Hydrocephalus shunt infections and their treatment

Hydrocephalus shunts are used to drain cerebrospinal fluid (CSF) from the cerebral ventricles to another body cavity, usually the cardiac atrium (ventriculo-atrial, VA) or the peritoneal cavity (ventriculo-peritoneal, VP). The fluid must be drained at a controlled rate, and unidirectional valves are incorporated for this purpose. Shunts are made from medical grade silicone rubber, and sometimes with stainless steel components.

The major cause of failure of CSF shunts is infection or colonization. The organisms responsible are mainly coagulase-negative staphylococci, of which Staphylococcus epidermidis sensu stricto is the commonest type involved, but other organisms such as candida, enterococci, coryneforms and propionibacteria account for a smaller proportion of cases. Staph. aureus and Gram-negative bacilli are sometimes reported and appear to be more common in some units than in others, though they usually cause either ventriculitis or wound infections around the shunt and their role in true, internal colonization of the shunt is doubtful.

The treatment of shunt colonization usually involves removal of the shunt, followed by external ventricular drainage or the insertion of an Ommaya reservoir for pressure control, though in a few cases no further shunting is needed. However, some workers have recommended the attractive method of giving antibiotics without shunt removal, thus keeping a working shunt and avoiding two operations (Luthardt, 1970; McLaurin, 1973; O'Brien, Parent & Davies, 1979; Wald & McLaurin, 1980). Unfortunately this method has had a low success rate but this has often been associated with the use of unsuitable antibiotics or of inappropriate routes of administration. A further confounding factor is that almost any antibiotic to which the organism is sensitive will lead to clinical improvement whichever route it is given by, and the required careful, aggressive follow-up is often omitted so that relapses are not documented. In a review of such cases (Bayston & Rickwood, 1981) cloxacillin and chloramphenicol, while they often brought about clinical improvement, gave no success in actual eradication of the shunt infection, and subsequent experience has confirmed the unsuitability of these drugs. In the same study, the use of intraventricular gentamicin along with oral trimethoprim was successful where the organisms, which were all coagulase-negative staphylococci, were sensitive in vitro. We have since used a similar regimen of intraventricular gentamicin, intravenous trimethoprim and oral rifampicin in seventeen cases, eleven of which had their infections eradicated without shunt removal. A comparative study of shunt removal and antibiotics alone was not carried out until 1980 (James et al., 1980). Patients with shunt infections were treated according to one of three regimens. Group A underwent shunt removal and external ventricular drainage; Group B had shunt removal and immediate placement of a new shunt; and Group C had antibiotics without shunt removal. Patients in all three groups received both intravenous and intraventricular antibiotics. Almost all patients in Groups A and B were treated successfully but in Group C only 30% of patients were cured and they spent significantly longer in hospital. The drugs used in Group C were mainly methicillin, ampicillin and cephalothin and the results in this group were not as good as ours using gentamicin, trimethoprim and rifampicin as described above. Nevertheless, the overall results are broadly consistent and it is clear that, except in particular cases, the infected shunt should be removed as part of the treatment.

Once a colonized shunt has been removed, the sites likely to harbour organisms are the cerebral ventricles and, in the case of VP shunts, the peritoneal cavity. While infection due to coagulase-negative staphylococci in these sites will often resolve spontaneously, antibiotics are indicated to ensure rapid eradication so that a new shunt can be inserted as soon as possible, thus minimizing the risk of
secondary infection from external drainage or reservoir puncture. Virtually any suitable antibiotic to which the organism is sensitive will be effective against the peritonitis, and fluoxacillin, erythromycin or fusidic acid are useful for both coagulase-positive and -negative staphylococci. Where enterococci are involved, amoxyceillin or erythromycin may be used, but for more resistant organisms such as 'JK' coryneforms, vancomycin may be the only appropriate antibiotic. In the interest of speed of eradication and because, immediately after shunt removal, oral intake may have to be restricted, these drugs should be given intravenously. In the presence of florid ventriculitis with high CSF neutrophil count and protein, these drugs will also penetrate into the cerebral ventricular system. In some cases ventriculitis, usually with an extremely weak inflammatory response, persists despite intravenous antibiotics and the problem is often one of achieving therapeutic CSF levels. In such cases intraventricular gentamicin should be given and the intravenous therapy can usefully be changed to trimethoprim with addition of oral rifampicin. Both of these drugs will give therapeutic levels in the CSF in most patients even when ventricular inflammation is minimal, as is often the case. They will also eradicate the organisms from the peritoneal cavity. In the case of coagulase-negative staphylococci, this site can be assumed to be clear after five to seven days' treatment if the patient is comfortable and apyrexial, if there is no local erythema of the abdominal wall and if the serum C-reactive protein levels have returned to normal. Resolution of the ventriculitis should be determined by examination of the ventricular CSF.

Nowadays, multiple antibiotic resistance is common in coagulase-negative staphylococci, and shunt infections due to resistant enterococci and JK coryneforms are also encountered. In such cases, vancomycin has been used, but unfortunately, this drug does not usually penetrate sufficiently into the CSF when given intravenously, and for this reason it has occasionally been given directly into the cerebral ventricles (Raoult, et al., 1981; Sutherland et al., 1981; Young, Ratner & Clarridge, 1981). We have recently treated ten cases of shunt-associated ventriculitis due to coagulase-negative staphylococci and enterococci with intraventricular vancomycin (Bayston et al., 1984). The treatment failed in only two cases, in one of which the infected shunt was not removed. In the other case the patient experienced a histamine-like reaction to the first dose of intravenous vancomycin which was also given in this instance, and this was stopped. No detectable toxicity resulted from the intraventricular administration of the drug. In such cases, intraventricular vancomycin should be accompanied by oral or, in some cases intravenous rifampicin. Except in cases of infection with multi-resistant organisms, intravenous vancomycin is not indicated and risk of toxicity can thus be avoided. Chronic, undiagnosed or untreated VA shunt colonization will eventually give rise to immune complex nephropathy in the majority of cases, and the resulting diminished renal function should be borne in mind when drugs such as vancomycin and aminoglycosides are administered systemically. Subject to the antibiograms, other less toxic drugs are usually more appropriate for the extracranial lesions.

Shunt infections due to Candida may or may not be confined to the shunt lumen, but in any case the shunt should be removed without delay and amphotericin B and flucytosine administered as for other systemic candida infections. In cases of refractory candida ventriculitis, amphotericin B may have to be given intraventricularly but the possibility of toxicity (Fisher et al., 1983) should be appreciated. Candida pseudomycelium is often produced abundantly inside the shunt system and usually causes malfunction due to blockage.

Most shunt-associated infections due to Gram-negative rods such as E. coli involve the ventricles and abdomen (in VP shunts) and often tissues around the shunt track, as well as commonly giving rise to septicaemia. The whole shunt system should be removed without delay and the patient treated vigorously for ventriculitis, septicaemia and peritonitis. Most cases due to Staph. aureus should also be treated in this way, and attempts to retain the shunt lead to increased mortality rates.

Bacterial meningitis due to Haemophilus influenzae, Neisseria meningitidis or Streplococcus pneumoniae probably occurs with normal frequency in shunted patients and, perhaps surprisingly, the symptoms are usually no more severe. While a natural reaction is to remove the shunt as part of the management, this is rarely necessary and these organisms can usually be eradicated in the usual way with the shunt in situ. They do not appear to be capable of colonizing the shunt, and if the system is functioning satisfactorily it should not be removed. In cases where treatment of meningitis is delayed and a VP shunt is used, this may block at the distal end, but the blockage does not usually present until the meningitis has
resolved. In such cases, revision of the distal catheter only is often sufficient.

While hydrocephalus shunt infections are often low-grade and sometimes asymptomatic, failure to diagnose and treat them effectively leads to repeated operations for malfunction in VP shunts and chronic ill health and immune complex nephropathy leading to renal failure in VA shunts. There is also reason to believe that early diagnosis improves the chances of successful treatment, and the problem of infections in which the symptoms are absent, vague or misleading can be overcome by the use of a serological surveillance system (Bayston, 1975; Bayston & Swinden, 1979).

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References