COMPLICATIONS OF VENTRICULOPERITONEAL

SHUNT INSERTION AS SEEN AT

KENYATTA NATIONAL HOSPITAL

A PROSPECTIVE STUDY

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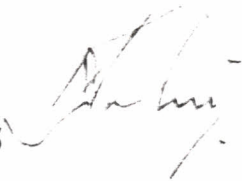
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M. B. Ch. B (Nbi) 1995

DECLARATION

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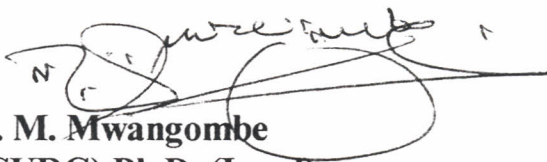
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ABBREVIATIONS

CT scan- Computerized (axial) Tomography scan

VP   - Ventriculoperitoneal

VA   - Ventriculoatrial

HC   - Hydrocephalus

CSF   - Cerebrospinal fluid

WBC   - White blood cells

SHO   - Senior house officer

KNH   - Kenyatta National Hospital

ACKNOWLEDGEMENTS

<u>DEDICATION</u>

This work is dedicated to my daughters Sumayya and Suhaila.

SUMMARY

A prospective study of 76 patients operated on for non-tumour hydrocephalus by Ventriculoperitoneal (VP) shunt insertion was carried out at the Kenyatta National Hospital between 1ST September 2001 and 28TH February 2002. The commonest cause of the hydrocephalus in this study was congenital (72.4%). The ages of the patients ranged from one month to 54 years and there were more males than females at a ratio of 1.5:1.

Twenty-three patients developed shunt complication, giving a complication rate of 30.3%. Shunt infection accounted for 65.2% of the complications and shunt malfunction/blockage for 30.5%.

The commonest clinical features associated with shunt infection in this study were fever and irritability.

Vomiting and a bulging tense anterior fontanelle were the commonest clinical features seen in shunt blockage.

The average time interval between diagnosis of hydrocephalus and first V-P shunt insertion was 4 months in about fifty percent of the patients.

Of the different variables studied, (age of patient, interval between onset of hydrocephalus and shunt insertion, length of surgery, seniority of the surgeon and use of prophylactic antibiotics) none had any correlation to the presence of shunt infection ($p > 0.05$). However, type of shunt used was associated with the development of shunt complication ($p < 0.05$).

The mortality in patients who developed shunt complications in this study was 30.4 %. All the patients who died were being managed for shunt infection.

INTRODUCTION

Hydrocephalus implies an increase in the amount of cerebrospinal fluid (CSF) within the ventricular system. It is one of the commonest conditions seen in neurosurgical units. Studies between 1965 and 1967 by Beck and Lipschitz in Bantu children in Johannesburg showed an incidence of 1:1500[1]. Similar incidences have been quoted in studies by Lawrence and Coates (1962)[2].

Hydrocephalus can be congenital or acquired, and may be due to a disturbance in its secretion, flow, or absorption. Treatment is mainly surgical, by use of a CSF diversion technique .The earliest major successful advances in CSF shunting were made at the turn of the 20th century when Ferguson in 1898 diverted CSF into the peritoneal cavity, and Kausch in 1905 performed a ventriculo-peritoneal (VP) shunt insertion [3]. CSF shunts are devices incorporating a valve to allow only a controlled unidirectional CSF flow. The shunts are surgically placed to drain CSF from a cerebral ventricle to another body cavity.

Ventriculo-atrial and ventriculo-peritoneal shunt systems are the most widely used, with V-P shunts being the preferred alternative in recent times. Our discussion will mainly center on V-P shunts, since they are the ones commonly used at the Kenyatta National Hospital (KNH).

A retrospective study by Mwangombe and Omulo which reviewed cases of non-tumour hydrocephalus seen at KNH between 1982 and 1991 showed a post-operative shunt infection rate of 24.6%[4]. A study done in 1989 by Gichuhi found infection to be the second most common complication of V-P shunt insertion after shunt malfunction at KNH [5].

This six-month prospective study was done to determine the common causes of non-tumour hydrocephalus and the different types of shunt complications and their frequency as seen at the Kenyatta National Hospital. Also looked at in this study were factors that influenced shunt infection.

LITERATURE REVIEW

SURGICAL ANATOMY

The Central Nervous System develops from a hollow tube whose cavity persists, giving rise to the ventricular system. This is lined throughout with ependyma, a single epithelial-like layer of cells. Cerebrospinal fluid is secreted by the choroid plexus, which is a combination of capillaries, pia and ependyma [6].

Each cerebral hemisphere possesses its cavity, the lateral ventricle. The diencephalon has a cavity, the third ventricle, while the pons and medulla share a cavity which reaches the surface at the upper medulla, where the roof is invaginated by the right and left choroid plexuses of the fourth ventricle.

The bulk of the CSF is produced by the choroid plexuses of the lateral ventricles, each opening into the third ventricle by the interventricular foramen of Monroe. From the third ventricle the aqueduct of Sylvius opens below into the fourth ventricle. Below this the central canal extends as a tiny tube through the spinal cord into the upper end of the filum terminale. The only apertures in this system, the foramina of Magendie and Luschka, lie in the roof of the fourth ventricle, whence the CSF escapes into the subarachnoid space [6,7].

The studies of Dandy (1919) provided the basis of our knowledge of CSF formation, circulation, and absorption [8].

PATHOPHYSIOLOGY

Hydrocephalus is more commonly due to a decrease in the absorption of CSF, although there are rare cases of choroid plexus papillomas causing hydrocephalus by an increase in CSF production [9]. Obstruction to the flow of CSF results in expansion of that part of the pathway, which lies proximal to the obstruction. Thus, if the obstruction is in the third ventricle, both lateral ventricles enlarge symmetrically; if at the exit foramina in the fourth ventricle, the entire ventricular system enlarges. Obstruction at any of the above sites results in non-communicating hydrocephalus. By contrast, when the obstruction is in the subarachnoid space at the base of the brain, the entire system again enlarges but hydrocephalus is of the communicating type [10].

It is common to divide obstructive hydrocephalus into congenital and acquired types as depicted in the table below [11]

Congenital	Acquired
Arnold-Chiari malformation	Infectious meningitis
Dandy-Walker malformation	Infectious ventriculitis
Aqueductal atresia/stenosis	Late-onset aqueductal stenosis
Development cyst	Intraventricular hemorrhage
Encephalocele	Subarachnoid hemorrhage
Neoplasm	Neoplasm

Arnold-Chiari malformations, one of the commoner causes of congenital hydrocephalus, consist of tongue-like prolongation of the inferior cerebellar vermis through the foramen magnum. The lower part of the fourth ventricle lies in the upper part of the vertebral canal and the foramen magnum is blocked by the displaced tissue from the posterior fossa. This leads to gross enlargement of the ventricular system. A meningomyelocoele is an almost invariable accompaniment of the Arnold-Chiari malformation [10].

Other relatively common congenital abnormalities causing hydrocephalus are faulty development of the aqueduct and atresia of the foramina of Luschka and Magendie. Aqueduct stenosis is a cause of infantile hydrocephalus and may give rise to increased intracranial pressure for the first time in adult life [12].

Congenital hydrocephalus may also occur in the absence of any apparent developmental malformation. One example is hydrocephalus following intra-uterine meningitis or ventriculitis due to toxoplasmosis. Hydrocephalus may also occur following neonatal meningitis or subarachnoid hemorrhage from cerebral birth injury.

Acquired hydrocephalus can result from obstruction of arachnoid villi by inflammatory, neoplastic, or leukeamic cells in infective or neoplastic meningitis. Obstruction of the subarachnoid space and the arachnoid villi by blood accounts for the hydrocephalus seen with subarachnoid haemorrhage and head injury [13].

A previous study by MacNab and Cohen reviewed 200 cases of non-tumour hydrocephalus and found that 18 per cent were due to aqueduct block, 42 per cent to cistern block, and 40 per cent were associated with an Arnold-Chiari malformation [14].

TREATMENT MODALITIES AND SURGERY

Hippocrates knew that in congenital hydrocephalus, the child had 'water on the brain' [15]. Hence he attempted treatment by ventricular puncture. Subsequently, many surgeons practiced intermittent ventricular tapping or established permanent ventricular drainage into the subaponeurotic space using catheters, trochers, or needles. A high rate of infection bedeviled those efforts.

The application of compression head wrapping in the treatment of congenital hydrocephalus was first suggested in 1823. This method of treatment has recently been re-introduced by Epstein and others [16], using an elastic bandage or silastic helmet with an inflatable inner lining.

Different types of treatment, which have been tried to treat hydrocephalus surgically, can be classified into (a) the surgical reduction of CSF formation; (b) bypassing the site of CSF obstruction intracranially; (c) diverting the CSF into another body compartment for excretion or absorption [17,18.19,20,21,22,]. Currently diversion of CSF into another body compartment is the method of choice.

The earliest major successful advances in CSF shunting were made at the turn of the 20th century when Gartner, in 1895, suggested that hydrocephalus could be treated by connecting the ventricle with either the venous or lymphatic system of the head and neck [23]. As a result of this idea, Ferguson in 1898 diverted CSF into the peritoneal cavity through a spino-peritoneal system, and in 1905 Kausch performed a ventriculo-peritoneal shunt [3].

An important advance was made by Dandy and Blackfan [8,24] who through experimentation showed that CSF comes from the choroid plexus. Dandy recommended destruction of choroid plexus for communicating hydrocephalus and third ventriculostomy for non-communicating disease. Torkildsen created a shunt between the lateral ventricles and the cisterna magna to treat aqueductal stenosis [25].

Modern shunting procedures, one of the earliest of which was the ventriculo-cardiac shunt described by Nulsen and Spitz [26], are now well established for diverting CSF from the ventricles into an appropriate receiving part of the body for the relief of hydrocephalus. The fluid has been shunted into the heart, pleura, peritoneum; into epithelialised ducts like the gall bladder, fallopian tube, ileum, thoracic duct and the ureter [27].

The earliest of the devices used for shunting is the Spitz-Holter valve, which was invented by John Holter in 1956 to save his son [28]. Other valves have since been introduced such as Ames, Cordis-Hakim, Denver, Farr, Holter-Hausner, Macpherson, Pudenz-Heyer, Raimondi, Till-Dahl-Wade, and the Indian valves of Upadhyaya and Chabbra.

In 1992, as a result of shortage of conventional shunts Adeloye [29] devised the local Malawi shunt made from siliconised rubber tubing and right angled metal connector between its ventricular and peritoneal parts.

Advances in materials and shunt design have led to significant advances in the treatment of hydrocephalus. Prior to 1954, when a Silastic shunt was first used, [30] rubber, Portex vinyl, and polyethylene tubing were used [31,32]. These materials stimulated foreign body reactions and led to a relatively high incidence of distal tip obstruction [33]. Kinking and breakage of the tubing also caused distal obstruction.

Silastic tubing was first used in 1955 for ventriculoatrial (VA) shunts [30]. Mazza et al reported that 45 percent of their patients with VA shunts had one or more revisions, compared with 51 percent of those with VP shunts [34]. Although complications in the VA group were less frequent, they are more severe; the absolute mortality rate for shunt related complications was 14 percent for VA as compared with 7 percent for VP shunts. In other studies VA shunts were associated with more frequent revisions (ratio of revisions of VA shunts versus VP shunts of 1.46:1.02 before the age of 5 years). Patients also had more severe late complications with VA shunts [35,36]. The relative ease of VP shunt insertion, the less severe complications, and the ease of revision all are reasons that the peritoneal cavity is the receptacle of choice for CSF diversion [37,38,39].

Ventriculostomy is done via a parietal skin flap and the distal catheter is passed subcutaneously. Passage of the shunt tubing is facilitated by using a

nondisposable shunt passer, which has the advantage that it can be bent to ease over the clavicle. The abdominal portion of the catheter is placed intraperitoneally via a minilaparatomy. Recent studies indicate that laparascopic assisted distal VP shunt placement is a good alternative to the open technique [40,41,42].

COMPLICATIONS OF SHUNT INSERTION-CLINICAL FEATURES AND DIAGNOSIS

Various complications of VP shunting are encountered. Some complications occur more frequently than others.

Commonly encountered complications:
1) SHUNT INFECTION

Shunt infection rates vary from 2.5 to 40 percent, [43,44,45] with most series reporting a 10 to 20 percent infection rate. Mwangombe and Omulo [4], at KNH showed an infection rate of 24.6 percent. Quigley [46] working mainly with VP shunts reported an infection rate of 6.9%. O'Brien [47] reported a rate of 2.7%, while Renier [48], Lambert [49], and Fitzgerald [50] reported rates of 7.9, 13.2, and 2.4 percent respectively.

It is now agreed that most shunt infections occur as a result of organisms gaining access to the shunt, during surgery, from the patient's own flora [51,52]. It has also been shown that the most common organisms in shunt infection are coagulase negative staphylococci [44,46,48,51,52]. These

organisms have the capability to colonize shunts due to an extracellular mucoid substance (slime), which enables them to form microcolonies adherent to the luminal surface of the silicone rubber shunt catheters [51,53]. Other members of the skin flora have also been encountered in shunt infections although they do not have this ability to adhere to and colonize shunt surfaces. In a study by Davis et al [54], type of infecting organism was divided roughly in three groups, with relatively equal representation from Staphylococcus epidermidis/coagulase negative and Staphylococcus aureus. The remaining third group was comprised of a wide variety of organisms

Age, aetiology of hydrocephalus, concurrent infections, and previous surgical procedures have been mentioned as some of the factors associated with increased risk of developing shunt infections. Premature infants have been seen to have a particularly high risk of developing shunt infections [55,56]. However a recent study by Davis et al, which reviewed 2,325 VP shunting procedures, showed an infection rate of 3.2 percent. There was no statistically significant difference between age groups (p>0.05). In the same study aetiology of hydrocephalus was not a factor, nor was the presence of an open neural tube defect. The presence of fluid accumulation along the shunt tract or at another neurological operative site was associated with a significant increase in incidence of infection (8.9%) when compared to those with no fluid accumulation (p<0.001)[54].

Presence of other concurrent infections elsewhere in the body before or at the time of shunt surgery has also raised concern as a risk factor in shunt

infection. Renier et al [48] found a positive correlation between shunt infection and other intercurrent seats of infection outside the central nervous system for example ear, nose, and throat infections. He also found higher rates of infections in patients who had poor skin conditions e. g dermatitis, post-operative wound dehiscence and scalp necrosis.

Other studies however found that concurrent infections do not pose any additional risk of developing shunt infection [46,57,58,59].

The number and nature of previous surgical procedures have been considered in relation to risk of developing shunt-associated sepsis. Renier [48], in his study on 1174 shunt operations found an increased risk in relation to the number of previous procedures performed on the shunt. George [60] also reported a similar finding.

Other factors have been considered but have not been found to correlate with shunt infection. These are length of surgical procedure [59], type of valve used [58,59], emergency versus non-emergency surgery [61].

Internalization of an external ventricular drain has been found to be consistently associated with an increased risk of shunt infections in most reports [46,48,62,63].

The relationship between the experience of the operating surgeon and risk of sepsis has also been addressed in some studies. Although many reports have shown no differences in infection rates between experienced and

inexperienced surgeons (e. g consultant neurosurgeons and registrars) [48,59], there are also reports which show inexperience of the operating surgeon as a risk factor in shunt infection [55,60]. George et al [60] actually found a twenty-five fold difference in infection rate between experienced and inexperienced surgeons. Some have argued that these isolated apparent relations between surgeons and infections could be a reflection of some individual proneness to have high infection rates [46].

There have been attempts to classify CSF shunt infections into internal or external infections [46,50]. External infections are those that are associated with wound infection, cutaneous infection with signs of inflammation along the shunt course or breakdown of the skin exposing the shunt mechanism [47].

Internal infection is considered to occur when there is colonization of the shunt mechanism with possible features of ventriculitis, meningitis, or peritonitis without features of skin infection [47].

Most studies confirm the tendency to have shunt infections presenting in the first few months following surgery [44,60]. In Schoenbaum's series [44], 70 percent of the infections presented within the first two months while 78 percent presented within the first four months. In Quigley's study [46], the time interval from surgery to infection averaged 21 days, while in George's study 66 percent of the infections presented within one month of surgery [60].

Fever has been reported by several workers as the most common clinical manifestation in shunt infection [44,46,47]. VP shunt malfunction, which has been shown in many reports to be a sign of shunt infection [51,52], is reported as another major mode of presentation in shunt infection [46,47,51]. The signs of malfunction associated with sepsis include headache, seizures, bulging or tense fontanelles, excessive rate of head growth, backing up of CSF along shunt track and malaise [44].

Local features of shunt infection which may be encountered and have been reported include wound sepsis [44,60], features of inflammation or cellulitis along the shunt tract (redness and tenderness) [44,47,57], and wound breakdown with exposure of shunt system [47].

Features of ventriculitis, meningitis, or peritonitis with general ill health may also be encountered.

Diagnosis of shunt infection is usually suspected from the history and physical examination, however laboratory evidence may be useful.
Increased white blood cell counts are expected in shunt infections. Schoenbaum [44] found leukocytosis of more than 20,000 / dl in 32 percent of his infected shunt patients.

CSF microscopy and biochemistry have also been mentioned as aids in diagnosis of shunt infection [52]. CSF leucocytosis of more than 10 per mm^3 is considered diagnostic of ventriculitis in shunt infection [46], while reduced CSF sugars and increased protein are suggestive of shunt infection [52]. Culture of CSF and isolation of the causative agent is a confirmatory

diagnosis. Tapping or aspiration of VP shunt systems has been used by many with varying isolation rates in cases of shunt infection [44,46]. In a recent study [64], incidence of VP shunt meningitis was 6.3%. The most common organisms isolated from CSF and shunt were coagulase-negative Staphylococci (52.8%), followed by Staphylococcus aureus (13.1%) and Pseudomonas aeruginosa (7.5%) [64].

2.MECHANICAL MALFUCTION/BLOCKAGE

Another common complication of VP shunts is blockage of the shunt system. This occurs more commonly at the distal (peritoneal) end than at the proximal (ventricular) end. In a recent study by Miyake et al [65], the most common cause of shunt complications was mechanical trouble with the abdominal catheter. Gichuhi [5] found malfunction of the shunt system as the commonest complication (32.8%). In his study he found equal proportion of proximal and distal blockage. In another study [66] of 128 consecutive patients with symptoms of shunt malfunction, 50% had proximal malfunction, 14% distal, and 10% had malfunctions attributable directly to the valve itself. In another study [67], disconnection in the multicomponent shunt system (Holter), accounted for 15% of the malfunction. The more distal the connection was from the ventricle, the higher the likelihood of disconnection. Furthermore, occipitally placed shunts had a significantly higher tendency to dislocate than frontally placed shunts.

The clinical manifestations of shunt malfunction include headache, irritability, vomiting, seizures, bulging or tense fontanels, increase in head circumference, and backing up of CSF along shunt track. It has been suggested that early (within 3 months) presentation of shunt malfunction is associated with shunt infection. This is thought to occur due to formation of local adhesions and fibrinous deposits and often encystment by the greater omentum due to discharge of microorganisms into the peritoneal cavity by the colonized shunt [51].

Diagnosis is usually clinical. Shunt obstruction may be confirmed with radioisotope examination or with fluoroscopically guided injection of iodinated contrast material into the shunt reservoir [68].

3.SHUNT MIGRATION AND PERFORATION

Migration of VP shunt system has been mentioned in many studies. Gichuhi [5] noted a shunt migration rate of 5.2% in his study. The catheter tip can erode through the bowel, causing presentation with catheter per rectum, ventriculitis, or peritonitis. Wilson and Bertan [69] first reported this complication in 1966. In 1973 Sells and Loeser [70] reported a case of transrectal passage of a catheter tip associated with peritonitis, shunt malfunction, and ventriculitis. Perforation of other organs has been reported [71], including the vagina [72], the bladder [73], the gallbladder [74], and the liver [74].

Just as the catheter can perforate abdominal organs, it can also perforate the abdominal wall through the umbilicus [29].

Panagea et al [75] reported two cases of VP shunt infection attributable to intestinal perforation. One patient developed a brain abscess, the other ventriculitis. Microbiology consisted of fecal flora and the peritoneal catheter was found to be fecally stained in both cases. There were no abdominal signs or symptoms. It is thought that infection developed via the ascending route. Silent bowel perforation and transanal prolapse has also been reported [76]. Nakano et al [77] reported CSF leakage from the nipple after VP shunt insertion. In a recent case report a 3-year-old boy with hydrocephalus was observed to have a painless 2.0cm right scrotal mass. Abdominal radiograph showed VP shunt tubing in the right scrotal sac. Removal of a detached shunt catheter and inguinal hernia repair resolved the problem [78]. A similar case has also been reported by Ozveren et al [79]. Diagnosis is usually clinical or by help of radiography.

Other uncommon complications encountered:

1.PNEUMOCEPHALUS

Pneumocephalus secondary to colonic perforation by VP shunt catheter has been reported [80]. The patient presented with features of meningitis and CT-scan done showed an air-fluid level within both lateral ventricles, raising the possibility of colonic perforation since no other aetiology for the pneumocephalus could be found.

2.SLIT VENTRICLES

The patient with a shunt who presents with collapsed or "slit-like" ventricles can present a clinical challenge. The patient can suffer from low-pressure symptoms (e. g headaches and/or vomiting) that improve with supine posture and worsen with upright posture. This can happen in the setting of a disconnected distal shunt valve. It is suggested that the presence of patent fibrous tract allowed the overdrainage of CSF [81].

3.SHUNT-RELATED METASTASIS

Extraneural metastasis is an unusual complication of CSF diversion, but spread of tumor cells into either the vascular system or the peritoneal cavity via a shunt. A recent review by Rickert [82] revealed 35 VP shunt-related abdominal metastasis from pediatric brain tumours. The male to female ratio was 1.9:1. The four most common sources of metastasis were germinomas, medulloblastomas, endodermal tumours, and astrocytomas. To avoid this complication it is advisable to primarily excise pediatric posterior-fossa tumours and leave an external ventricular drain.

4.CSF ASCITES

This rare complication has been reported in two children in India by Chidambaram et al [83]. No definite explanation has been offered for the inability of the peritoneum to absorb the CSF. The children presented with abdominal swelling and ascites.

5.ABDOMINAL CSF PSEUDOCYST

Abdominal CSF pseudocyst was first reported in 1954 by Harsh [84] and has since been recognized by others and variably described as omental cyst. Pathological examination of the cyst shows thickened fibrous tissue with chronic inflammatory cells on the inner wall, the outer wall being formed by the matted bowel wall Infection is implicated in the etiology of pseudocysts

Several recent studies have reported CSF pseudocysts as complications of VP shunting [85,86,87]. Diagnosis is usually made by ultrasound of the abdomen. It is agreed that predisposing factors for pseudocyst formation are multiple shunt revisions and infection [86].

6.OTHERS

Many other infrequent complications have been reported in the literature. Some of these are as follows:

(i)   Expanding septum pellucidum cyst [88].

(ii)   Spontaneous Bacterial Peritonitis [89].

(iii)   Hydrothorax [90].

(iv)   Intrahepatic cyst [91].

(v)   Epidural haematomas [92].

(vi)   Intraparenchymal pericatheter cyst [93].

(vii)   Spread of Tuberculous Meningitis into multiple disseminated abscesses [94].

MANAGEMENT OF SHUNT COMPLICATIONS

Making a correct diagnosis is of utmost importance in management of shunt complications. Radiographic studies are very helpful in confirming the right diagnosis [68]. Early intervention reduces mortality.

Shunt infections may be treated by removal of the shunt and insertion of an external ventricular drain [46,47]. CSF and shunt cultures are obtained, as well as daily samples of CSF from the external drain. Appropriate intravenous antibiotics are administered according to organism sensitivity. There is some evidence that aggressive, multiple antibiotic administration might reduce need for complete shunt removal [95]. The child is maintained on external ventricular drainage until CSF cultures have been negative for 7-10 days. Antibiotics are then stopped for 48 hours. If the CSF remains sterile, the external drain is removed and a new shunt is placed. Intrathecal antibiotics are occasionally given through the external ventricular drain.

Bayston [51] argues that antibiotic treatment with infected shunt in situ is associated with low success rates as coagulase negative Staphylococci which are the usual organisms have been known to produce protective slime [96,97]. He therefore recommends a treatment schedule that involves removal of the infected shunt, external ventricular drainage in addition to intravenous and intraventricular antibiotics. This is followed by reshunting after 5-10 days of treatment. Many workers, as the standard practice, have accepted removal of the infected shunt [44,46,47,51,60,97], due to evidence that infection will not clear until the foreign body is removed. During the

phase of external drainage close supervision and fluid, electrolyte, and protein monitoring is of utmost importance.

It is thought that prophylactic antibiotics and improved surgical techniques play a role in reducing the incidence of shunt infection.

Management of shunt malfunction, if associated with infection, is handled in the same manner as described above for shunt infection. If there is no associated infection, the patient is taken to theatre and the shunt externalized. If the blockage can be overcome, the same shunt is again internalized, otherwise a new shunt is inserted. Minimally invasive recanalization of obstructed ventricular catheters is a new treatment modality currently under investigation. It has been shown that blocked ventricular catheters can be at least partly reopened with ultrasonic cavitation, fibreoptic delivery of laser energy and electrocautery [98].

Laparascopy has been used to retrieve disconnected distal portions of VP shunts. Tanaka et al [99] reports two cases where the distal catheters were disconnected and lying free in the peritoneal cavity. Laparascopic retrieval and revision of the distal portion was done with good success.

Migrated and perforated shunt systems have to be removed, any associated infection treated and later a new shunt inserted.

The treatment of choice in CSF ascites is conversion of the VP shunt to a ventriculoatrial shunt [83].

In cases of pneumocephalus treatment is temporarily draining the CSF externally across a small pressure gradient. Persistent fistula may require craniotomy and direct repair.

Treatment of abdominal CSF pseudocyst consists of removal of the VP shunt and placement of an external ventricular drain. Antibiotics are administered for 10 days. The cysts can be aspirated under ultrasound guidance or can be left alone for spontaneous resolution [87]. A VP shunt can be safely re-inserted.

Slit ventricles are treated by subtemporal craniectomy to allow for improved ventricular expansion.

The reported revision rate of VP shunts differs very much, but many papers report it close to 50 percent. Commonest causes are defect or obstruction in the ventricular catheter, followed by defect or obstruction in the distal catheter, displacement of the distal catheter and acute infection [100]. Iskander et al [101] in their study found a 4% mortality in the first 32 months after the first shunting and concluded that despite modern technology a number of hydrocephalic children still die of shunt failure.

OBJECTIVES

Main Objective:

To outline the clinical presentations, and rates of complications of post-VP shunt insertion.

Specific Objectives:

1. To determine the common causes of non-tumour hydrocephalus needing VP shunt placement at KNH.

2. To determine the clinical features and rate of shunt infection.

3. To determine the clinical features and rates of any other types of shunt complications.

4. To determine the time interval between onset of hydrocephalus and shunt insertion.

5. To determine any factors which might be related to the rate of shunt complications, and give suggestions on ways of prevention.

6. To review management of shunt complications at Kenyatta National Hospital and to give suggestions on ways of reducing morbidity and mortality.

MATERIALS AND METHODS

Study design

This is a six-month descriptive prospective study from 1ST September 2001 to 28TH February 2002. The study included all patients with non-tumour hydrocephalus undergoing their first VP shunt insertion by the neurosurgical team at the Kenyatta National Hospital. All patients with the above diagnosis were recruited in the first three-month period of the study and followed up for a further three months after surgery.

Patients were randomly chosen to receive an intravenous antibiotic at induction of anaesthesia as a prophylaxis. All patients recruited in the first two months did not receive the antibiotic, while all patients in the third month of recruitment received the antibiotic. The antibiotic used was FEXAL (Ceftriaxone) 250mg, because of affordability and broad-spectrum cover. Apart from the prophylactic antibiotic, other variables studied included time interval between diagnosis and shunt insertion, status of surgeon, type of shunt used, type of hydrocephalus, presence of dysraphism, and time taken for surgery.

All the shunts were dipped and cleansed in gentamicin solution before insertion. VP shunt surgery was performed in the conventional manner after appropriate cleaning and draping technique (see literature review pages 8-11)

Study Area

The study was conducted at Kenyatta National Hospital's Neurosurgical and Paediatric wards.

Sample size

All the patients with a diagnosis of non-tumour hydrocephalus within the three-month recruitment period were included. A rough estimate from theatre records had shown twenty cases per month giving a sample size of sixty, but a total of 76 patients were recruited in the study.

Inclusion Criteria

All patients with non-tumour hydrocephalus undergoing their first V-P shunt insertion.

Exclusion Criteria

All patients with hydrocephalus due to an intra-cranial space-occupying lesion.

Data collection

Data collection was done by the researcher on the pre-designed data collection form (Appendix 1). After getting an informed consent from the parent all pre-operative data was filled. Intra-operative data was collected in the operating room. Post-operatively each patient was seen after 48 hours and then followed up at the surgical outpatient clinic (SOPC) at 2 week and 3 month intervals and the data sheets completed. In case of any shunt complication the patient was re-admitted and managed.

The final outcome of patients managed for shunt complication was based on the duration of hospital stay, which was used to gauge how fast the complication had resolved with management. Thus:

GOOD- improved on treatment and discharged within 7-10 days

FAIR-improved on treatment and discharged within 10-21 days

POOR-patient still on treatment after 30 days

DEAD-patients who died while being managed for shunt complication during the study period but not in the immediate post-operative period.

Data management and presentation

All the data obtained was transferred from the data collection form onto a coded sheet for computer analysis. This was done using the SPSS statistical package. The results were presented in graphical, tabular and chart forms. Statistical significance was determined using the Chi-square test and a p value of < 0.05 was considered significant.

Ethical considerations and patient consent

The research proposal was submitted to the hospital Ethical and Research Committee for approval before embarking on the study. All patients were recruited on informed consent basis provided to their parents or guardians who were required to sign a consent form. All information has been treated in confidence and has not been made public in any form.

RESULTS

Seventy-six patients underwent shunt surgery for non-tumour hydrocephalus at the Kenyatta National Hospital during the three-month recruitment period. Twenty-three of the seventy-six patients developed V-P shunt associated complications in the subsequent three month follow up period resulting in a complication rate of 30.3%.

1. Sex distribution

There were forty-six males (60.5%) and thirty females (39.5%) giving a male to female ratio of 1.5: 1. (Fig 1)

**Figure 1-Sex distribution in patients with non-tumour hydrocephalus**

Female 39.50%

Male 60.50%
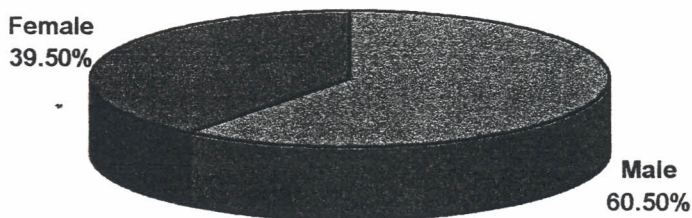
2.Age profile and presence/absence of complications

The ages of patients who underwent VP shunt surgery ranged from one month to 54 years. Seventy-one point one percent (71.1%) of the patients were below the age of one year and more than half of these were below six months of age. Four patients were above thirteen years of age and had developed hydrocephalus following head trauma (Table 1, Figure 2). It was also noted that patients between 3-24 months had a slightly lower rate of complication than those below 2 months of age (Table 1).

Table 1-Age profile and presence/absence of complication

Age category (months)	No of patients with complications (%)	No of patients without complications (%)	Total no of patients
< 2	5 (41.7)	7 (58.3)	12
3-4	4 (28.6)	10 (71.4)	14
5-6	3 (33.3)	6 (66.7)	9
7-8	2 (22.2)	7 (77.8)	9
9-10	1 (20.0)	4 (80.0)	5
11-12	1 (20.0)	4 (80.0)	5
13-24	1 (20.0)	4 (80.0)	5
25-36	3 (60.0)	2 (40.0)	5
37-60	2 (33.3)	4 (66.7)	6
> 60	1 (16.7)	5 (83.3)	6
Total	23(30.3)	53(69.7)	76

Figure 2-Age profile and presence/absence of complications

Legend:
- No of patients with complications%
- No of patients without complications%
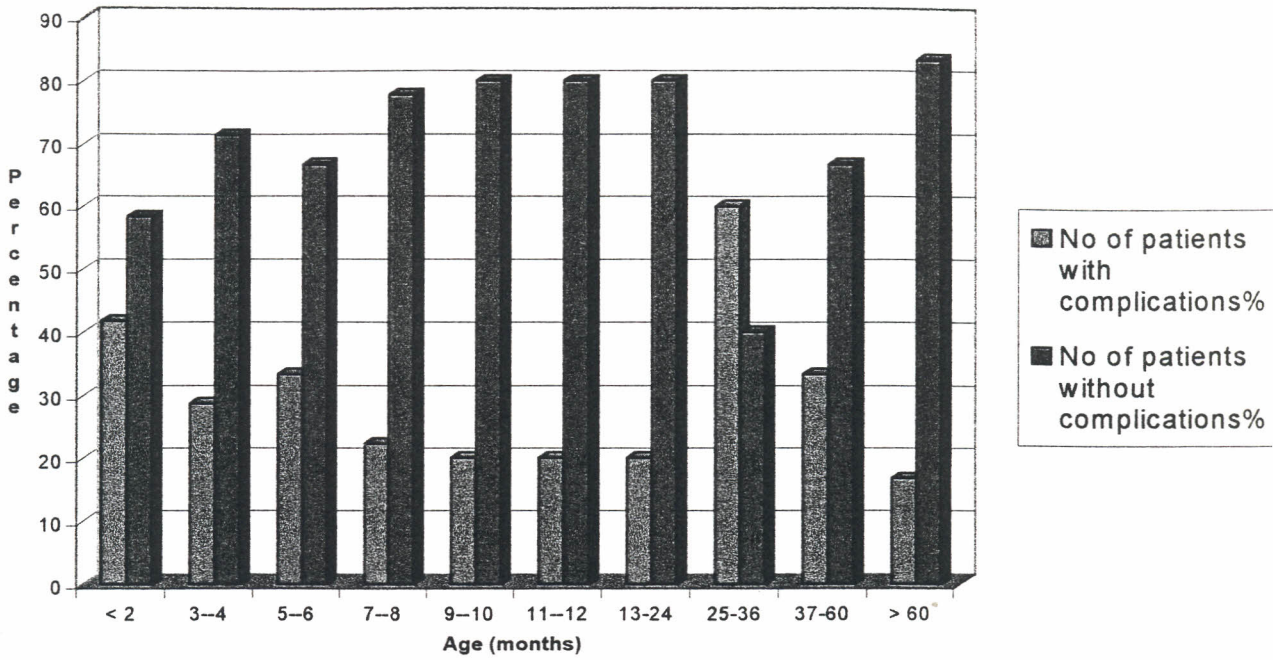
3.Causes of non-tumour hydrocephalus

The commonest cause of hydrocephalus seen in patients in this study was congenital (72.4%). Most of these were unspecified congenital causes, since the patients could not afford to pay for ultrasonography or CT-Scans. Aqueductal stenosis was found to be the cause in 18.4%. Only one patient had obvious features of Arnold Chiari malformation.

Of the acquired type (27.6%) post-meningitic accounted for the majority of the cases. Three cases of hydrocephalus were as a result of trauma and subsequent sub-arachnoid haemorrhage. [Table2, Figure 3].

Table-2 Causes of non-tumour hydrocephalus (n=76)

Type of hydrocephalus	Cause	Total no of patients	%
Acquired	Post meningitic	18	23.7
	Post traumatic	3	3.9
	Total	**21**	**27.6**
Congenital	Aqueductal stenosis	14	18.4
	Arnold Chiari	1	1.3
	Unspecified congenital	40	52.7
	Total	**55**	**72.4**
Total		**76**	**100**

Figure 3 - Causes of non-tumour hydrocephalus (n=76)

Legend:
- POST MENINGITIC
- POST TRAUMATIC
- AQUEDUCTAL STENOSIS
- ARNOLD CHIARI
- UNSPECIFIED CONGENITAL

Values shown on chart: 18, 3, 14, 1, 40
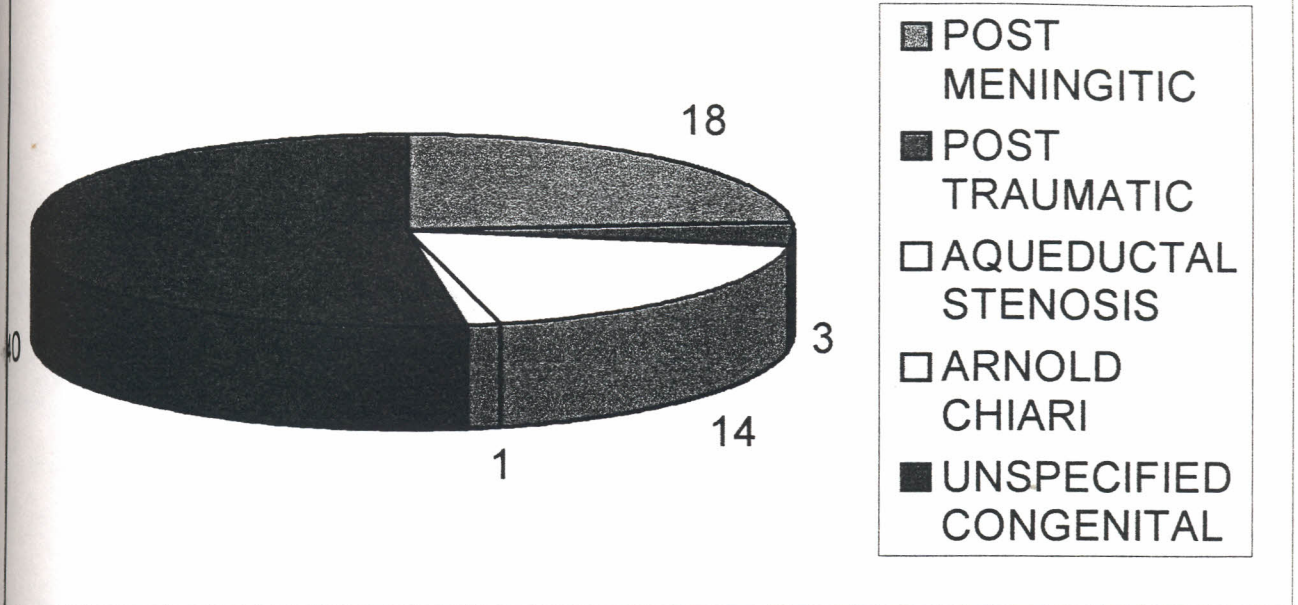
4.Time interval between onset of hydrocephalus and first v-p shunt insertion

Generally there was a considerable delay from the time the patient was noted to be having features of hydrocephalus to the time the patient underwent insertion of the first V-P shunt. This time interval was considered and it was noticed that twenty-eight patients (36.8%) underwent VP shunt surgery within two months of diagnosis and 42.9% developed shunt complication. Twenty-five patients (32.9%) underwent VP shunt surgery between 2-4 months from the time of diagnosis and 25.0% developed shunt complication. Comparing the complications in the below 2-month interval group with the rest did not show a significant reduction in the rate of shunt complication ($p=0.0698$). A longer time interval was not associated with an increase in rate of shunt complication. The majority of cases (85.5%) had their shunts inserted within six months from the onset of hydrocephalus [Table 3, Figure 4].

Table 3-Time interval between onset of hydrocephalus and first v-p shunt insertion versus complication

Interval (months)	Patients with complications (%)	Patients without complications (%)	Total no of patients
< 2	12(42.9)	16 (57.1)	28(36.8)
2-4	5 (25.0)	20 (75.0)	25(32.9)
4-6	3 (25.0)	9 (75.0)	12(15.8)
7-12	1 (20.0)	4 (80.0)	5(6.6)
> 12	2 (33.3)	4 (66.7)	6(7.9)
total	23 (30.3)	53 (69.7)	76(100)

Figure 4: Time interval between onset of hydrocephalus and first V-P shunt surgery versus complication
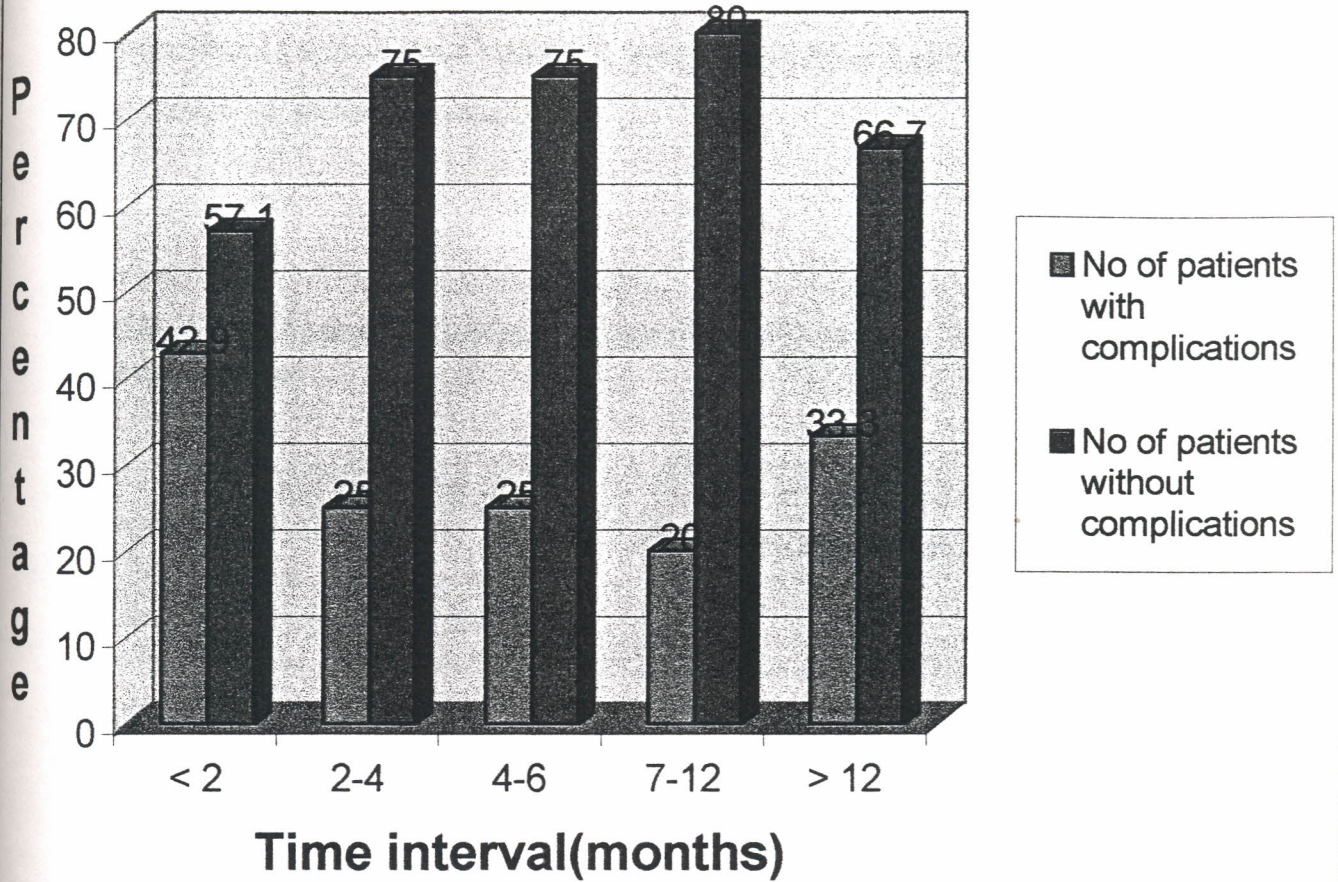
5.Time interval between shunt insertion and onset of complications

Seventeen of the patients with shunt complications presented within one month of shunt insertion, out of which twelve (12) had shunt infection, four (4) had shunt blockage, and one (1) shunt migration [Table 4]. It is important to note that 17 out of the 23 patients with complications presented within one month of shunt insertion (74%).

Table 4–Time interval between shunt insertion and onset of complications

Time interval (weeks)	Episodes of complications
0-2	9
2 -4	8
4-8	5
>8	1
Total	**23**

6.Complications of v-p shunt surgery

The rate of development of complication in this study was 30.3% during the three- month follow up period. Shunt infection accounted for 65.2% of the complications. Diagnosis of shunt infection was mainly clinical, however few patients had a laboratory diagnosis reflected by high WBC counts and positive CSF cultures.

Shunt malfunction/blockage accounted for 30.5% of the total complications. Diagnosis was clinical in all cases.

One patient presented with shunt prolapsing from the anus during defecation but was draining well and had neither signs nor symptoms of shunt infection or blockage.

One patient was readmitted because of a high fever and convulsions but was found to have bronchopneumonia and the shunt was functioning well. [Table 5, Figure5].

Table 5- Complications of v-p shunt insertion

Type of complication	Number of patients	Percentage(%)
Shunt infection	15	65.2
Shunt blockage	7	30.5
Shunt migration	1	4.3
Total	**23**	**100**

Figure 5- Complications of v-p shunt insertion
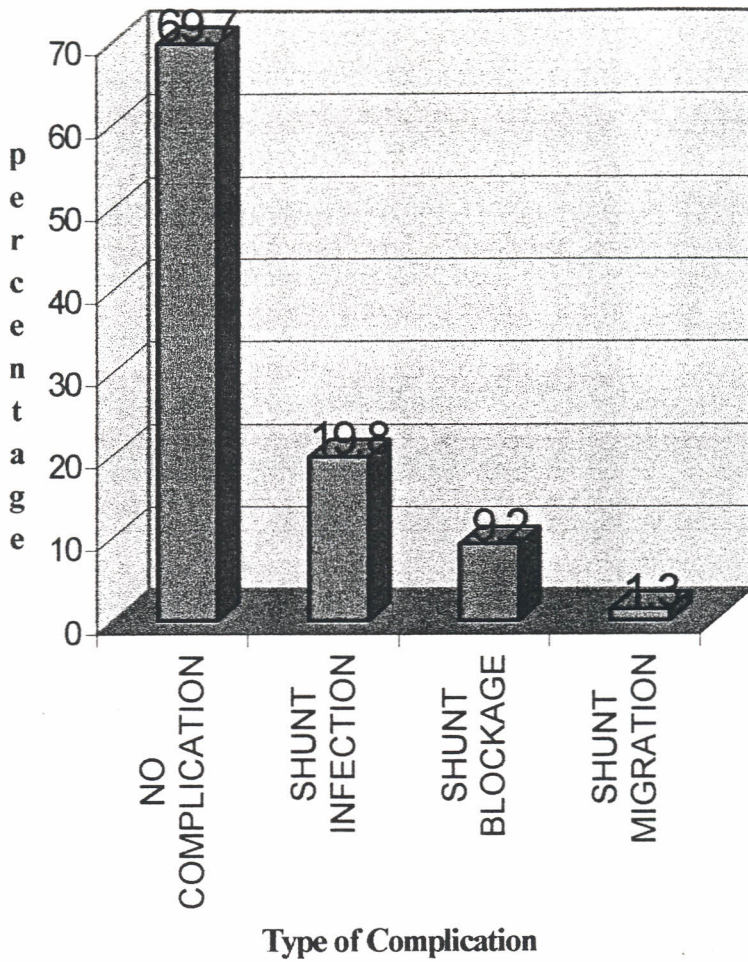
7. Treatment modalities used in the management of shunt complications

Out of 15 patients who developed shunt infection six (6) were treated conservatively with intra-venous antibiotics and improved. The remaining nine were managed by externalizing the shunt, covering with intra-venous antibiotics and later complete shunt revision in theatre.

All the patients with shunt blockage and migration were managed by complete shunt revision in theatre [Table 6]. Therefore a total of seventeen (17) patients had to undergo shunt revision in theatre.

Table 6- Treatment modalities used in the management of shunt complications

Procedure	Number of episodes
non surgical(improvement on antibiotics)	6
surgical(shunt re-inserted in theatre)	17
Total	23

8.Use of prophylactic antibiotic

Twenty-seven patients were randomly chosen to receive a stat dose of antibiotic with induction of anaesthesia. The antibiotic was 250 mg of a generic of Ceftriaxone. Out of 27 patients who received antibiotics 3 patients developed shunt infection. Out of 49 patients who did not receive antibiotics 12 developed shunt infection. It can be seen that out of the 15 patients who developed shunt infection, three (3) had received antibiotics while twelve (12) had not [Table 7, Figure 6]. Using the antibiotic in this study did not show any statistical significance in reducing shunt infection (p=0.160)

Table 7-Number of patients on prophylactic antibiotic who developed complication.

Use of prophylactic antibiotic	Patients with complications			Patients without compli-cations	Total no of patients	%
	infection	block	migration			
No	12	6	1	30	49	64
Yes	3	1	0	23	27	35
					76	10

Figure 6- Patients on prophylactic antibiotic who developed complication
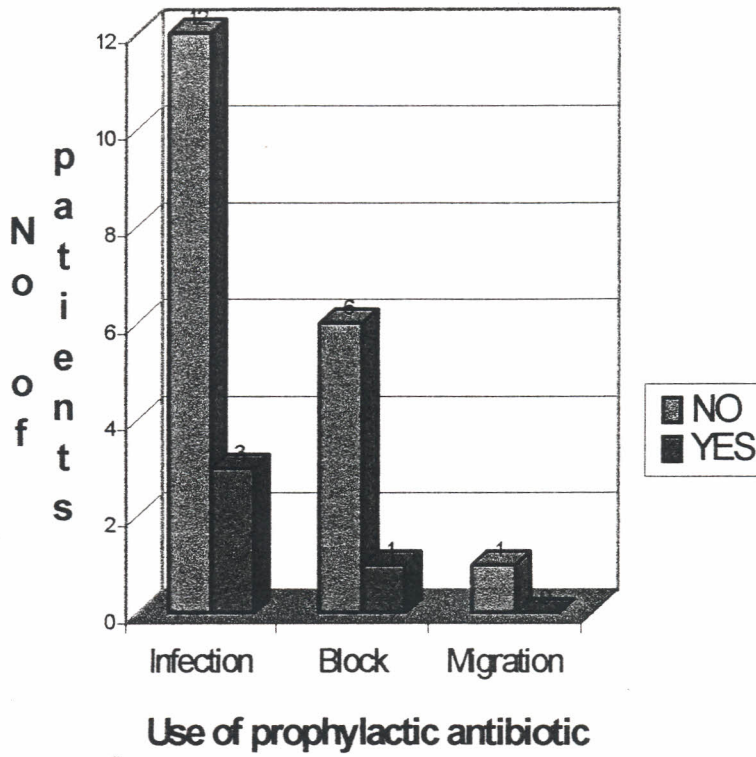
9.Signs and symptoms of shunt infection

The commonest signs and symptoms of shunt infection in this study were fever and irritability [Table8, Figure7].

Table 8 -Signs and symptoms of shunt infections

symptom/sign	number of episodes
fever	13
irritability	11
convulsions	6
vomiting	3
dehydration	4

Figure 7 -Signs and symptoms of shunt infections
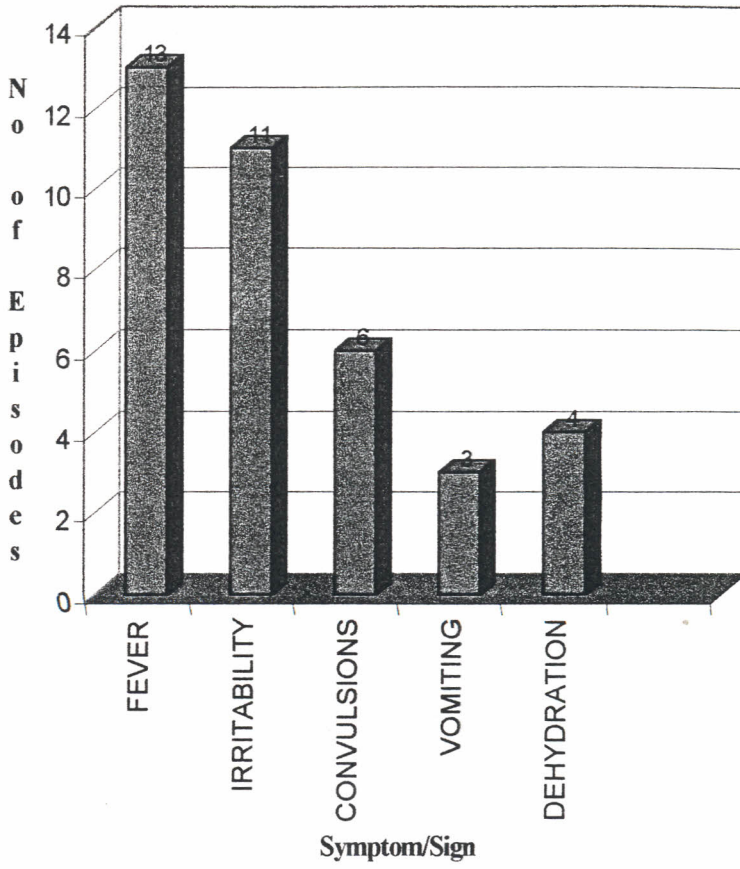
10.Presenting signs and symptoms of local shunt infection

There were two episodes of infection with features of shunt tract inflammation. This was as evidenced by presence of redness and/or tenderness along the shunt tract.

The shunt was found exposed in three cases but still in place in two while in one it had actually eroded out of the abdominal incision, and the distal tip hanging out.

There were four wounds out of which two were leaking CSF. [Table9, Figure8].

Table 9-Local features of shunt infection

presenting sign	number of episodes
sepsis/wounds	4
csf leak	2
exposed shunt	3
inflammation along shunt tract	2

44

Figure 8-Local features of shunt infection
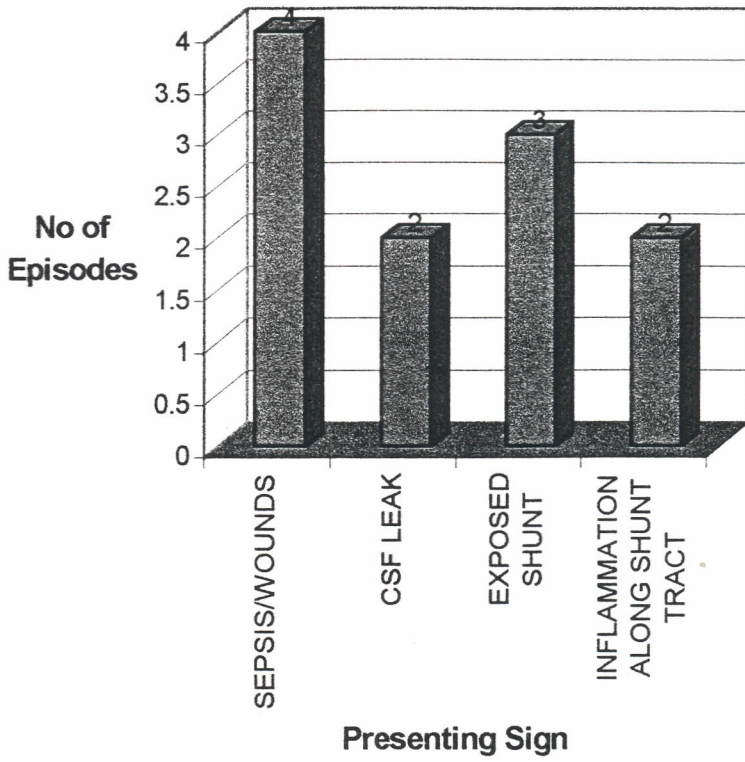
11.Features of shunt blockage

Signs and symptoms of shunt blockage or malfunction most commonly seen were vomiting and a tense bulging anterior fontanelle. Seven patients were found to have shunt blockage, which was confirmed intraoperatively during shunt revision. (Five of the patients with vomiting and a tense bulging anterior fontanelle were classified as shunt infection because of presence of fever).

Shunt malfunction/block was also confirmed by applying pressure on the shunt valve system behind the ear to confirm whether it would empty and fill easily. A significant delay in emptying and filling would indicate malfunction or block [Table 10, Figure9].

Table 10-Clinical features of shunt malfunction and block

Signs/symptoms	Number of episodes
Tense anterior fontanelle	12
Convulsions	2
Visual disturbance	1
Papilloedema	1
Headache	0
Vomiting	14
Shunt migration	1

Figure 9-Clinical features of shunt malfunction/block

Figure 9-Clinical features of shunt malfunction/block
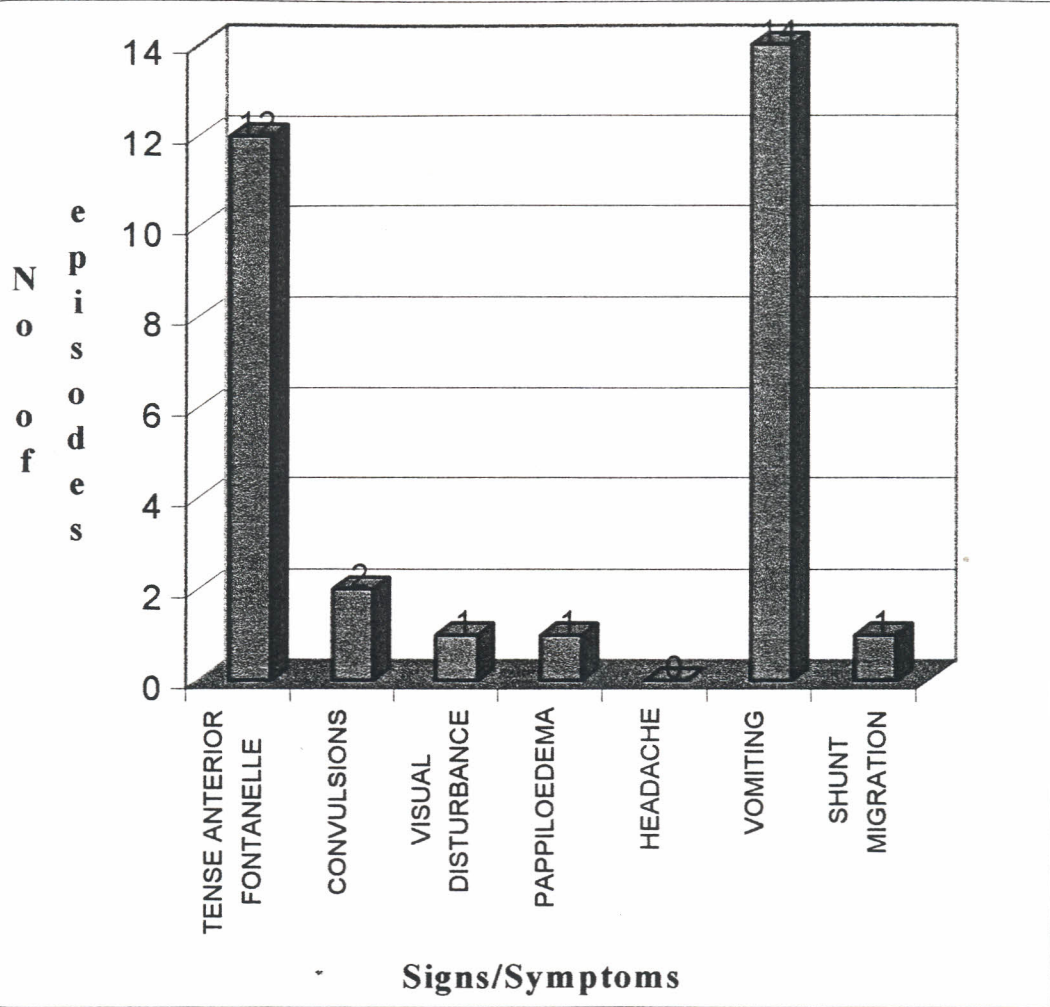
12.Other systemic manifestations associated with v-p shunt infection

Out of 15 cases of shunt infection, features of meningitis were seen in 6 episodes of infection (as evidenced by neck stiffness and or Kernig's positive sign).

Intestinal obstruction, ventriculitis, and peritonitis were each seen in one case [Table11, Figure 10]. This shows that systemic manifestations of shunt infections are common.

Table 11-Other systemic manifestations associated with v-p shunt infection

Complication	Number of episodes
Meningitis	6
Abdominal distention	1
Peritonitis	1
Intestinal obstruction	1
Ventriculitis	1

Figure 10-Other systemic manifestations associated with v-p shunt infection
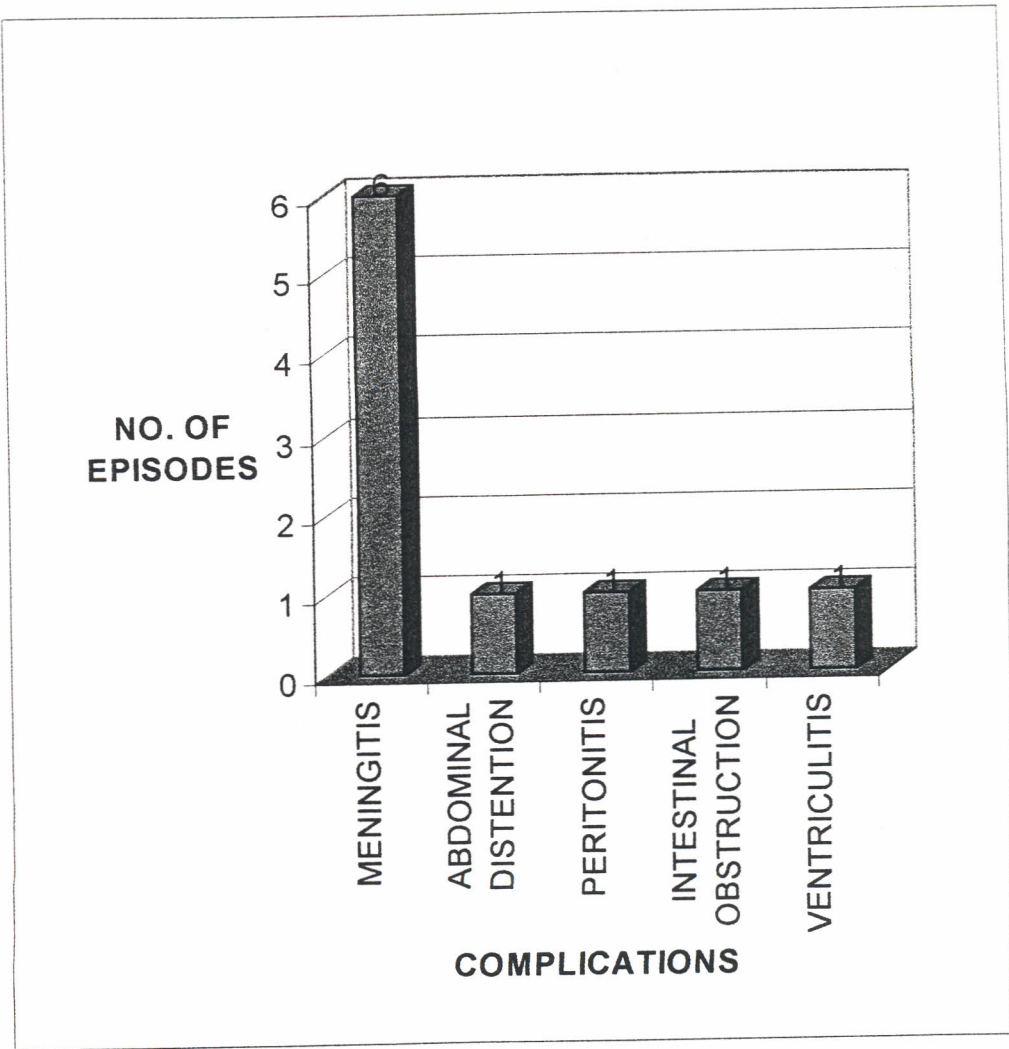
13.Causes of hydrocephalus in patients who developed shunt complications .

It was apparent from the study that 69.6% of the patients with shunt complications had congenital hydrocephalus while 30.4% had acquired (Table 12). Out of the 3 patients with post-traumatic hydrocephalus 2 developed shunt complication, while 5 out of the18 with post-meningitic hydrocephalus also developed complication (Table 12).

Seventy two percent of the patients seen in this study had congenital hydrocephalus while twenty eight percent had acquired hydrocephalus [Table 3, Figure 4]; seventy percent of the patients who developed complications following V-P shunt surgery had congenital form of hydrocephalus while thirty percent had the acquired form. Comparing shunt complication in the acquired and congenital groups was not of any statistical significance (p=0.710). In this study, the cause of hydrocephalus did not seem to influence the development of shunt related complications following surgery [Table 12, Figure 11].

Table12-Causes of hydrocephalus in patients who developed shunt complications (n=23)

Type of hydrocephalus	Specific	Episodes of complication	Percentage
Acquired	Post meningitic	5	21.7
	Post traumatic	2	8.7
Acquired total		**7**	**30.4**
Congenital	Aqueductal stenosis	4	17.4
	Arnold Chiari	1	4.3
	Unspecified congenital	11	47.9
Congenital total		**16**	**69.6**
Total		**23**	**100**

Figure11-Causes of hydrocephalus in patients who developed shunt complication (n=23)

Legend:
- ■ POST MENINGITIC
- ▦ POST TRAUMATIC
- ☐ AQUEDUCTAL STENOSIS
- ■ ARNOLD CHIARI
- ▦ UNSPECIFIED CONGENITAL

X-axis: ACQUIRED, CONGENITAL

Y-axis: PERCENTAGE

Data labels: 21.7, 8.7, 17.4, 4.3, 47.9
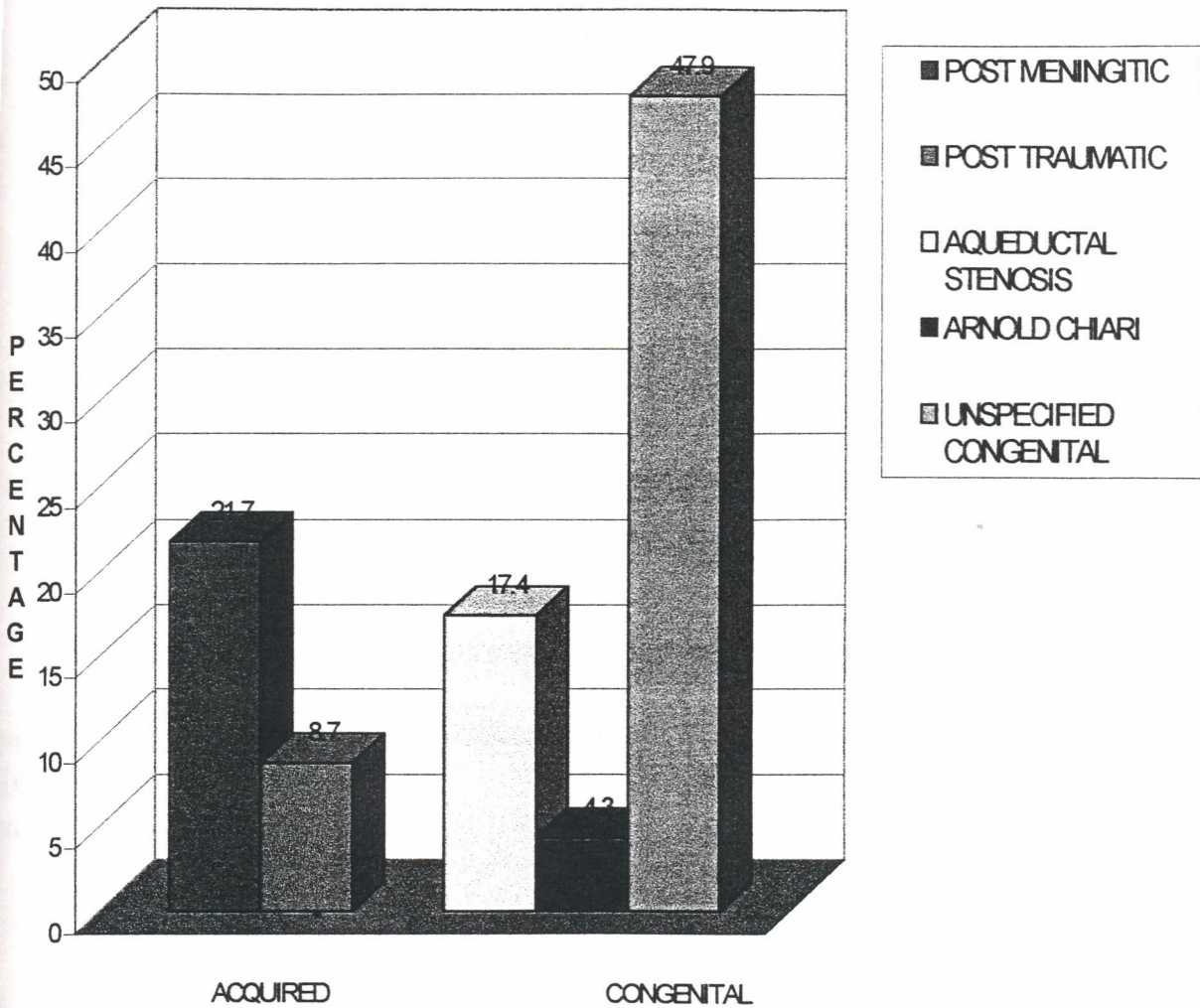
14. Association between time taken for v-p shunt surgery and development of shunt complication

Length of surgery was estimated as time taken from making the first incision to applying the last suture. Seven patients out of 34 (20.6%) whose surgery lasted between 15-30 minutes developed shunt infection. Seven patients out of 39 (18%) whose length of surgery was between 31-60 minutes developed shunt infection.

Comparing the outcome of surgery that lasted less than 30 minutes with that, that lasted more than 30 minutes was of no statistical significance (p=0.870). In this study, the time taken in performing V-P shunt surgery does not seem to be a significant factor in the development of shunt infection. This may be due to the fact that this is a straightforward procedure which in good hands rarely takes more than one hour [Table 13, Figure 12].

Table 13-Surgery time in minutes versus complication.

Surgery time in minutes	Patients with complications			Patients without complications	Total no of patients	%
	Infection	Block	Migration			
15-30	7	3	1	23	34	44.7
31-60	7	4	0	28	39	51.4
60	1	0	0	2	3	3.9

Figure 12-Length of surgery versus type of complication
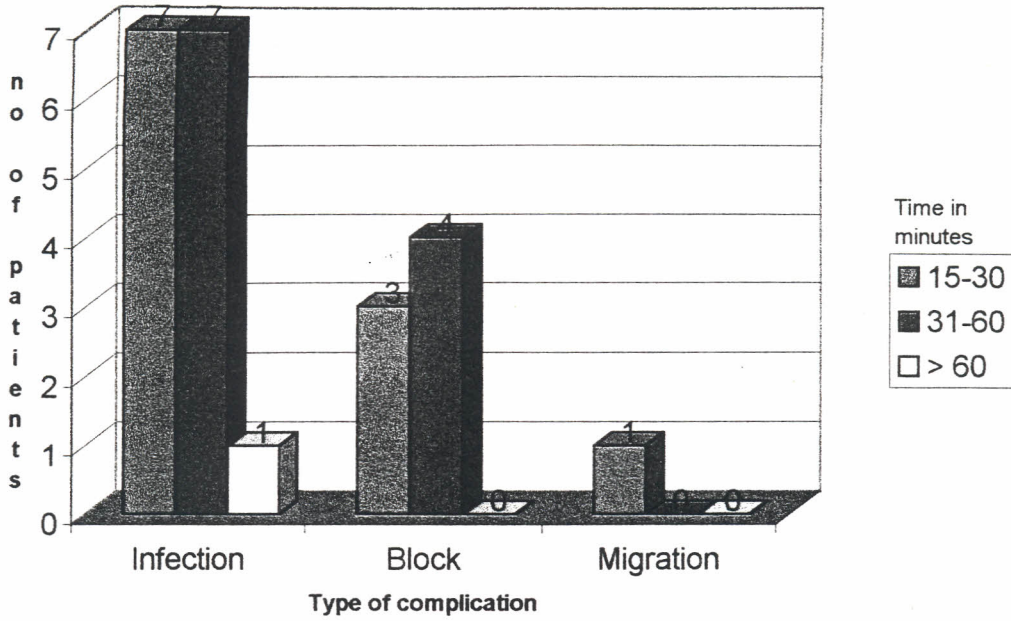
15.Association between status of surgeon and development of complications

Fifty-one patients (67.1%) were operated by Senior House Officers (SHO) out of which 8 developed shunt infection (15.7%). Registrars operated on a total of 24 patients out of which 7 developed shunt infection (29%). Status of the surgeon did not show significant relationship with development of shunt infection (p=0.130). A consultant operated on one patient, in this study, who developed shunt blockage [Table 14, Figure 13].

Table 14-Status of the surgeon and post-surgery complications.

Seniority of surgeon	Patients with complications			Patients without complica-tions	Total no of patients	%
	Infection	Block	Migration			
Consultant	0	1	0	0	1	1.3
Registrar	7	4	1	11	23	30.3
SHO	8·	2	0	42	52	68.4
Total (%)	15 (19.8)	7(9.2)	1(1.3)	53(69.7)	76	100

Figure 13-Status of surgeon andtype of complication
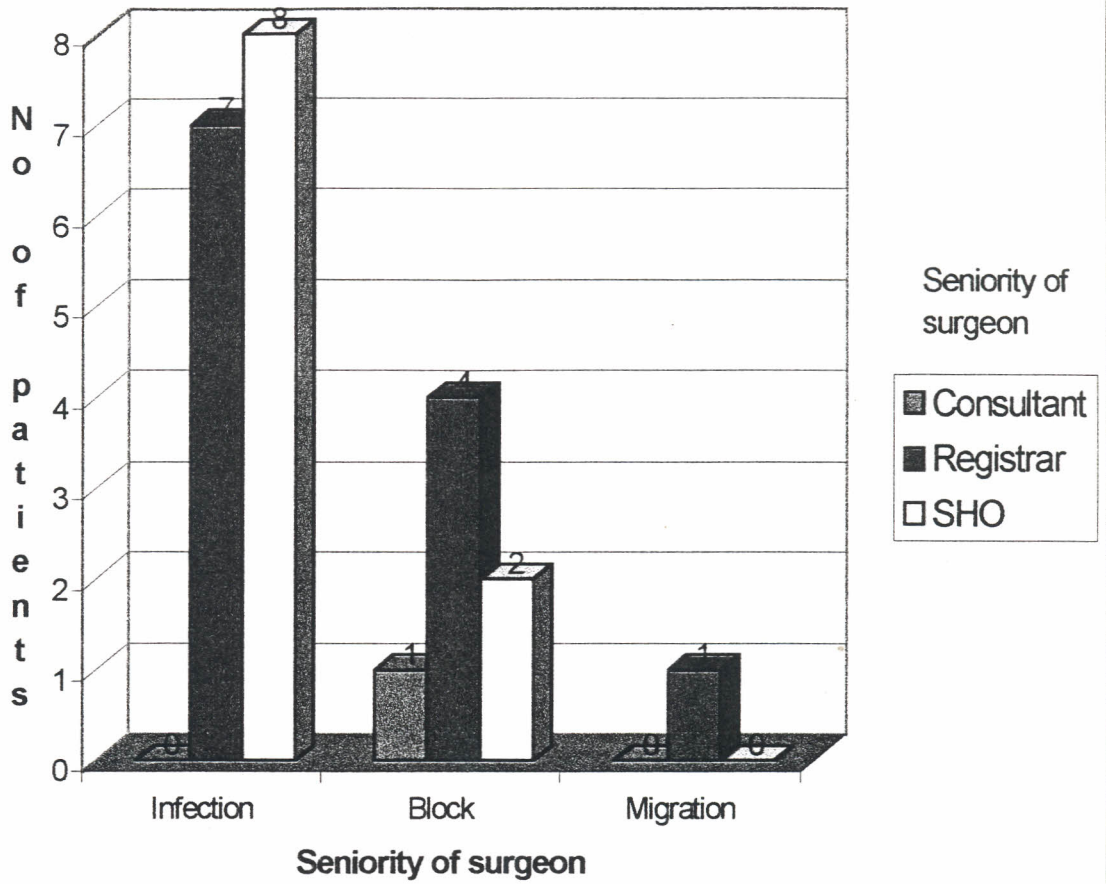
16.Association between type of shunt used and development of post-surgery complication.

Sixty-one patients were shunted using the Chabbra shunt (India), out of which 21.3% developed shunt complication. The Pudenz-Heyer and Cordis-Hakim had complications of 60.0% and 66.7% respectively. However it must be noted that the numbers of shunts used other than Chabbra were small [Table 15, Figure 14]. Comparing the Chabbra shunt with the others was of statistical significance in the development of shunt complications (p=0.0003).

Table 15-Association between type of shunt used and development of post-surgery complication.

Type of shunt	Patients with complications (%)	Patients without complications(%)	Total no of patients	%
Chabbra	13 (21.3)	48 (78.7)	61	80.2
Pudenz – Heyer	3 (60.0)	2 (40.0)	5	6.7
Cordis-Hakim	6 (66.7)	3 (33.3)	9	11.8
Elekta	1 (100)		1	1.3
Total	23 (30.3)	53 (69.7)	76	100

Figure 14-Association between type of shunt used and development of post-surgery complication.
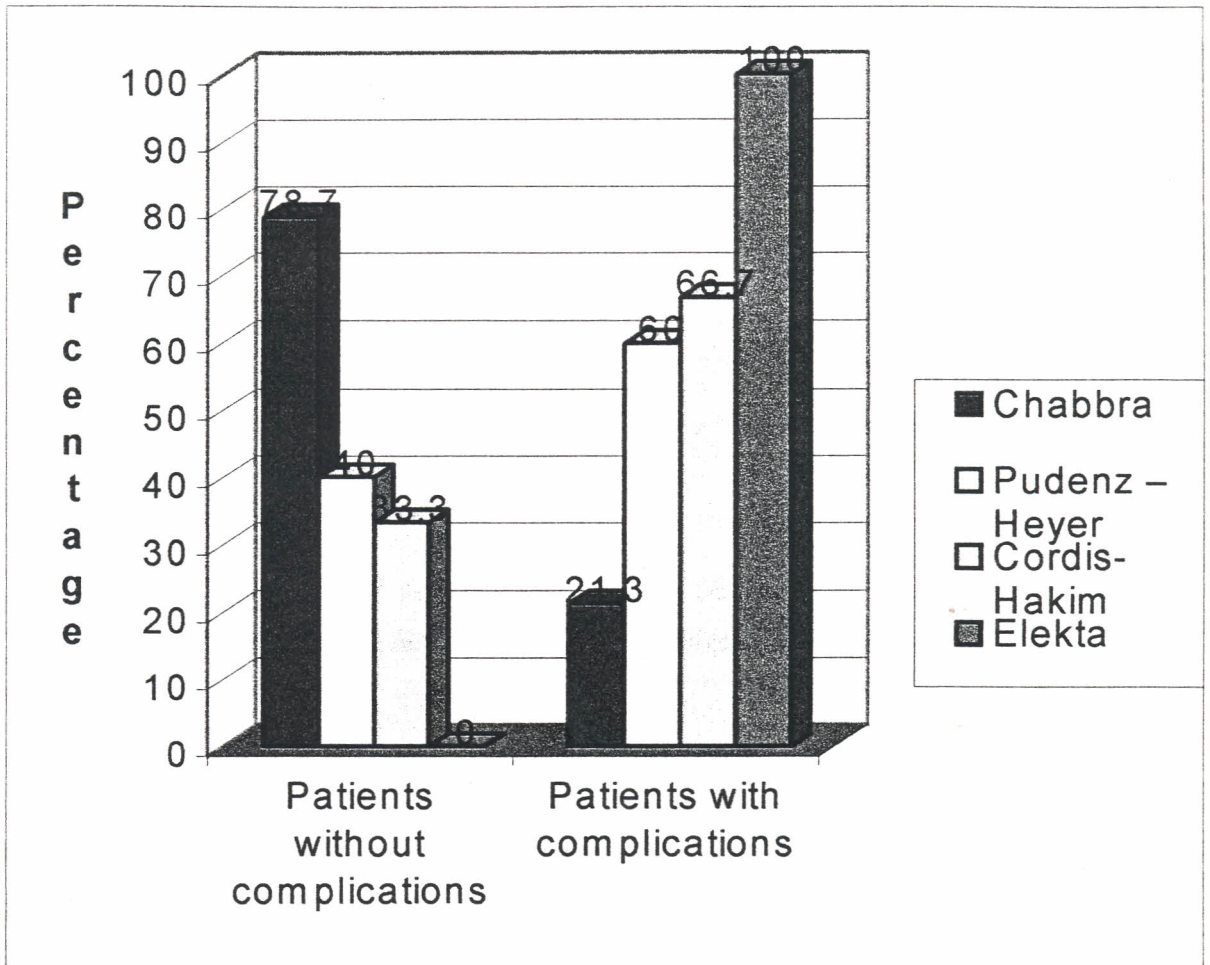
17.Presence of spinal and cranial dysraphism in congenital hydrocephalus and v-p shunt infections

Twenty-two patients had hydrocephalus and spina bifida or occipital encephalocoele and five of them developed features of shunt infection (22.7%). Fifty-four patients had hydrocephalus without an associated spina bifida or encephalocoele and ten of them developed shunt infection (18.5%) [Table 16, Figure 15].

Presence of spinal and cranial dysraphism in patients with hydrocephalus in this study was not associated with an increased risk of developing V-P shunt infection (p=0.670)

Table 16 -Presence of spinal and cranial dysraphism in congenital hydrocephalus and v-p shunt infections

Presence/ absence of dysraphism	Total no of patients with hydrocephalus	No of patients with shunt infection	%
Present	22(28.9%)	5 (33.3%)	22.7
Absent	54(71.1%)	10(66.7%)	18.5
Total	76	15	

Figure 15 - Presence of spinal and cranial dysraphism in congenital hydrocephalus and v-p shunt infections
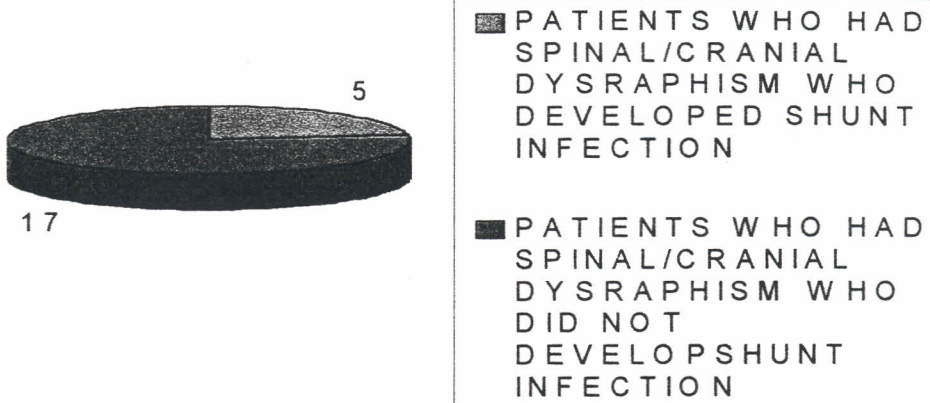
18. Final outcome in patients managed for shunt complications

Fifteen of the patients who developed shunt complication were managed and discharged (65.3%). One patient was still in the ward undergoing treatment for shunt infection (had developed features of ventriculitis) by the end of the study period.

Seven out of the twenty-three patients re-admitted for management of shunt complications died giving a mortality of 30.4%. Four of these died while still in the ward, all from shunt infection following revision. Three were discharged but re-admitted 3-4 weeks later in the paediatric wards for shunt infection and died while undergoing treatment.

Since all the deaths were due to shunt infections, therefore mortality in patients who developed shunt infection was 46.6% and it was the most life threatening complication following shunt surgery in this study.

There was no mortality in the patients managed for shunt blockage or migration. [Table17, Figure 16].

Table 17-Final outcome of patients managed for complications (n=23)

Condition of patient	Number of patients with complications			Percentage
	Infection	Block	Migration	
GOOD	4	3	1	21.7
FAIR	3	4	0	43.6
POOR	1	0	0	4.3
DEAD	7	0	0	30.4
Total	**15**	**7**	**1**	**100**

Figure 16-Final outcome of patients managed for shunt complication
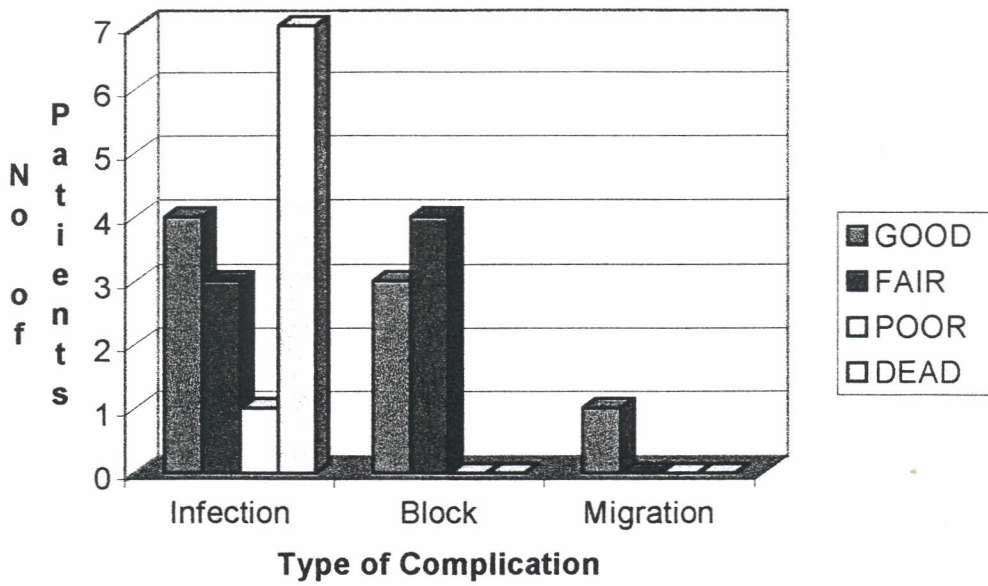
DISCUSSION

This is a prospective study of 76 patients with non-tumour hydrocephalus who underwent V-P shunt insertion at Kenyatta National Hospital over a three-month period (Sep 2001-Nov 2001). All the patients were followed up for three months after shunt insertion.

Shunt complication rate in this study was found to be 30.3%. Shunt infection and malfunction accounted for 19.8% and 9.2% respectively. Bayston [51, 53] argues that shunt malfunction that presents early (within 3-4 months of shunt insertion) is particularly indicative of shunt infection. He says that colonized V-P shunts present with features of shunt block or malfunction due to formation of local adhesions and fibrinous deposit and often encystment by the greater omentum due to discharge of microorganisms into the peritoneal cavity by the colonized shunt. Mwangombe and Omulo [4] had quoted an infection rate of 24.6% based on findings from a retrospective study. In this study the infection rate was 19.8%. This rate is considerably high but falls within the range of 0.0-38.0% that has been reported in the literature [46].

A number of factors may be responsible for this relatively high rate of infection some of which maybe related to the general hospital and theatre environment rather than specific operations as this high rate of infection is reported in other operations at the Kenyatta National Hospital [102]. Other reasons may be patient related e.g. poor nutritional status or surgeon and theatre related e.g. theatre overuse, overcrowding in theatre, poor aseptic

techniques. Relatively long waiting time in the ward may be an added factor [102].

Our male to female ratio of 1.5:1 is similar to that reported in other studies that have shown male predominance in children with hydrocephalus undergoing shunt surgery [5, 46].

The age in this study ranged from one month to fifty-four (54) years, but over 70% of the patients were below twelve months. The peak age of shunt insertion was at 3-4 months. This is expected because majority of patients had congenital hydrocephalus. A similar pattern was seen in Mwangombe and Omulo's study [4] and other studies as well [46]. It should be noted that there were three adults whose hydrocephalus was as a result of trauma. Fourty one point seven percent (41.7%) of patients below 2 months of age developed shunt complication. There was no clear relationship between age of patient and development of shunt complication in this study, however there were more complications in patients below two (2) months of age compared to those between 3-12 months.

The time interval from the onset of hydrocephalus to first shunt placement appears to be little reported in the literature. This is because in the developed countries, where most literature comes from, shunts are inserted soon after diagnosis. The time interval is of interest because it has a bearing on the neurological status of the child and the state of the brain before shunt insertion. Most of the patients in this study were shunted after an interval period of 1-4 months after diagnosis of hydrocephalus. This was a considerable delay. Several reasons were attributed to the delay namely, inability to afford the shunts, poor referral patterns from the periphery,

inability to afford transport costs from periphery when referred and unwillingness on the part of the parents to have V-P shunt insertion.

When time interval between onset of hydrocephalus and 1st V-P shunt insertion was compared with the rate of complications no statistical significance was found (p=0.0698).

The commonest cause of hydrocephalus in this study was congenital, out of which 14 were due to aqueductal stenosis, and one due to Arnold Chiari malformation. The diagnosis of a congenital cause was based on C-T scanning or ultrasonography. Eighteen patients (23.7%) had post-meningitic hydrocephalus. Other studies have reported a similar pattern [4, 46, 60]. Post-meningitic hydrocephalus appears to be more common in our study possibly due to delay in effective treatment of meningitis.

This study also addressed the risk of developing shunt infection in relation to the aetiology of hydrocephalus. Statistically no association was found between the acquired and the congenital type with the development of shunt complication (p=0.71). Most other studies have found no association of the same [44, 48, 57]. Presence of spina bifida or myelomeningocoele did not seem to increase chances of developing shunt complication in this study (p=0.670). A previous study by Davis et al [54] showed that factors such as aetiology of hydrocephalus, age of patient, and presence of an open neural tube defect were not associated with development of shunt infections.

The time interval from shunt insertion to development of shunt infection ranged from 2 days to 3 months (mean of 24 days) in this study. Seventeen out of the twenty-three episodes of shunt complications presented in the first month (73.9%). Many studies report the time interval between shunt insertion and onset of infection to range from a few days to several years [47, 60]. Schoenbaum [44] reported 78% of his infections presenting within the first four months.

Because of financial constrains most of the patients were diagnosed clinically without radiographic evidence. Only about 43% of patients in this study could afford a C-T scan or ultrasonography. Therefore clinical features played a major role in diagnosis.

All the patients with a diagnosis of shunt infection had presented with fever. It was the commonest presenting symptom. Other features included irritability, convulsions and dehydration. Of the general features of shunt infection, fever is seen by many as the major indicator of shunt infection [44, 46,47].

Inflammation along the shunt tract was seen in two of our patients with shunt infection. Schoenbaum [44] has suggested that the finding of erythema overlying the shunt tubing is pathognomonic of infection.

Wound breakdown and exposure of the shunt system was seen in three cases of shunt infection. In one case it had actually dislodged from the abdominal site.

V-P shunt malfunction/block has been shown in some reports to be a sign of shunt infection [51, 52, 46, 47, 51] especially in the initial 3-4 months after insertion. The signs of malfunction include headache, seizure, bulging of anterior fontanelle, excessive rate of head growth, and backing up of CSF along shunt tract among others. In our study the commonest features of malfunction were tense/bulging anterior fontanelle and vomiting. Twelve patients had bulging anterior fontanels, five (5) of whom were diagnosed as shunt infection due to presence of fever. Quigley [46] had 12% of his patients with shunt infection presenting with features of shunt malfunction.

Meningitis (evidenced by neck stiffness, Kernig's positive sign, or isolation of bacteria from the CSF) has been reported in many series [47, 50,60]. Features of meningitis were seen in 6 of our cases of shunt infections (30%). George [60] found a very high rate of meningitis in his series as 74.3% of his infections presented with meningitis.

Peritonitis resulting from shunt sepsis where CSF containing bacteria is discharged into the peritoneal cavity has been reported by some workers [51, 52]. In this study peritonitis was a feature in one case of shunt infection.

One patient presented with features of intestinal obstruction, and one with features of ventriculitis.

Shunt migration has been mentioned in many studies. Gichuhi [5] noted a shunt migration rate of 5.2% in his study. The catheter tip can erode through the bowel, causing presentation with catheter per rectum, ventriculitis, or peritonitis. In our study one case presented with prolapsed shunt from the

anus on defecation. However the patient had no features of shunt infection or malfunction.

Specimens of CSF were taken for bacteriological studies in all 15 patients with a clinical diagnosis of shunt infection. The CSF was either obtained by a ventricular tap or during shunt revision in theatre. Only 4 cultures were positive. Two specimens grew Staphylococcus aureus while out of the remaining two one grew Coagulase negative Staphylococcus and one grew a mixed growth of Proteus and Enterobacteriacae. No case of CSF aspiration from the shunt system for bacteriological studies was done in this study. It has been established that Coagulase negative staphylococci (including those referred to in the literature as staphylococcus albus or staphylococcus epidermidis) are the commonest organisms involved in V-P shunt infections [47, 51, 52, 57] with staphylococcus aureus being reported as the second most common organism isolated [51, 52, 57].

Other factors that might influence shunt complication were also studied. Some of these factors include length of surgery, type of V-P shunt used, seniority of the surgeon, and peri-operative prophylactic antibiotic.

Length of surgery was calculated for each patient from the time of first incision to the last sutures, in minutes. It is thought that the longer the length of surgery the higher the chance of complications. It was noted from this study that from the 15 patients with shunt infection, 7 had an operating time of between 15-30 min, while another 7 had an operating time of between 31-60 min. Only 1 out of the fifteen (15) had an operating time of greater than 60 min. Statistically there was no correlation between length of surgery and rate of shunt infection (p=0.870). A study done by Shurtleff et al [59] mainly

looking at ventriculoauriculostomies showed no correlation between length of surgery and rate of shunt infection. Since shunt insertion is a relatively straightforward procedure and does not take more than an hour, length of surgery should not be a factor in determining rates of shunt infection.

The qualification of the surgeon was also looked at in this study. The SHO (Senior House Officer) is the postgraduate resident in general surgery at the Kenyatta National Hospital. All the SHO's have to do a 3-month rotation in the neurosurgical unit.52 (68.4%) of the patients in this study were operated on by SHO's out of which 8 (15.4%) developed shunt infection. A Registrar is a qualified surgeon who is doing his residency in the neurosurgical unit. The Registrars operated on 23(30.3%) of the patients in this study out of which seven (30.4%) developed infection. A consultant operated on one patient. Comparing the outcome between the Registrars and SHO's did not show any statistical significance (p=0.130).

Some reports [48, 59] have shown no differences in infection rates between experienced and inexperienced surgeons (example consultant neurosurgeon and registrars) while other reports [55, 60] showed inexperience of the surgeon as a risk factor in shunt infection. Another study [46] showed that these differences between surgeons and shunt infection could be a reflection of some individual proneness to have high infection rates.

Type of shunt used was also looked at in this study. Patients operated at Kenyatta National Hospital for shunt surgery have to buy the shunt themselves and the cheapest available shunt is the Chabbra (India) shunt (cost approximately 4000 Kenya Shillings, 50 US dollars). Chabbra shunt

was used in 61 patients (80.2%) in this study, out of which 13 patients (21.3%) developed shunt complications. The remaining 15 patients (19.8%) in this study were shunted using the Pudenz-Heyer and the Cordis-Hakim shunts, out of which 10 (66.7%) developed shunt complications. This was statistically significant (p=0.0003). These shunts are sometimes available at the Kenyatta National Hospital as donations from the developed countries. Other studies [58, 59] have shown no co-relation between the type of shunt used and complications developed.

There has been no standard protocol at the Kenyatta National Hospital on use of prophylactic antibiotics in shunt insertion surgery. Use of prophylactic antibiotic was also looked at in this study. A generic of Ceftriaxone was used and patients were randomly chosen and received an intravenous dose of 250mg of antibiotic during induction of anesthesia. A total of 27 patients (35.5%) received the antibiotic out of which 3(11.1%) developed shunt infection. Out of 49 patients who did not receive antibiotics 12 developed shunt infection giving an infection rate of 24.5%. Of the 15 patients who developed shunt infection, 3(20%) had been given prophylactic antibiotic while 12(80%) had not been given. Statistically no significant reduction in shunt infection was found using the antibiotic (p=0.160).

Different treatment modalities are used in the management of shunt complications. All the patients with a diagnosis of shunt infection in this study were put on intravenous antibiotics for a total duration of 7 - 14 days. Many workers have recommended the use of intravenous antibiotics in the management of shunt infection [51, 103]. In our study, 6 patients improved on intravenous administration of antibiotic alone without any surgical

intervention. Many workers have reported unfavourable results in treatment of shunt infection with antibiotics alone without removal of the shunt [44, 103, 104]. Although this option was used in some of our infections, it is to be discouraged. No patients in this study were managed with intraventricular use of antibiotics.

Nine (9) of the patients with shunt complications were managed by externalization of the distal end of the shunt. The externalized end was connected to a urine bag or a large syringe and the CSF allowed to drain out. These patients were also covered with intravenous antibiotics. Once fever and other clinical features had settled down the patients were taken to theatre for revision of the shunt.

External ventricular drainage as has been cited by James [103], requires constant and experienced nursing, facilities to monitor and replace fluids, electrolytes and proteins. There is also the added risk of superinfection.

Seventeen patients with shunt complications had to be taken to theatre for revision of the shunt. One patient who had his shunt removed was not reinserted because a diagnosis of arrested hydrocephalus was made.

It is now established that the best option in the treatment of any CSF shunt infection is immediate removal of the infected shunt [44, 46, 47, 51, 61] combined with antibiotic administration intravenously [44, 51, 103] and intraventricularly [51, 103] to control infection before a new shunt is reinserted. External ventricular drainage is adviced in the intervening period

[44, 46, 51, 103] to avoid complications that may arise from raised intracranial pressures.

Outcome of patients managed for complications was also judged. Fifteen out of the twenty-three patients (65.3%) managed for complications in this study improved within 7-14 days and were discharged for follow up in the clinic. One patient was still in the ward being managed for shunt infection at the end of the 3-month follow up period. A total of seven patients died due to shunt infection. None of the patients being managed for shunt malfunction died. A mortality of 30.4% in the management of shunt complication was observed in our study, although all the deaths were related to shunt infection or systemic complication.

In conclusion it has been observed that rates of shunt complications at the Kenyatta National Hospital still remain high with shunt infection as the commonest cause. The study also shows us that the commonest cause of hydrocephalus is congenital. The commonest clinical feature of shunt infection was presence of fever. Most of the shunt complications are treated by shunt removal and reinsertion. Mortality in the management of shunt infection remains high.

RECOMMENDATIONS

I recommend the following:

- Use of the Chabbra shunt as a standard.
- Use of a different antibiotic for prophylaxis (a study should be done using a known effective antibiotic like Zinacef)
- Follow strictly the standard principles of infection control
- Effective use of intraventricular antibiotics in treatment of shunt infection should be encouraged and a study to gauge its efficacy should be done
- The high mortality in patients with shunt infection should be addressed to and a multidisciplinary team approach including the neurosurgeons, neurologists and microbiologists should be encouraged
- Closer clinic visits in the first four weeks after shunt insertion for early detection of complications and institution of treatment
- A larger prospective study over a longer period is recommended. Other factors e.g. emergency v/s elective surgery should be looked at. Differences in culture results between CSF and catheter tip should also be looked at.

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APPENDIX 1

DATA COLLECTION FORM

POST VP SHUNT COMPLICATIONS

1. PATIENT PROFILE

IP NO: ------------------------

NAME: --

AGE: -----------

SEX: Male -----   Female -----

DATE OF BIRTH ----------------------

AGE OF PATIENT AT ONSET OF HYDROCEPHALUS ------------

DATE OF 1st VPSHUNT SURGERY ----------------------

AGE OF PATIENT AT 1st VPSHUNT SURGERY ---------------------

ETHNIC GROUP: ---------------- HOME DISTRICT: --------------

2. TYPE OF HYDROCEPHALUS:

  (a)   CONGENITAL -------------------------------------- (specify which type if known)

  (b)   ACQUIRED _ POST-MENINGITIC -----------------------

                _ POST-TRAUMATIC -----------------------

              _ OTHERS -------------------------------------

  (c)   ANY ASSOCIATED CONDITION AT TIME OF SURGERY ----- -------------------------------- (eg Anaemia, URTI, Skin Sepsis, Scabies or any other infection)

  (d)   OTHER ASSOCIATED CONDITION e. g.

                _SPINA BIFIDA -------------------

                _OTHER (Specify) -------------------

  (e)   HEAD CIRCUMFRENCE AT 1ST VISIT ------------------------ cm.

3. PERI-OPERATIVELY:

(a)   Prophylactic Antibiotics YES ---------- NO--------

(b)   Length of surgery in minutes -----------------

(c)   Qualification of Surgeon   CONSULTANT-------------

                                       REGISTRAR ----------------

                                       S.H.O ------------------------

(d)   Type of Shunt used ---------------------------------

4. POST-OPERATIVE FOLLOW UP

(a)   Condition of patient in the first 48hrs after surgery;

          Excellent --------- Good-----------Fair--------Poor----------

(b)   Any of the following features apparent in the first 48hrs after surgery;

                  FEVER-------------------

                  VOMITING-------------

                  IRRITABILITY-------------

                  LETHARGY-------------------

(c)   How many days post-op was the patient discharged------

(d)   Interval between surgery and onset of complication----------------- --

(e)   Clinical presentation during follow-up in the clinic;

            i) ·   General feature

                  Fever---------Irritability----------

                  Convulsions--------- Diarrhoea----------

                  Dehydration---------- Others-------------

            ii)   Local features

                  Wounds/Sepsis-----------------------------------

                  CSF leak from wound------------------------

                  Exposed shunt--------------------------------

           iii)   Signs of inflammation along shunt tract------

           iv)   Signs of shunt malfunction/block

Tense anterior fontanelle--------------------

Convulsions------------------------

Visual disturbance-----------------

Papilloedema----------------------

Headache--------------------------

Vomiting---------------------------

Shunt migration---------------------

Head circumference --------------------

v)   Features of meningitis--------------------------

vi)   Abdominal signs; Distention----------------

Peritonitis----------------

Intestinal Obstruction------

vii)   Mal-positioned shunt leading to neurological deficit
----

vii)   Other complications --------------------------------------
----

5 .INVESTIGATIONS :

PRE-OP
  i. L.P CSF _____

 ii. Ultrasonography
      results_____

iii. CTScan
      results_____

 iv. WBC count

      _____

  v. HB _____

POST-OP(if   shunt complications present)
  i. L.P CSF _____

 ii. Ultrasonography
      results_____

iii. CTScan
      results_____

 iv. WBC count

      _____

6. MANAGEMENT OF COMPLICATIONS:

  i) Non-surgical; Patient improved on antibiotics ?
     YES_____   NO_____

  ii) Surgical Treatment; Shunt externalised in the ward? YES_____   NO _____

  iii) Surgical Treatment; Shunt re-inserted in theatre ? YES_____   NO _____

  iv) Was there improvement after the surgical treatment? YES_____   NO_____

  v) Duration of hospital stay for managing the complication _____

7. FINAL OUTCOME:

  i) Any readmission after discharge for the 1st episode of complication? ----------------------------
----

  ii) Condition of patient at discharge

    >Good _____   >Fair_____

    >Poor_____   > Dead_____

THANK YOU !!!!

APPENDIX 2

CONSENT FORM

I / Parent of _____, do hereby freely consent to participate in this research study on COMPLICATIONS OF VP SHUNT INSERTION AS SEEN AT KENYATTA NATIONAL HOSPITAL. Dr Salim Noorani has explained the problem being dealt with and the nature of the study to me. It is understood that participation or otherwise in this study will not adversely affect my medical care, and that I can withdraw from the study at any time, again without any adverse consequences. I also understand that all information about myself/child shall be treated in the strictest confidence.

Signed: _____

Witnessed: _____

Dated: _____

APPENDIX 3

KENYATTA NATIONAL HOSPITAL
P.O. Box 20723,
NAIROBI.

Tel: 726300 - 19
    726550 - 9
    726562 - 6
    726450 - 9
    726581 - 2
Fax: 725272

Email: *knh@healthnet.or.ke*

Ref: KNH-ERC/01/1130   30 August 2001

Dr. Salim K. Noorani
Dept. of Surgery
Faculty of Medicine
University of Nairobi

Dear Dr. Noorani,

RE: **RESEARCH PROPOSAL** "COMPLICATION OF VENTRICULO-PERITONEAL SHUNT INSERTION AS SEEN AT KENYATTA NATIONAL HOSPITAL: A PROSPECTIVE STUDY"
(P67/7/2001)

This is to inform you that the Kenyatta National Hospital Ethical and Research Committee has reviewed and **approved** your above cited research proposal.

On behalf of the Committee I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of data base that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Thank you.

Yours faithfully,

PROF. A.N. GUANTAI
SECRETARY, KNH-ERC

c.c.   Prof. K.M. Bhatt,
        Chairman, KNH-ERC,
        Dept. of Medicine, UON.

        Deputy Director (CS),
        Kenyatta N. Hospital.

        Supervisor: Mr. N.J.M. Mwangombe, Dept. of Surgery, UON

        The Chairman, Dept. of Surgery, UON

        The Dean, Faculty of Medicine, UON