Intracranial Shunts: A Brief Review for Radiologists

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Abstract

Intracranial shunts are amongst the most commonly performed neurosurgical procedures. These are often inserted long term and consequently, imaged in both short term and long term for stability, complications and follow up. While the dedicated neuroradiologists are usually familiar with the shunts, imaging appearances and complications, those radiologists not closely working with neurosurgeons often do not necessarily know the basic features and complications of these shunts. Most of the literature provide either extensive or piecemeal information on the shunts that several radiologists find difficult to confidently follow and usually the radiologists are either under confident to report on such shunt appearances or their complications. This brief review intends to provide useful information to the radiologists to know the basics of these shunts and their potential complications.

Introduction

Cerebrospinal fluid (CSF) is a colorless clear fluid which is produced primarily by choroid plexus of the brain ventricles, ependymal cells lining, and the lining surrounding the subarachnoid space [1] the main functions of the CSF are providing buoyancy, protecting the brain by acting as a shock absorber and maintaining chemical stability [2].

Hydrocephalus is a term which describes an increased amount of CSF which may be due to increased production, abnormal absorption or flow, the end result of which is raised intracranial pressure and ventricular dilation [3] Intracranial hypertension due to hydrocephalus, develops in both acute and chronic neurosurgical pathologies and is an important predictor of morbidity and mortality in patients with severe brain injury [4,5]. Different studies reported surgical intervention for managing progressive hydrocephalus significantly reduces mortality and improve outcome the majority of cases [6-10].

CSF shunts are commonly utilized in the management of hydrocephalus. In fact insertion of CSF shunts has become one of the commonest procedures in the modern neurosurgical practice [11]. To achieve a long term internalized CSF diversion, CSF shunts generally consist of two tubes, one of which is placed directly into the ventricle, typically the frontal or occipital horns of the non dominant lateral ventricle (often referred to as the ventricular/proximal catheter or tube) [12] (Figure 1), the ventricular catheter exit through hemispheric parenchyma via a burl hole made on the skull, to connect to the “in-flow” side of a valve which maintains a unidirectional flow and prevents the backflow of CSF into the ventricle [13].

Attached to the “outflow” side of the valve is the second tube (often referred to as distal tube or catheter) that is tunneled subcutaneously before terminating in the peritoneal cavity (the commonest site), the right atrium, subclavian vein, pleura, gall bladder or other site from which CSF is ultimately absorbed [14,15]. (Figure 1) Shunt Catheters and valves are impregnated with radiopaque markings to allow radiographic visualization [13]. Often a reservoir is also present proximal to the valve which allows sampling of CSF and a crude method to clinically assess shunt malfunction via pumping test (Figure 1).

Ideally, every shunt reservoir should compress easily and refill rapidly. If the reservoir can be depressed easily but refills...
poorly or doesn’t refill, then the shunt is obstructed proximally and patent distally. If the pump refills rapidly, while the reservoir does not compress easily, then there is an obstruction to CSF flow along the distal limb, with a patent proximal shunt [13,16]. A previous study showed that the reservoir pumping test carries sensitivity of 19% and specificity of 81% if conducted properly [17].

As any other surgical procedure, despite the advancement in the technical and manufacturing aspects of CSF, the shunts remain prone to numerous complications [18] (Table 1). Shunt failure is defined as a shunt complication requiring revision or replacement [19]. Aetiologies of shunt failure include obstruction, valve failure, infection and excessive or insufficient drainage [19]. In pediatric shunt procedures, it has been noted that 14% of shunt failure occur within the first month of shunt placement [20,21]. Adults are not immune to this phenomenon, with 29% of them experiencing shunt failure within the first year [22]. Long-term studies have shown that approximately 50% of individuals will require shunt revision at some point [22,23].

**Table 1**: Usual sites of ventricular drainage and the terminology used.

<table>
<thead>
<tr>
<th>Shunt Type</th>
<th>Drainage Site</th>
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</thead>
<tbody>
<tr>
<td>Ventriculoperitoneal (VP)</td>
<td>Ventricle to peritoneum cavity</td>
</tr>
<tr>
<td>Ventriculoatrial (VA)</td>
<td>Ventricle to the right atrium</td>
</tr>
<tr>
<td>Ventriculopleural (VP)</td>
<td>Ventricle to pleural cavity</td>
</tr>
<tr>
<td>Lumboperitoneal (LP)</td>
<td>Lumbar spine to peritoneal cavity</td>
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**Infection**

Shunt Infections or ventriculostomy related infections (VRI) are usually the result of commensal organisms, which include coagulase negative staphylococcal infections, present on the skin gaining access to the shunt tubing during the procedure [24]. The reported incidence varies from 1% to 39%, with the average being approximately 10% [25].

In the majority of shunt infection there is radiological evidence of shunt malfunction often features of shunt underdrainage [18]. Enhancement of the ventricular ependymal lining or leptomeninges is often visualized in contrast CT and MR. In cases of infection shunt replacement is almost always indicated [26].

A meta-analysis study looking at 35 observational studies, estimated the VRI rate to be 11.4/1000 catheter days [27]. It also looked concluded that 64% of culture positive infections were caused by Gram-positive bacteria 39% of which grew S. Epidermidis and 15% S. Aureus. Two main issues posed by shunt infections are that they carry negative prognostic impacts for the patients involved and secondly, revisions cost about eight times the price compared to original insertion procedure [28,29].

**Shunt Obstruction (Under Drainage)**

This usually presents with clinical features of raised intracranial pressure as the shunt is draining too little or no CSF for a particular patient [30]. Shunt obstruction can occur at any time following the insertion and at any point along the course of the shunt system [30]. Although clinical methods to detect possible site(s) for shunt blockage have been described in the literature (see above), imaging studies remains the gold standard for assessing the shunt system and predicting the location of the blockage [31].

The scan shows evidence of increased intracranial pressure, and if the blockage was due to mechanical reasons (shunt disconnection or fracture) a shunt series scan will demonstrate evidence of discontinuity of the shunt system [30,32]. Catheter misplacement (either ventricular or distal) will inevitably lead to under drainage of CSF and possibly obstruction if left untreated [33] (Figure 3). Symptomatic Shunt obstruction will always require surgical revision [30,31,33].
Shunt Over-Drainage

This refers to the state where a shunt is functioning properly but is removing too much CSF than required for a particular patient [33]. If this happens over a short period of time following the shunt insertion, the early rapid reduction in ventricular size may result in collapse of the brain and accumulation of extraxial fluid (most commonly CSF) or blood [34] (Figure 4). These changes are often detected on a plain CT head. Surgery is not always indicated in these cases [35]. If shunt over drainage occurs over a long period of time untreated, it may results in small ("slit") ventricles syndrome [36] (Figure 5). In these patients the ventricular stiffness and lack of ventricular compliance will prevent any ventricular enlargement in cases of shunt blockage and hence making a diagnostic scan more challenging [37].

![Figure 4: Excess CSF drainage. MRI T2W (a) and Post contrast T1 (b). Black arrows show thin subdural fluid in (a) and pachymeningeal enhancement in (b) due to low intracranial pressure.](image)

![Figure 5: Slit ventricle syndrome: CT images showing slit like ventricles (white arrows) and several shunts (black arrows). Despite adequate drainage over a period of time, patient continues to have persistent headaches.](image)

On imaging, some of these features are easy to see, such as broken shunt or hydrocephalus, it is important that careful comparison with previous imaging is done. A non contrast CT is usually adequate for such cases, while the second tube usually needs a shunt series using plain radiographs through the entire course of the shunt. Occasionally, a CT may be necessary. Infections are more difficult and best assessed by contrast enhanced MRI. Enhancement of ventricular lining (ventriculitis) is a highly specific feature and usually carries a bad prognosis. A diffusion MRI can often show exudates as areas of restricted diffusion layered within ventricles (Figure 6). Leptomeningeal enhancement can also be seen, although pachymeningeal enhancement is non-specific since it can also be associated with low intracranial pressure rather than infection. A slit ventricle syndrome is more difficult since it would represent increased pressure in absence of dilated ventricles (rather slit like ventricles) and would often need close correlation with clinical and imaging findings, including previous studies. We have provided a brief review of the structure, pattern, imaging appearances and usual complications of intraventricular shunts, that most radiologists reporting CT or MRI heads should know, but there are often gaps in their understanding that they are afraid to ask.

![Figure 6: Exudates on Diffusion weighted images. White arrows show areas of restricted diffusion within ventricles and sulcal spaces consistent with exudates suggesting CSF infection.](image)

References


