the mainstay of treatment for hydrocephalus.
- One in five patients will develop complications within 1 year of ventriculoperitoneal shunt insertion.
- Complications can be classified as disorders of CSF flow, shunt infection or damage to local anatomical structures along the length of the shunt.
- Patients with a ventriculoperitoneal shunt who present with a reduced level of consciousness should initially be managed with an ABCDE approach.
- Once stable, a full history and neurological examination should guide initial imaging before referral to neurosurgery.


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