A Review in Pediatric Hydrocephalus: Physiology, Classification, Clinical Presentation, Imaging and Treatment

Iraj Lotfinia*

Department of Neurosurgery, Tabriz University of Medical Science Tabriz, Iran

Abstract

Hydrocephalus, increased cerebrospinal fluid (CSF) within the skull, is a common disease, and it may develop due to various causes, such as congenital anomalies, tumors, infections, intracranial hemorrhage, trauma and idiopathic disorders. The disease can occur in all ages, even prior to birth through late old age, and is associated with various clinical symptoms. Lack of treatment or delays in treatment can lead to permanent brain damage. Appropriate therapy can prevent the occurrence of this complication in most cases, and a ventriculoperitoneal (VP) shunt is often used to treat this disease. However, this method is accompanied with numerous complications and problems, and, as a result, the patient may require repeated surgeries. Careful selection of patients for surgical intervention and surgery using proper techniques can reduce these side effects.

INTRODUCTION

The term hydrocephalus is derived from two Greek words: hydro, meaning water, and cephalus, meaning head. The disease has been known since ancient times because of the strange appearance of these patients; Hippocrates were the first person who described decompression and trepanation in these patients [1]. Hydrocephalus is defined as an increased volume of intracranial cerebrospinal fluid CSF. Although this fluid accumulates in the ventricles in most cases, the CSF sometimes accumulates outside the ventricles and in the subarachnoid space around the brain, and such cases is called external hydrocephalus [1].

Prevalence

Hydrocephalus is the most frequent neurosurgical problem seen in pediatric medicine also, there is no preponderance between males and females [1]. Although the exact prevalence of this lesion is unknown, more than half of the surgeries performed by pediatric neurosurgeons involve embedding or revision the shunt [2]. Surgery for hydrocephalus is much less frequent in adults. According to investigations, the annual incidence of surgery for hydrocephalus is 3.4 per 100,000 in adults undergoing surgery [1]. Moreover, some cases of hydrocephalus are believed to be caused by genetic factors [3].

Physiology

Parts of the brain tissue that constitute the cerebral ventricles and the space surrounding the brain and spinal cord that include the subarachnoid space contain CSF. This liquid has several functions of which the most important are a significant reduction in the weight of the brain and spinal cord floating in the liquid, and a reduction in the likelihood of damage followed by sudden motion and impact on the inner surface of the skull. The other function of CSF is its nutritional role for the brain and spinal cord surface areas. In addition, CSF is probably the disposal pathway for excess neurotransmitters and some metabolic waste products from the central nervous system (CNS). The majority of CSF is produced by the choroid plexus in the lateral, third, and fourth ventricles. The CSF is synthesized naturally by the ultrafiltration through the choroidal capillary endothelium wall. This secretion is dependent on the sodium-potassium-ATPase pump in the choroid plexus apex and is an energy-dependent process. No change is found at the level of CSF secreted from the choroid plexus following a sharp rise in intracranial pressure (ICP), but a decrease in the secretion of CSF will occur in chronic increased ICP due to atrophy created in the choroid plexus [4]. A smaller amount of CSF is created by ependymal secretion within the ventricles and the subarachnoid space. The historical treatment of plexectomy for hydrocephalus has failed because of this fluid.
secretion from areas other than the choroid plexus. According to recent studies, secretion of CSF and interstitial fluid is the result of water filtration through the vessel walls. This is associated with the maintenance of electrolytes and therefore osmotic pressure in the vessels, which causes reuptake of liquid to the capillary venous [5]. This theory posits that the formation and absorption of fluid take place in different areas of the CNS, and sagittal sinus granulations play little role in fluid uptake due to low cross-sectional area [5]. Water constitutes more than 99% of CSF; all the water does not pass through the entire CSF route in the subarachnoid space, but rather is reabsorbed in the adjacent vessels. The distribution of other substances in the subarachnoid space occurs to a greater extent when the reuptake of them to the microvessels is slower [5].

The liquid enters from the lateral ventricles through the foramina of Monro into the third ventricle, and then into the fourth ventricle through the aqueduct of Sylvius. The CSF from the fourth ventricle flows into the subarachnoid space through a central foramen of Magendie and two lateral foramina of Luschka. According to the traditional hypothesis, this flow is unidirectional [5] and after flowing around the brain and spinal subarachnoid space, the fluid can be reabsorbed through the arachnoid villi around the superior sagittal sinus into the systemic circulation. The CSF formation requires energy consumption, but its absorption is passive [6]. This absorption is dependent on the CSF pressure and can be increased significantly with higher intra cranial pressure. On the other hand, when pressure in the sagittal sinus increases, the ICP must increase too for CSF to be absorbed [7]. In patients with subarachnoid hemorrhage and trauma, fibrosis along the route of CSF flow can induce hydrocephaly [8]. ICP has been defined as the hydrostatic pressure within CSF and is dependent on certain factors, such as active CSF secretion, resistance to exit routes, and passive CSF absorption into the dural sinuses. Due to the above factors, hydrocephalus may be caused by three mechanisms: increased production, obstruction in the route, and elevated venous pressure in the dural sinuses, which reduce CSF absorption [1]. For normal ICP, there must be balance and coordination among the production, storage, and absorption of CSF, and the occurrence of any imbalance in this area can lead to clinical symptoms. According to the site of obstruction and disruption in the CSF flow, hydrocephalus can be divided into two groups: the obstructive (non-communicating) type where there is blockage in the CSF flow through the ventricles, and which may occur in any part of the ventricular system such as the lateral, third, and fourth ventricles; and the non-obstructive (communicating) type where there is a normal ventricular CSF flow path and impaired CSF absorption through the arachnoid villi and, thus, non-normal absorption [9]. The non-obstructive type is uncommon and develops due to factors such as recurrent subarachnoid hemorrhage or meningitis, which disrupt the function of absorption. The CSF overproduction from the choroid plexus papilloma has been described as a cause of communicating hydrocephalus, but this concept is controversial [10]. The increase in CSF volume inside the skull results in enlargement of the ventricles and increased pressure inside the skull. Increased intracranial pressure can lead to neuronal loss and brain damage. The ventricles may not be dilated after hydrocephalus and CSF blockage in the early stages [11]. The enlargement of the ventricles requires time, which depends on the severity and velocity of hydrocephalus formation. In newborn infants with open sutures that are not surrounded by rigid skull, increased ICP in the early stages may be compensated for by bulging fontanelle and diastasis of sutures, and so ventricular dilatation may not be observed [11]. In other groups of patients, a new balance may be established between production and absorption of CSF following enlargement of the ventricles. In this case, the ICP is normal, and the infants probably will not benefit from the insertion of a shunt [11]. Da Silva et al. [12], showed that cerebral blood flow in acute hydrocephalus was reduced globally and the decrease in blood flow in chronic hydrocephalus was dominantly around the ventricles. This hypoperfusion can lead to brain damage due to the reduction of oxygen and glucose in the brain [11]. The increased fluid pressure also can cause significant oxidative changes and may lead to gliosis in all of the brain [13]. Some white matter abnormalities seen in patients with untreated hydrocephalus, such as ependymal rupture, periventricular edema, and collapse of periventricular capillaries, will be reversible with early treatment, but axonal degeneration and gliosis are signs of permanent damage in the white matter [11] and remain following the treatment of hydrocephalus.

Classification

Various types of hydrocephalus have been described. As mentioned earlier, hydrocephalus can be classified into two main forms based on the site of blockage: obstructive and non-obstructive. Instead of these two words, non-communicating and communicating can also be used. Simple classification of hydrocephalus into the obstructive and non-obstructive types is not enough if it is possible to identify the actual site of obstruction [7]. Studies have shown that acute obstruction of the Sylvius aqueduct is unable to produce a change in intraventricular pressure. This finding suggests a balance between production and absorption of CSF within the ventricles [5]; several factors besides obstruction, therefore, are likely involved in the development of hydrocephalus. Hence, it is better to modify the classification of obstructive and non-obstructive hydrocephalus to reflect patient age, the presence of lesions and rapidity and chronicity of lesions [7]. Various factors can affect the CSF system and cause communicating hydrocephalus, including tumor surgery, brain trauma, infections and subarachnoid and intraparenchymal hemorrhage within the brain. Nevertheless, according to the clinical experience of Xu [14], a large percentage of these patients do not suffer from hydrocephalus because of the compensatory CSF absorption capacity within a certain range.

Another form of classification used in hydrocephalus is based on time of onset of the lesion, which is divided into two types: congenital and acquired. In congenital hydrocephalus, the lesion is present in the infant prior to birth, such as a narrowing of the cerebral aqueduct and hydrocephaly associated with neural tube defects. In some cases, congenital hydrocephalus has idiopathic etiology. The incidence of congenital hydrocephalus is about 0.5–0.8 per 1,000 live births [15] and most commonly involves aqueductal stenosis [16]. It should be considered that hydrocephalus in all cases of the congenital form may not exist at birth, but may appear later in life. Other terms used in the classification of hydrocephalus are normal pressure
hydrocephalus (NPH) and high-pressure hydrocephalus (HPH). The term normal pressure is not appropriate, and attacks of increased ICP can be observed in these patients [17,18]. Another classification is applied in this case: internal hydrocephalus in which the ventricles are dilated and the CSF accumulates in the ventricles (Figure 1), and external hydrocephalus in which the CSF accumulates in the subarachnoid space surrounding the ventricles (Figure 2). Hydrocephalus due to clinical symptoms is divided into two types: active hydrocephalus in which the patient shows clinical features, and occult hydrocephalus in which the patient has an increase in intracranial CSF in imaging, but without clinical features. Arrested hydrocephalus implies a halt in the growth of the lesions and no increase in ventricular volume over time. The hydrocephalus based on the appearance rate of clinical symptoms is divided into three types: acute (within days), subacute (within weeks), and chronic (within months).

Clinical features

Symptoms: The clinical symptoms will vary depending on the primary cause of lesions leading to hydrocephalus, age, and acute or chronic condition of the lesion. Prior to closure of the cranial sutures and the fontanels at an early age, the dominant manifestations of the disease are a progressive increase in head circumference and bulging of the fontanels (Figure 3). Different symptoms may be observed at an early age, including poor feeding, vomiting, general irritability, drowsiness, seizures, impaired eye movements, bradycardia, hypertension, and prominence of scalp veins. After the closure of the cranial sutures, the dominant clinical symptoms are related to increased ICP, and the patient presents predominantly with headache, nausea, vomiting, irritability, drowsiness, blurred vision, and double vision, loss of bladder control and changes in personality and memory. Headache, nausea, and vomiting are usually worse in the morning, and there are two reasons for this. The ICP will
increase further at night because of the lying position, resulting in some venous stasis. Moreover, carbon dioxide retention occurs briefly during sleep due to mild respiratory depression, which causes dilation of cerebral blood vessels and, thus, the increase in blood volume within the skull, and, consequently, ICP will increase further. Because the corticospinal fibers of the lower extremities which pass along the lateral ventricle may be more prone to stretching and tension, their dysfunction will lead to weakness in the lower limbs, impaired gait, and frequent falls.

The patient may complain of neck pain that can be caused by the herniation of the cerebellar tonsils. The patient may also complain of transient attacks of blurred vision, indicating impaired blood flow and ischemic optic neuropathy. In these cases, prompt medical and surgical measures should be taken.

In rare cases, endocrinological symptoms, such as delayed growth, obesity, and early puberty, may occur due to the enlargement of the third ventricle and the pressure on the hypothalamus.

Signs: In the lower ages and prior to closure of the cranial sutures, the most important sign is excessive abnormal head circumference. Usually, there is no papilledema in these ages, but often it is seen after the closure of the cranial sutures. Spastic weakness may be seen in the lower limbs due to the stretching of corticospinal fibers. The patient may have the failure of upward gaze due to pressure on the tectal plate through the suprâpineal recess of the dilated ventricle. This condition can lead to sunset eye sign in infants (Figure 3). Unilateral or bilateral sixth nerve palsy also can be seen, which is often the result of high ICP.

Diagnostic techniques

Plain radiography: This technique is less frequently used to diagnose hydrocephalus. Increased skull size and open sutures can be seen in infants. The increase in skull size will lead to a disproportion between the sizes of the skull and face. The pathologic calcification, such as that due to brain tumor or neonatal infection (toxoplasmosis, rubella, cytomegalovirus, and herpes simplex) may be detectable by this method. Also, signs of increased ICP, such as destruction of dorsum sellae and silver beaten appearance in the skull, may be seen in plain radiography.

Ultrasound: This is a noninvasive method that does not have any proven side effects. Although it is able to detect prenatal hydrocephalus, the etiology is not usually diagnosed by this method. It can be used in infants with open fontanelle, and can determine the size of the ventricles very well. Fluid accumulation in the brain and the presence of lesions, such as cerebral hemorrhage and space occupying lesions can be detected by ultrasound.

Computerized tomographic scan (CT-Scan): CT-Scan is able to show the size of the ventricles and lesions, such as bleeding, pathologic calcification, vascular lesions and tumors, as well as some of the signs of increased ICP, such as edema surrounding the ventricles, flattened cerebral gyri, and reduced subarachnoid space.

Magnetic resonance imaging (MRI): MRI can diagnose hydrocephalus and pathologic factors, without the complications associated with radiation. In addition, phase-contrast cine MRI application can be used in patients with suspected hydrocephalus to assess how CSF flows and to locate possible obstruction sites. Due to the time required and the need for immobilization of the patient during the examination, sedation may be needed in young children and infants.

Transcranial doppler ultrasonography (TCD): TCD ultrasonography can be used as a noninvasive method at the bedside, which indirectly measures ICP [11]. In this way, the ICP status can be assessed with regard to systolic and diastolic blood flow in the brain and its changes. This method is useful, especially in young babies, whose signs and symptoms of increased ICP are often uncertain [11].

Treatment

The treatment process is specified after the necessary studies to determine the underlying causes of the hydrocephalus. In cases where other lesions, such as tumors, vascular lesions, or infection are present, relevant therapeutic measures should be done. In the following section, we will primarily discuss the treatment of hydrocephalus.

Non-surgical treatment:

Drug therapy: There are no compelling non-surgical therapies for hydrocephalus [19], but, in cases like cerebral hemorrhage leading to hydrocephalus, since hydrocephalus may be transient, drug treatment can be considered for a short time. For this purpose, acetazolamide (50-100 mg/kg/day) can be used alone or in combination with furosemide (1 mg/kg/day). The effect of these drugs on CSF secretion is rapid and reversible [20].

The mechanism of action of acetazolamide is the inhibition of carbonic anhydrase enzyme, which thereby reduces CSF
secretion. The mechanism of action of furosemide that reduces ICP is unclear, but its impact is likely due to a decrease in interstitial fluid in the brain.

**Lumbar puncture:** Similar to medical treatment, in cases where the cause of hydrocephalus is temporary, lumbar puncture can be attempted repeatedly and for a limited time to reduce ICP. It should be noted that lumbar puncture is allowed to be performed only in communicating hydrocephalus. This method can lead to death if used for the obstructive type due to the occurrence of brain herniation and its complications.

**Surgical treatment:**

**CSF diversion with shunt placement:** For this purpose, a ventriculoperitoneal shunt is commonly used [21]. In cases where there is no possibility of using this method because of factors such as abdominal infection or previous surgical adhesions, a ventriculocisternal or ventriculopleural shunt can be used. With the ventriculocisternal shunt, the distal portion of the shunt is inserted through the jugular vein and its tip must be parallel to the junction of the superior vena cava and right atrium. For this purpose, the shunt tip should be controlled by intraoperative radiography; the perfect position of shunt tip placement is between the sixth and seventh thoracic vertebrae. In the case of communicating hydrocephalus, and when the ventricular size is small, a lumboperitoneal shunt can be used. The ventriculoperitoneal shunt is preferred due to several factors, the most important of which is related to the unique ability of the peritoneum to easily localize infection and prevent its spreading. This is also the easiest method of shunt surgery [21]. Moreover, with this method, a substantial length of the peritoneal catheter can be placed inside the abdomen to avoid disruption of shunt function by child development and shunt peritoneal catheter stretching upwards, which avoids the need for repeated surgery. In these cases, use of antibiotic-impregnated catheters will be associated with a significant reduction in infection rate [22].

These shunts basically consist of three parts: ventricular catheter, pump, and peritoneal, lumbar, or arterial catheter (depending on the site of shunt insertion). There are many types of shunts which have fundamental differences in the pump. The shunt pump acts as a one-way valve, and, depending on the pressure set for it, the CSF comes out of the brain when the CSF pressure is higher than the set pressure, and no fluid will be discarded from the system at lower pressure. The cerebral shunt pump is manufactured in two forms. The first type is adjustable, and its settings can be changed after surgery depending on the CSF volume required to drain from the brain. With the changes, intracranial pressure will be reduced or increased. The changes in the pump take place through the skin, using a special magnetic field. The second type of pump has a constant pressure and allows draining CSF from the brain at a constant pressure setting. These pumps are made in low, medium, and high pressure settings. The pump valve opens at the pressures of 5, 10, and 15 cm water, respectively. The type used is selected depending on the medical condition, the lesion causing hydrocephalus, and the volume required to drain the CSF. The pressure required for these pumps to drain CSF from the brain is not changeable. However, most of the time the medium pressure setting is used. Despite a few complications reported with programmable valve [23], and its large artifacts in MRI [24] it is efficient and safe tool in the treatment of high pressure and normal pressure hydrocephaly [25,26].

In this way, the ventricular catheter is inserted into the lateral ventricle through surgery and is connected to the pump that is placed under the skull skin. The peritoneal catheter is also connected to other side of the pump and is inserted in the peritoneal cavity via subcutaneous tunneling. The ventricular part is embedded by frontal entry point (10 cm above the nasion and 2.5 cm on the right) or occipital entry point (6 cm above the inion and 3 cm off the midline on the right side). The catheter is conducted toward the lateral ventricle frontal horn. Maximum catheter entry is 6 cm from the frontal region and 11 cm from the occipital region. For the peritoneal region, the right upper quadrant of the abdomen is used. The catheter is placed about 20-30 cm [27] into the peritoneum. In these cases, use of antibiotic impregnated catheters will be associated with a significant reduction in infection rate [22].

One of the shunt complications is over drainage in the standing or sitting positions as a result of gravity. To deal with this problem, an anti-siphon device is added to the shunt system.

**Endoscopic third ventriculostomy (ETV):** Although the insertion of a ventriculoperitoneal shunt is one of the most common neurosurgery procedures in our daily practice [28] and often children with hydrocephalus are treated with it [9], other treatments should also be considered due to the high prevalence of the complications of this method. In the last two decades, there have been shifts in the therapeutic pattern from the ventriculoperitoneal shunt toward the ETV [29]. In cases where there is a certain obstruction in the CSF pathway in the third or fourth ventricular system, the ETV may be a useful approach [30]. The use of this method is effective in both children and adults [29]. Also, ETV seems to be the first-line therapy in the narrowing of the cerebral aqueduct due to its low rate of complications [21,31].

ETV is a routine and safe method for treating obstructive hydrocephalus [32]. The use of this technique has been on the rise in the past two decades [33], and today it is the first choice for treatment of obstructive hydrocephalus [32]. In this approach, age is a determining factor for prognosis [34]. The success rate with this method is higher in children over one-year-old and patients who have no history of surgery [35]. Because there is no need for an implant in ETV, the incidence of long-term complications is reduced [36].

ETV can be used in the patients with non-communicating hydrocephalus. This method is therefore appropriate for treatment of hydrocephalus secondary to obstruction due to tumor, aqueductal stenosis, myelomeningocele, postinfectious hydrocephalus and in triventricular hemorrhage [35]. In this way, an endoscope is inserted through a small burr hole in the skull into the lateral ventricle and then the third ventricle through the foramina of Monro and creates foramina in the floor of the third ventricle. As a result, the CSF will be able to exit from the third ventricle into the subarachnoid space and can continue its normal flow. In the patients who are appropriately selected and where the CSF absorption in the subarachnoid space is appropriate, the
acceptable result will be obtained. However, the basilar artery status in these patients should be evaluated prior to surgery. In cases where an artery is lower than the ventricular floor, the risk of intraoperative damage increases, and this method should not be used in the surgery [37]. ETV is a preferred method for the treatment of the narrowing of an aqueduct in most neurosurgical centers [31]. According to Warf [38], the result will be better using bilateral ventricle choroid plexus cauterization during this surgery.

It should be considered that only the presence of ventriculomegaly in ultrasound, CT-Scan, or MRI cannot be considered as an indication for surgery [11]. In cases with normal or slightly elevated ICP, even if the size of the ventricles is large, the CSF drainage will be contraindicated [31]. In this case, on the one hand, the surgery is not associated with a beneficial result, and on the other hand, the shunt complications will be added.

Complications

Following the insertion of a ventriculoperitoneal shunt, several complications have been reported, including shunt malfunction, infection, subdural hematoma, seizures, migration to different organs or shunt kinking, and disconnection of the shunt components [21].

Following surgery, a small amount of blood is often seen within the ventricles, brain parenchyma, and catheter passing tract, but the occurrence of clinically significant symptoms is uncommon in these lesions [28]. Also, symptomatic intracerebral hemorrhages often occur shortly after surgery [39]. Different risk factors have been described for the occurrence of an intracerebral hemorrhage following a cerebral shunt insertion, including disseminated intravascular coagulation (DIC) caused by the shunt, vascular damage caused by the shunt passing through brain tissue, bleeding from the tumor or hidden vascular lesion, coagulation disorders, and brain trauma shortly after shunt insertion [40].

In the delayed hemorrhages, most probably the mechanism of bleeding is vascular injury and its wall erosion due to the pulsation transmitted by the shunt tube [28].

Other possible complications of using a shunt are over drainage, which may lead to a slit ventricle, and, in tumor cases, metastasis of tumor cells through the shunt tube may occur.

Over 50% of patients within two years after ventriculoperitoneal shunt placement experience shunt failure [17,41]. Most of these cases are caused by obstruction that occurs most frequently in the proximal part of the shunt (proximal to the pump) [9]. The shunt is usually inserted into the lateral ventricle through the burr hole created in the right parietal bone. The shunt tip is best placed in the lateral ventricle anterior to the foramen of Monro that lacks the choroid plexus. This consequently reduces the likelihood of growth and sticking of the choroid plexus to the holes in the shunt tube and, thus, prevents its obstruction; however, these adhesions lead to the occurrence of intraventricular hemorrhage during the removal and replacement of the shunt. The shunt tip placed anterior to the foramen of Monro reduces these problems.

Shunt dysfunction can lead to headache, nausea and vomiting, drowsiness, abdominal pain, irritability, and seizures. In children with a history of meningo(myelo)cele repair, swelling and pain may be seen in the surgical site. In infants, whose sutures and fontanelle are still not closed, the bulging fontanelle and increased size of head circumference may be observed. To examine the shunt function, observation of the emptying and filling of the shunt pump after finger pressing will be very helpful to determine dysfunction and also to locate the disorder. In normal mode by pressing on the pump, the pump can be easily emptied and slowly filled afterward. If the pump is not emptied by hand pressure or is emptied by high pressure, this means that the blockage has occurred in the distal or abdominal parts of the shunt. If the shunt pump is emptied easily and is filled with delay or is not filled, this means that the blockage may occur in the proximal or intracranial parts of the shunt. In such cases, all aspects of the shunt need to be checked in terms of kinking and disconnection of the shunt components. Given that the shunt tubes are visible on plain radiography; plain radiography can be employed for this purpose. Also, a brain CT scan can confirm shunt dysfunction by showing changes in ventricular size and their enlargement, as well as increased ICP symptoms, such as periventricular edema and the narrowing of subarachnoid cisterns. Further, MRI can help to diagnose increased ICP symptoms, intracranial bleeding or infection. The cine MRI application can determine CSF flow and possible obstruction.

Radioisotope methods can be recruited to evaluate shunt function. Technetium is used for this purpose. However, the injection should be done in the proximal tube of the shunt, and the injection in the pump cannot evaluate the proximal obstruction of the shunt because of the one-way valve in the pump [9].

The ventriculostrial shunt and lumboperitoneal shunt are rarely used. Ventriculoatrial shunt infection can lead to life-threatening complications, such as septic endocarditis, septic pulmonary emboli, and immune complex glomerulonephritis [9]. The lumbo(peritoneal) shunt is associated with a high incidence of herniation of the cerebellar tonsils, brain stem compression, and syringomyelia, as well as the possibility of leading to scoliosis and nerve-root deficit [9].

To reduce the complications associated with the use of shunts, the surgery should only be performed on patients with hydrocephalus who also have symptoms of increased ICP. Mild or moderate dilation of ventricles and mild ventricular enlargement following surgical repair of meningo(myelo)cele can be controlled without shunt placement [42].

Prognosis

Fundamentally, prognosis depends on the underlying condition causing hydrocephalus and duration of symptoms and treatment-related complications.

Increased intracranial pressure can lead to neuronal loss and brain damage [43], according to Laurence and Coates [44], if the natural course of hydrocephalus is not treated, the disease will lead to death within 10 years in 54% of the cases. They also noted that 62% of children who did survive had cognitive impairments. In child-ren who were treated, the results are far better. Shurtleff et al. [45], showed that the 10-year survival rate of these children was 90% and only 30% of them had cognitive impairments.
REFERENCES


38. Warf B. Comparison of endoscopic third ventriculostomy alone and combined with choroid plexus cauterization in infants younger than 1


