# Myelomeningocele Primary Repair Surgical Technique

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# CHAPTER 10
# Myelomeningocele Primary Repair Surgical Technique

Massimo Caldarelli, Concezio Di Rocco

## Introduction

A clear understanding of the pathologic anatomy of the spinal malformation is a fundamental prerequisite of the pre-operative work-up in spina bifida patients [1]. The malformed spinal cord or primitive neural plaque (placode) presents as a flat tongue of neural tissue with its borders merging into the contiguous malformed meningeal coverings. As an effect of the failed neurulation process, both ventral and dorsal spinal roots exit from the ventral aspect of the placode, the dorsal roots exiting laterally to the ventral ones, and corresponding to the boundary between the placode and the arachnoid membrane (junctional zone). The presence of an intact subarachnoid space ventral to the placode confirms the lesion as a myelomeningocele (MMC) (Fig. 10.1), whereas its absence confirms it to be a myelocele, which more closely resembles the deranged anatomy of failed neurulation (Fig. 10.2).

The residual function of the placode has been much debated [2]. Spontaneous movements are present in many myelodysplastic newborns, as well as in response to intraoperative electrical stimulation of the placode [3]. Likewise, somatosensory evoked potentials can be elicited within the placode by means of peripheral nerve stimulation. Cortical evoked responses can be recorded after placode electrical stimulation [2]. Furthermore, even in the absence of cortical control, the placode may still maintain intact local spinal reflexes that may contribute to bowel and bladder function [4]. All of the above considerations make it plausible that some residual functional neural elements are still present in the placode; these con-

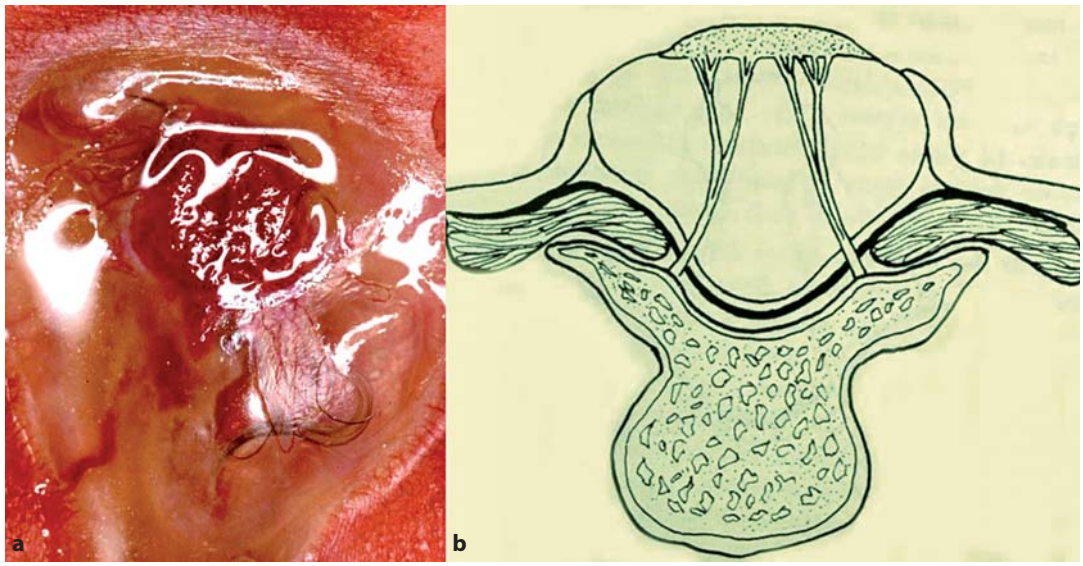


**Fig. 10.1 a, b.** Clinical appearance of a lumbar myelomeningocele (**a**) and schematic drawing (**b**) of the malformation, demonstrating the relationship of the placode with the subarachnoid space and cutaneous layers, and the exit and course of the spinal roots within the malformed sac

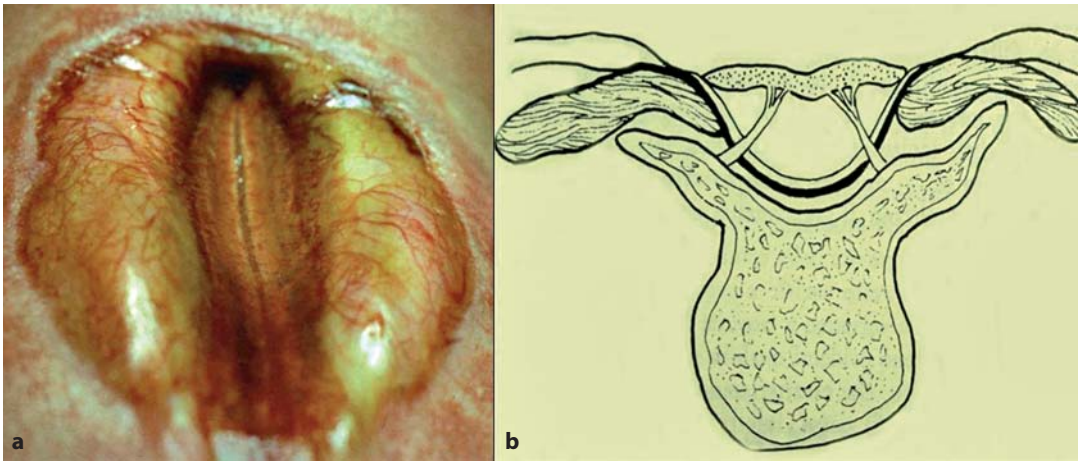


**Fig. 10.2 a, b.** Clinical appearance of a lumbar myelocele (**a**) and schematic drawing (**b**) of the malformation, demonstrating its relationship with the surrounding meningeal and cutaneous layers

sequently deserve special attention and care. From a practical point of view the placode must be handled with care to minimize the risk of harming that residual functional nervous tissue, and be protected from dehydration by covering it with a gauze dressing soaked in a sterile saline solution. The application of a plastic wrap over the gauze will help to keep the placode adequately moistened [5, 6]. The usefulness of systemic antibiotic therapy to prevent cerebrospinal fluid (CSF) infection is debated; conversely, local antibiotics or iodine-containing medications are to be avoided for their potentially adverse effect on the placode [5, 6].

## Timing of Surgery

MMC repair should be performed soon after birth provided that the newborn's general condition is good and signs of meningeal infection are absent. According to the literature the operation is usually performed within the first 48 hours of life [2, 5-9]. Such delay, whilst not deleterious for neurological function nor for increasing the risk of CSF infection, allows the neurosurgeon to obtain more comprehensive information on the child's clinical condition (including thorough neuroradiological investigation), and for the parents to become better acquainted with the problems related to the malformation in order to give adequately informed surgical consent [10, 11]. Surgery should not be delayed beyond 72 hours of life, as it has been demonstrated that after this time there is a 37% risk of ventriculitis, compared to only 7% when operated upon in a more timely fashion [5, 9]. A delay in surgical repair also exposes the myelodysplastic newborn to the risk of deterioration in neurological and bladder function [12].

At present, surgical repair is generally performed at an early stage. Since MMC is detected prenatally in up to 90% of cases, the spina bifida team has usually already been alerted prior to birth, and the parents usually sufficiently informed of the various clinical and surgical aspects of the spinal malformation, as to make their consent to surgery readily available soon after birth [2, 5-8, 11]. Due to prenatal diagnosis and counseling, the birth of a child affected by MMC is a planned event in the vast majority of cases (Fig. 10.3). There is still enduring debate as to the best means to deliver a child with myelomeningocele, namely, whether a cesarean section reduces the risk of neurological dysfunction [13-15]. Without addressing specific obstetric issues, these children are usually delivered at term, or slightly before term, after reaching pulmonary maturity, by means of a planned pre-labor cesarean section [11, 12, 15]. As the team responsible for the initial care of the myelodysplastic newborn (neonatal intensivist, anesthesiologist, neurosurgeon) has already been mobilized prior to delivery, the time interval to surgical repair is presently much shorter than previously necessary. At our institution, cesarean section is the modality of choice to deliver a fetus with a prenatal diagnosis of open spinal dysraphism, even in cases theoretically amenable to vaginal delivery [16]. Consequently, when a fetus with MMC reaches term, a cesarean section is planned to be performed early in the morning in order to have the

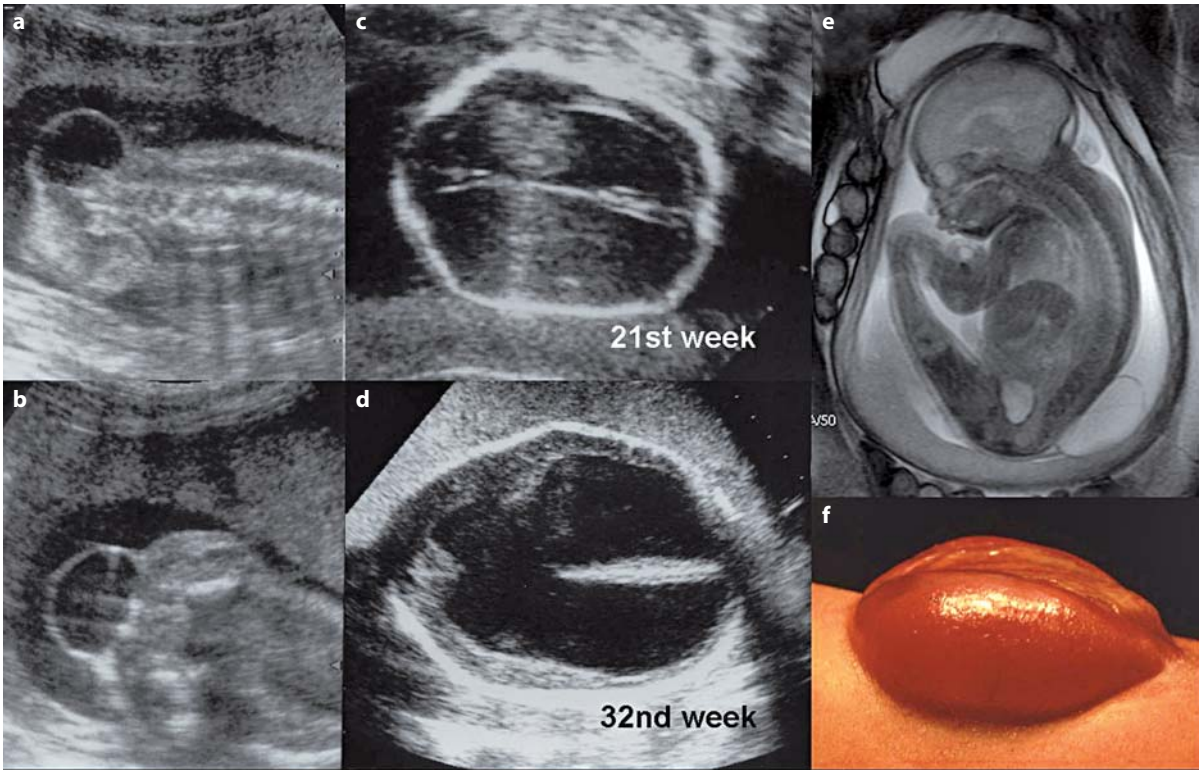


**Fig. 10.3 a-f.** Prenatal diagnosis of myelomeningocele. **a, b** Ultrasound demonstration of the spinal defect (AP and lateral views) **c, d** and evolution of the ventriculomegaly from the 21st to the 32nd gestational week. **e** T2-weighted fetal MRI demonstrating the spinal malformation; note the absence of ventricular dilatation at this stage. **f** Clinical appearance of the malformation at birth

newborn ready for neurosurgical closure early in the afternoon, as long as the newborn's general condition does not require further investigation. Under such circumstances the spinal defect is repaired within the first 6-12 hours of life, i.e., much earlier than as described in the literature.

On the other hand there are still cases, fortunately increasingly rare, of children affected by MMC who are delivered without a prenatal diagnosis and who are consequently sent to a tertiary hospital only hours or days after birth. In these circumstances, mobilizing the team, acquiring all clinical data as well as giving the parents adequate information and obtaining their consent to the operation, may require more time.

Whenever the operation is delayed beyond 72 hours of life, either due to delayed parental consent or to delayed referral from a peripheral hospital, it seems appropriate to obtain CSF cultures prior to undergoing surgical repair. When these are positive the newborn should be treated with antibiotic therapy and definitive closure delayed until the infection is cleared.

## Early Management of Hydrocephalus

Hydrocephalus is so frequently associated with MMC (85-90%) as to be considered part of the malformation. As largely debated in the literature, many factors may contribute to its occurrence, namely aqueduct stenosis, fourth ventricle outlet obstruction, and obliteration of the posterior fossa subarachnoid spaces or their obstruction at the tentorial notch (Fig. 10.4) [17, 18]. It is this great variety of pathogenesis that accounts for the different modalities of clinical presentation and their different ages of onset. In fact, in less than 15% of the cases hydrocephalus is already overt at birth, manifesting with the classical signs of raised intracranial pressure (ICP) (split sutures, tense anterior fontanel, sunsetting eyes, vomiting, etc.) or even with the life-threatening signs of brainstem dysfunction (poor feeding; poor sucking and swallowing; nasal regurgitation; repeated coughing; weak or high-pitched cry; stridor; apneic spells; pneumonia, etc.), secondary to the impaction of neural

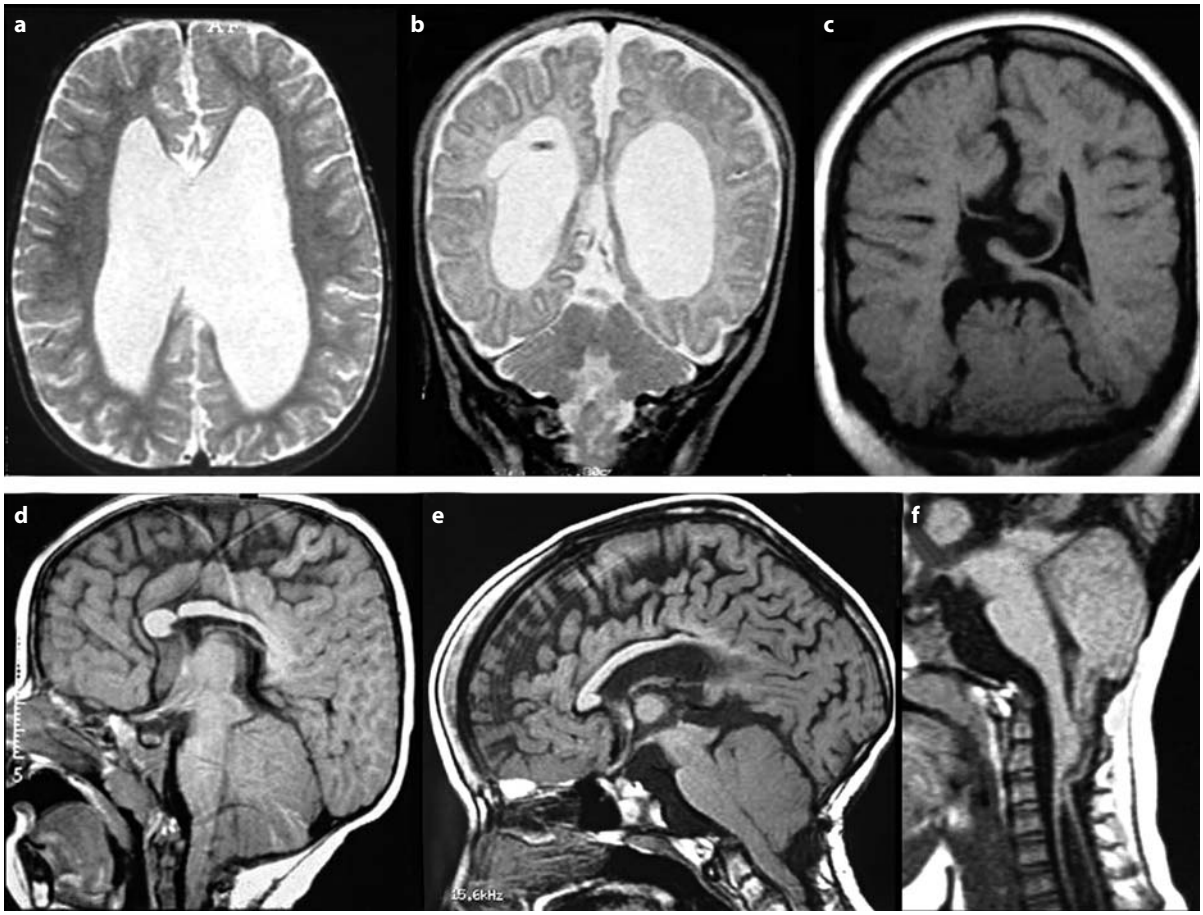


**Fig. 10.4 a-f.** Radiological features of hydrocephalus associated with myelomeningocele. **a** T2-weighted axial and **b** T2-weighted coronal views demonstrate a huge ventricular dilatation with disproportionately enlarged occipital horns. **c** Post-operative T1-weighted coronal view shows the reduction in ventricular volume and the abnormal shape of the midline cerebral cortex. **d-f** T1-weighted sagittal images demonstrate the small posterior fossa and the descent of the cerebellar tonsils and inferior vermis into the spinal canal, as well as the upward herniation of the superior vermis associated with other typical aspects of the Chiari II malformation (beaking of the tectum; thinned and malformed corpus callosum; large massa intermedia; stenogyric appearance of the occipital cortex; etc). **f** Note also the associated hydromyelia

structures within the small posterior fossa (due to the Chiari II malformation). This particular subset of myelodysplastic newborns with significant hydrocephalus warrants early surgical treatment [2, 7].

Among the presently available surgical modalities for treating hydrocephalus, ventriculo-peritoneal (VP) shunting remains the treatment of choice. Standard VP shunting not only ensures immediate relief of intracranial hypertension but is also beneficial to spinal wound healing by avoiding CSF pooling and leakage at the site of surgical repair [2, 5, 7, 8]. There has been some debate in the literature as to the best site for positioning the ventricular catheter, i.e., frontal or occipital. Authors almost equally suggest either frontal [19] or occipital [20, 21] positioning as associated with a lower percentage of mechanical shunt malfunctions. We utilize the occipital route almost exclusively. The main reason for our choice is the asymmetrical ventricular dilatation, with disproportionally large occipital horns and relatively small frontal horns, which is typical of the hydrocephalus associated with MMC. This asymmetrical enlargement also minimizes the risk of the ventricular catheter coming in contact with and becoming occluded by the choroid plexus. Furthermore, tunneling of the shunt system to the parietal region is easier and the maneuver requires fewer skin incisions than placing the ventricular catheter frontally.

There is no general agreement as to the most appropriate timing of the two surgical procedures (MMC repair and VP shunt), should they need to be performed at the same stage. The question is obviously limited

to the small percentage of myelodysplastic newborns that require immediate treatment of the associated hydrocephalus; in fact, in the vast majority (more than 85% of cases) treatment of hydrocephalus is usually postponed for weeks to months following MMC repair. When the hydrocephalus requires urgent treatment, many reports in the literature underline the advantages of unifying MMC repair and VP shunting at the same procedure [22-24]. Usually the newborn undergoes insertion of the VP shunt first, and then MMC repair. The advantages derived from combining the two operations are the rapid relief of intracranial hypertension and its beneficial effect on spinal wound healing. One study indicates that there is no significant difference in the infection rate between patients undergoing the two operations at the same stage or separately, provided that they are performed within the first 48 hours of life [25]. We too have adopted that policy on occasions; however, in our experience the association of MMC repair and CSF shunting in the same surgical procedure has been marked by a higher incidence of infectious shunt complications, compared to the cases where the two operations were performed separately [26]. This result has caused us to be more cautious and frankly reluctant to combine the two procedures. In addition, the problem seems overestimated as, in our experience, cases of hydrocephalus requiring such immediate treatment are quite rare. Given that most of the observed infectious complications in our patients were demonstrated to be due to a preexisting CSF sub-clinical infection, in the limited number of patients with significant hydrocephalus requiring prompt neurosurgical treatment, we have adopted the policy of performing temporary external ventricular drainage contemporaneously to MMC repair. In patients where CSF infection is confirmed, the same route can also be utilized to administer intraventricular antibiotics until resolution of the infection. In conclusion, our present position is to no longer treat hydrocephalus and spinal dysraphism at the same stage, but rather to perform MMC repair first as an independent procedure. As soon as clinical signs of intracranial hypertension occur, or if there is local pooling of CSF at the site of the spinal wound, we proceed to VP shunting. In our experience, postponing VP shunting in this manner neither increases the risk of infection nor alters the final neurological outcome.

Recent reports in the literature have proposed endoscopic third ventriculostomy (ETV) as an alternative to VP shunting in children affected by MMC [27-29]. However, these same reports, whilst indicating a relatively high success rate in cases of "secondary" ETV (i.e., in those myelodysplastic children presenting with VP shunt malfunction), underline a poor success rate in cases of "primary" ETV. Furthermore, the young age of the patient, which is typically a contraindication to ETV, acts as a further adverse factor in myelodysplastic newborns [27, 29]. The only report dealing with a significantly high population of myelodysplastic patients reflects the same considerations, i.e., only 29% success in cases of "primary" ETV and 12.5% success rate in children less than 6 months old [27]. We perform ETV in myelodysplastic children as either a primary or secondary procedure. Our preliminary results [30] have been more positive than those previously reported in the literature, with a significantly higher success rate of "primary" ETV in myelodysplastic newborns. However, further investigation is warranted.

## Myelomeningocele Repair

Aims of the surgical treatment of MMC are: (1) to remove the malformed sac; (2) to prevent central nervous system infection by creating a barrier between the spinal canal and the exterior; and (3) to restore the normal CSF environment around the malformed spinal cord, thus preserving its residual motor and sensory functions. These results may be obtained by means of reconstruction of the placode, and multilayer closure of the meningeal, fascial, subcutaneous and skin layers; in other words, surgical repair should complete the interrupted neurulation process [2, 6, 7, 31, 32].

Contemporary surgery of MMC requires magnification (both operative microscope and loupes); with optional laser and intraoperative electrical stimulation [31, 32]. Utilization of the operative microscope enables the neurosurgeon to perform the surgery in a safer way than previously possible, especially during neural structure manipulation. Such technical tools have allowed neurosurgeons to obtain better functional results as demonstrated by the frequent observation of postoperative neurological improvement [6].

As myelodysplastic children are prone to develop latex allergy it is appropriate when planning the surgical repair to utilize a latex-free setting, if such a dedicated facility is already available in the hospital, or to prepare a similar surgical environment by sterilizing the operating room with regard to latex proteins and by utilizing only latex-free products [33].

### Anesthesia and Positioning of the Patient

Although local anesthesia associated with mild sedation has been utilized in the past, at present the operation is performed only under general anesthesia

with orotracheal intubation [34]. During pre-operative anesthetic procedures (positioning of venous and arterial lines, bladder catheterization, orotracheal intubation) the newborn is positioned supine over a gauze or jelly donut to protect the spinal malformation. After completing the anesthetic work-up, the newborn is positioned prone on firm chest rolls to allow optimal thoracic expansion; pads are positioned on all pressure points to avoid pressure sores. We usually place the newborn in an inverted position, with his/her back in front of the operator. A mild Trendelenburg position is suggested to avoid CSF escape from the spinal canal and the risk of pneumocephalus. During these procedures the body temperature must be strictly regulated, to avoid the deleterious effects of hypothermia.

## Skin Preparation and Draping

An adequate amount of the skin surface around the spinal defect is bordered with plastic wraps, proportional to the diameter of the malformation and to the amount of skin to be presumably mobilized to optimize skin closure. Such initial definition of the surgical field reduces the surface to be prepared and consequently reduces heat loss. It also assures a separation of the anal and perineal regions from the surgical field, thereby minimizing the risk of infectious complications. Subsequently the skin is cleansed with Povidone iodine solution, paying particular attention to avoid any contact with the placode. After completing the skin preparation, the placode is simply cleaned with saline or Ringer's solution.

The operative field is subsequently covered in the usual fashion with an adhesive plastic drape in which a central hole corresponding to the inner part of the malformation has been fashioned and then bordered with drapes, again paying attention to leave an adequate amount of skin around the malformation (Fig. 10.5) [35].

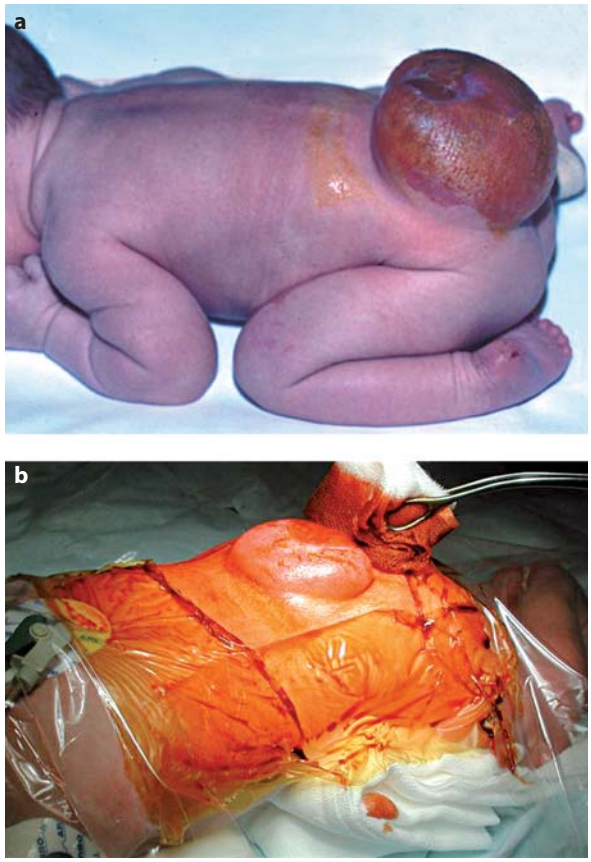


**Fig. 10.5 a, b.** Newborn infant with a large lumbar myelomeningocele. **a** Patient positioning for surgical correction. Definition of the surgical field by means of adhesive drapes and skin preparation with respect to the placode

## Planning the Surgical Procedure

As with any other surgical procedure, MMC repair should be accurately planned, with the specific characteristics of the affected child taken into consideration. Inspection of the spinal malformation allows the appreciation of the site, extension and characteristics of the lesion, as well detection of any associated spine deformity that may influence surgical management [2, 6-8]. For example, kyphosis that is present at birth in about 15% of the affected children may prevent, although very rarely, perfect closure of the spinal malformation and thus require correction at the same time as the MMC repair [36]. Similarly, severe kyphoscoliosis associated with significant asymmetry of the lower limbs suggests an associated split cord malformation that may alter the surgical strategy [37, 38].

A further fundamental aspect of pre-operative neurosurgical evaluation is appreciation of the quality and amount of the skin available for reconstruction. In fact, skin closure may be, on some occasions, the most difficult aspect of the surgical procedure. For example, in large lumbosacral myeloceles where the skin defect is significant, and the subcutaneous tissue scarce, reconstruction of the superficial layers is almost impossible, and plastic surgical reconstruction becomes necessary. On the other hand, a large lumbar MMC with redundant skin covering the defect poses no problem, but the excess tissue needs to be removed. In these

cases it is advisable to delay the sacrifice of the redundant skin until all the other cutaneous layers are closed.

Apart from these considerations, a perfect knowledge of the pathologic anatomy of the spinal malformation is mandatory in order to correctly manipulate the placode during dissection and reconstruction (Figs. 10.1 and 10.2) [1]. It is known that the placode retraces the primitive neural plaque, with its edges continuing into the contiguous arachnoidal and dural coverings (junctional zone). As an effect of the failed neurulation, both ventral and dorsal spinal nerve roots exit from the ventral aspect of the placode (the dorsal roots exiting laterally to the ventral ones), at the border between the placode and the adjacent arachnoid layer. It is important to recognize this region in order to avoid injury to the nerve roots during the initial dissection.

## Skin Incision

A limited midline linear skin incision is performed at the upper limit of the malformation, over the spinous processes of the first two normal vertebrae rostral to the spinal defect. Thereafter the incision is brought along the border between the dystrophic skin and the arachnoid that surrounds the malformation (Fig. 10.6), and circumferentially until the entire placode (with its incomplete arachnoid margin) is completely freed; thus saving all available skin so that it can be utilized for the final reconstruction of the superficial layers. Finally a further midline linear incision is performed at the lower extremity of the spinal defect. These two supplementary vertical incisions help to identify the normal elements rostral and caudal to the malformation. Identification of nor-

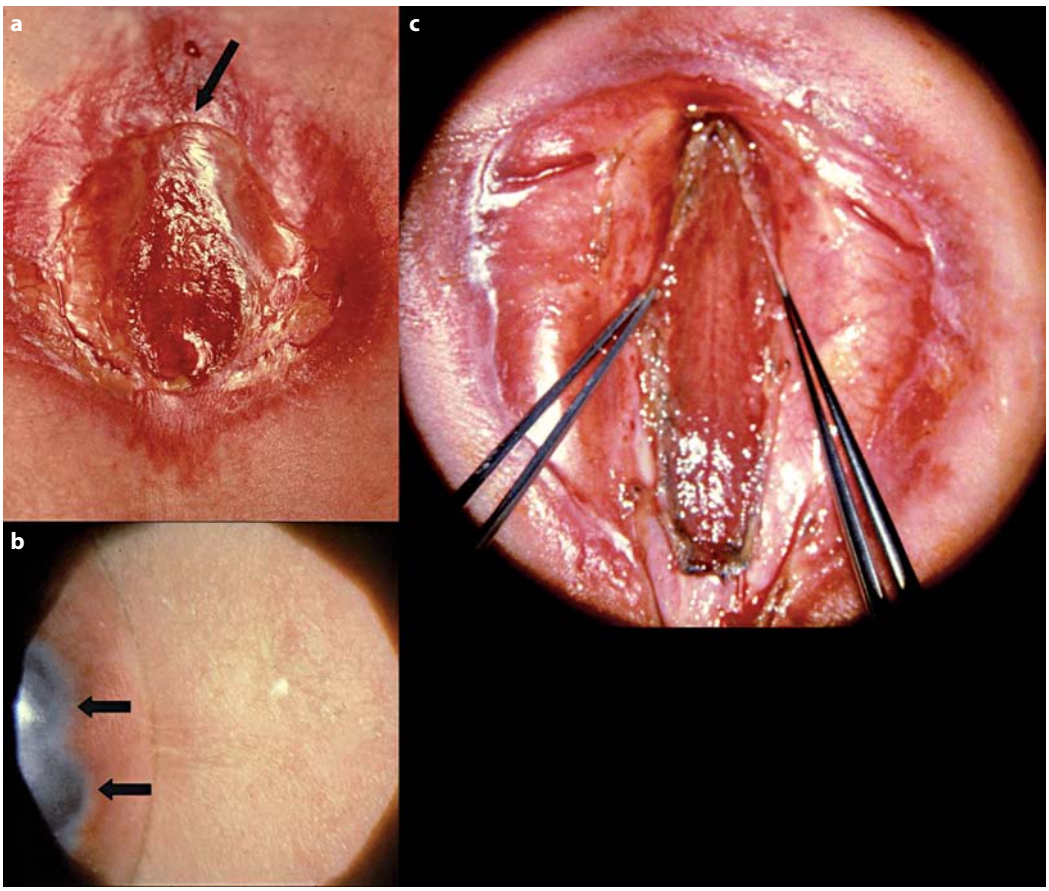


**Fig. 10.6 a-c.** Initial steps in the surgical repair of the myelomeningocele (under microscope magnification). **a** The placode with its arachnoid membrane is circumferentially connected to the cutaneous layers; the arrow indicates the site of the initial incision. **b** Under further magnification the site of the initial incision is indicated (*arrows*) at the edge of the arachnoid membrane (junctional zone). **c** After completing placode dissection, the margins of the malformation can be approximated in the midline

mal dura rostral to the malformation is fundamental for the safe dissection of neural structures. The scalpel is preferred to the Colorado needle for the skin incision. The latter, which is so helpful in minimizing blood loss in many surgical procedures, should be avoided as it may necrotize the already dystrophic skin margins, thus interfering with the healing process.

## Placode Dissection

After completing the skin incision, the placode is dissected free from the surrounding arachnoid remnants along the junctional zone under microscope (or loupe) magnification [6, 32, 39, 40]. Dissection usually begins at the upper limit of the malformation, where the placode fuses with the normal spinal cord, and proceeds circumferentially along its borders. As already underlined, the dorsal root entry zone is immediately adjacent to the border of the spinal malformation. This relationship places them at risk of inadvertent injury; and care must be taken when manipulating the border of the placode. Any dystrophic arachnoid remnants should be meticulously removed, utilizing wherever possible sharp dissection, as they may contribute to late tethering of the spinal cord [2, 5, 7, 41-43]. Likewise, the accurate removal of any epidermal or dermal remnants is extremely important since they are potentially responsible for delayed dermoid/epidermoid cyst formation [40, 41, 44]. These maneuvers are best performed with microdissectors and microscissors, which allow more delicate manipulation of the neural structures (Fig. 10.6). Bipolar coagulation should be as limited as much as possible during this step of the surgical repair.

After completing the circumferential dissection, the filum terminale is usually identified beneath the most caudal portion of the placode. Section of the filum terminale (whenever clearly identifiable) is an integral part of the procedure as doing so minimizes the risk of secondary tethering [2, 5, 7, 8, 31, 40-42]. Notably, there are other anatomical variants that favor late tethering of the spinal cord, namely anomalies in the shape and length of the spinal roots, which cannot be surgically corrected [43].

Inspection of the inner aspect of the open dural sac often reveals the presence of aberrant nerve roots that terminate in the dural sac; these neural elements are devoid of any functional activity and can be divided without the risk of harming the neurological status of the child.

Frequently, relatively large vessels are encountered that enter the ventral aspect of the placode. These vessels must be manipulated carefully and dissected free from arachnoid adhesions in order to optimize their mobilization during the following phase of placode "tubulization". It is essential that any injury or coagulation of these vessels is avoided as their thrombosis may interfere with placode vascularization and viability, thus resulting in further neurological damage [40, 41]. As indicated above, coagulation should be avoided; and as such, hemostasis should preferably be obtained by local application of Spongostan or Flo-seal.

Before proceeding with the following steps, an accurate inspection of the intradural space should rule out any other associated malformations such as lipomas or dermoids that also deserve surgical treatment.

## Neural Tube Reconstruction

Once dissection has been completed, the placode will lie at the bottom of the widely open dural sac, continuing rostrally with the intact spinal cord, and with the spinal roots emerging ventrally to it. The following step of surgical repair is represented, whenever possible, by an attempt to "reconstruct" the spinal cord. To this end the lateral edges of the placode are approximated in the midline, and their pia-arachnoid borders are sutured under microscope magnification, with a 7.0 non-absorbable monofilament (Fig. 10.7) [6, 31, 32]. This maneuver will transform the malformed placode into a structure resembling the primitive neural tube, completely invested by normal leptomeningeal coverings, thus completing the failed neurulation process. Care should be taken during this phase to avoid any compression to the neural structures originating from an excessively tight closure. Should the placode be too bulky to allow its "tubulization", it is advisable to abandon this part of the surgical procedure. We are aware that placode reconstruction is potentially devoid of any beneficial effect on the child's neurological outcome; nevertheless, it usually facilitates dural closure and minimizes the arachnoid scar by reducing the area of dorsal adhesion. It is widely accepted, and it is also our experience that pia-arachnoid suture significantly reduces the occurrence of symptomatic late tethering [6, 10, 31, 40, 43].

Unfortunately, too large a placode or too flat a spinal canal may risk compression of the reconstructed spinal cord at the time of dural closure. In these cases utilization of a dural patch for dural repair will usually resolve the problem.

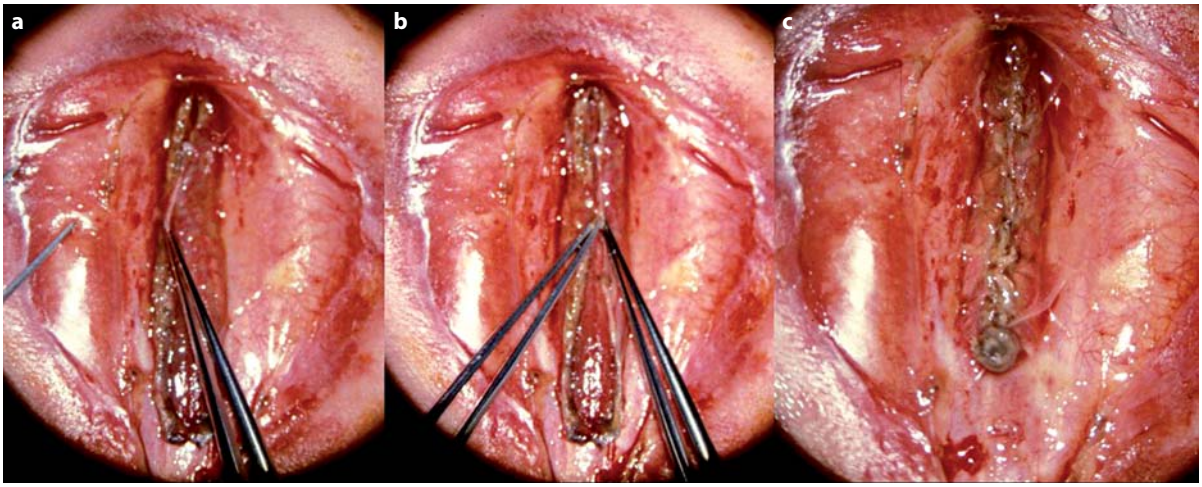


**Fig. 10.7 a-c.** Surgical repair of myelomeningocele: closure of the placode. **a, b** Under microscope magnification the placode has been detached from the surrounding arachnoid and cutaneous layers, and its margins approximated in the midline to allow **c** a pial-to-pial suture of its dorsal aspect with a 7.0 monofilament

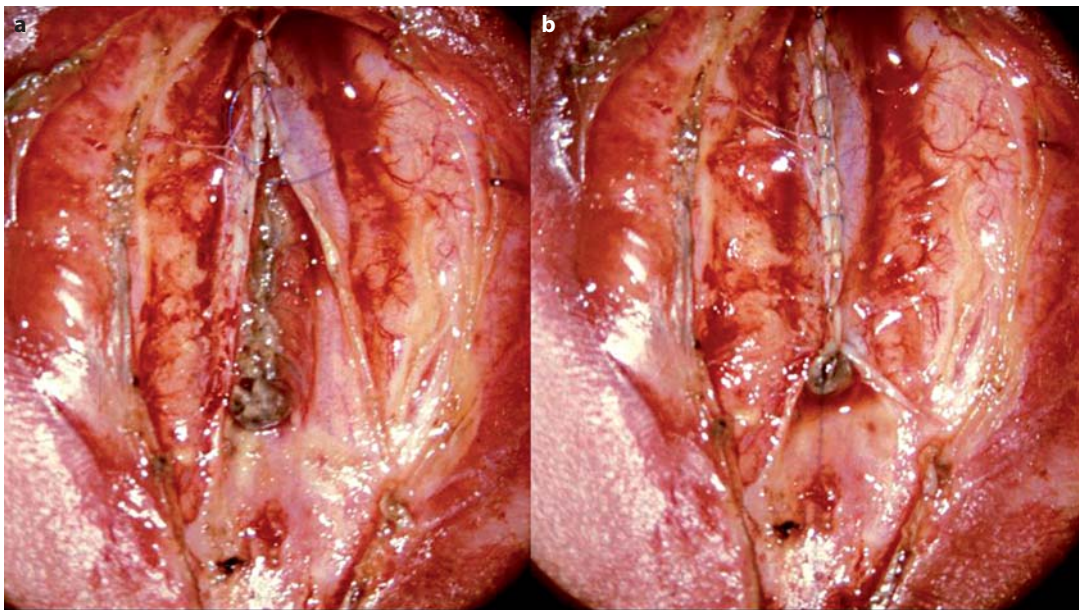


**Fig. 10.8 a, b.** Surgical repair of myelomeningocele: dural closure. **a** Two dural sheets have been sharply separated from the underlying lumbar fascia under microscope magnification, and approximated in the midline. **b** The dural edges are being sutured on the midline paying attention to avoid tension on the underlying neural structures

## Dural Dissection and Closure

The following step of surgical repair consists of reconstruction of the dural sac. The intact meningeal coverings must first be identified at the upper extremity of the malformation, just beneath the first normal vertebra, where the malformed dura continues into the normal dural sac. Starting from this point the dural layer is dissected circumferentially along the borders of the defect, proceeding centripetally from the periphery to the midline (Fig. 10.8a). Although preferable, sharp dissection alone does not always enable the development of a dural layer. Isolation of the dura is more difficult at the edges of the defect where the dura fuses with the thora-

columbar fascia. Dissection, therefore, should start close to the midline where a true epidural space is already present and the small amount of epidural fat facilitates dissection in the correct plane. We recommend using a scalpel for the initial circumferential incision, and subsequent sharp dissection for completing the development of the bilateral dural flaps utilized for dural sac reconstruction.

The newly developed dural sheets are then approximated on the midline and sutured with either 5.0 silk suture or monofilament (Fig. 10.8b). Occasionally dural flaps may be so large as to permit a double layer suture. Continuous suture is generally preferable as it assures better impermeability of the reconstructed dural sac; occasionally interrupted sutures may be used when the size of the meningeal flap is insufficient.

As in the previous surgical step, dural closure should avoid any compression to the underlying neural structures. In fact, too tight a dural closure may compromise the blood supply to the reconstructed placode and facilitate its adhesion from its dorsal aspect to the dural envelope, representing the basis for secondary re-tethering. To avoid this potential complication, a dural patch should be used when the dural sheets appear insufficient.

Dural patching may be performed using various dural substitutes, the most physiologic being autologous tissues, such as muscle and fascia. In this case a sufficient amount of thoracolumbar fascia is dissected over the child's back and separated from the underlying muscles by sharp dissection. The newly developed fascial flap is then interposed between the insufficient dural sheets and sutured to cover the dural defect. The patient's anatomy may not allow for such autologous grafting, thus making it necessary to utilize allografts such as cadaveric dura or bovine pericardium [45]. Unfortunately, these substitutes share the same adverse effect, i.e., the development of arachnoiditis resulting in late tethering. Synthetic materials such as silicone have similar adverse effects. A recent innovation is the new dural substitute composed only of colloidal collagen (TissuDura), which acts as a non-porous biomatrix for dural regeneration by triggering the formation of new extracellular collagen and rapid fibroblast migration. One of the most positive aspects of this material is the limited host inflammatory response [46, 47]. We have been using TissuDura over the last 3 years and have found that the patch ensures adequate dural closure and protection against CSF leak [48].

Dural closure should be water-tight to avoid postoperative CSF leak or even CSF pooling at the surgical site, which retards healing of the spinal wound [49] and exposes the newborn to the risk of infectious complications. To verify water-tightness a Valsalva maneuver should be performed after completing the dural suture. The presence of even a minimal CSF leak warrants further sutures or the use of dural glues.

Dural closure should be reinforced, whenever possible, by suturing the thoracolumbar fascia over the reconstructed dural sac. To this end the fascial borders are approximated and sutured over the midline defect, with the aid where necessary of lateral relaxing incisions (Fig. 10.9). Frequently this is not possible due to the width of the spinal defect; in this case, two fascial flaps are dissected from the underlying thoracolumbar muscles in the same fashion as for the dural flap, starting from the posterior iliac crest (or even more medially if less amount of tissue is needed). Of note, the sacral attachment of the fascia should not be incised [31]. Occasionally these fascial flaps can be integrated with periosteal flaps by dissecting the periosteum from the lumbar pedicles and transverse processes [50]. These fascial flaps are then approximated and sutured in the midline to protect the neural structures.

## Subcutaneous Dissection and Skin Closure

The following step of MMC repair is the development of the subcutaneous layer and skin closure. Dissection of the superficial layers from the fascial plane is performed by simply undermining the skin (often solely utilizing digital dissection) all around the just closed spinal defect. Attention must be paid to avoid excessive coagulation of perforator vessels to the skin that may compromise the blood supply of the cuta-

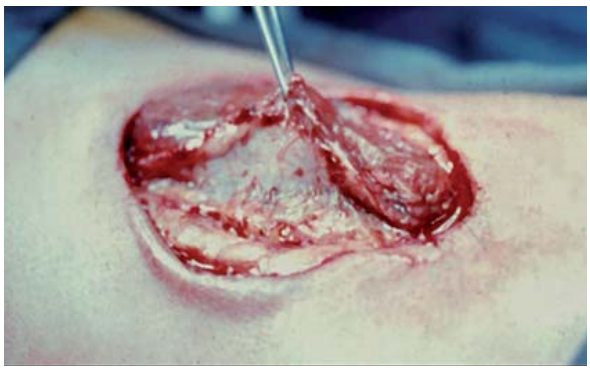

**Fig. 10.9.** Surgical repair of myelomeningocele: dissection and suture of the fascia. Two fascial flaps have been dissected and approximated in the midline to provide further protection to the dural suture

neous coverings [51-53]. The subcutaneous layer is usually minimal at the border of the malformation, becoming thicker beneath the junctional zone. For this reason, we place subcutaneous sutures at this level, to approximate the suture margins without excessive tension. Alternatively, in children with very large defects, sutures are placed 2-3 cm away from the skin edge and anchored to the underlying fascia with the same result of approximating the suture margins while reducing tension at the suture site. These "stay sutures" facilitate subsequent skin closure. The interposition of an adequate subcutaneous layer beneath the superficial skin layers is very important to reduce the incidence of unpleasant and potentially deleterious retracting scars. Initially, following closure, the skin may be blanched as a sign of tension. Such initial discoloration usually improves rapidly and wound dehiscence rarely occurs. A nitroglycerine ointment has been suggested to be helpful in some cases [54].

While perfect approximation of suture edges is always possible in cases of well-epithelized MMC with redundant skin, this is not the case when the surgeon is faced with large flat myeloceles with deficient cutaneous layers that will require a more complex plastic surgery reconstruction.

Once the subcutaneous layer has been reconstructed, the skin margins are debrided and closed in the habitual fashion with the non-absorbable sutures generally preferred when there is residual skin tension. Skin closure should be performed in a midline vertical fashion, where possible, with skin edges having little or no tension (Fig. 10.10). Occasionally a horizontal or oblique suture may be needed.

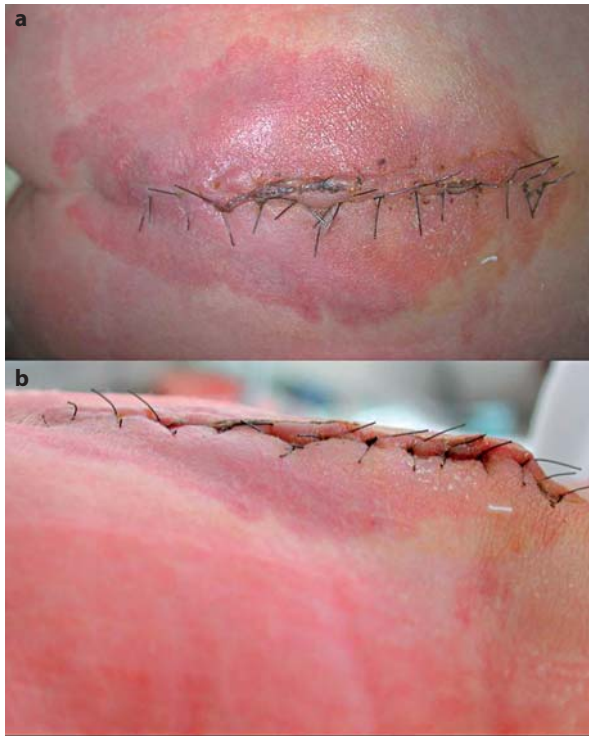


**Fig. 10.10 a, b.** Surgical repair of myelomeningocele: skin suture. The skin has been undermined circumferentially, approximated in the midline and sutured in a linear fashion

## Myocutaneous and Cutaneous Flaps

As already mentioned, the skin defect and adverse patient anatomy may prohibit skin closure in the usual fashion [55]. The kind of skin closure performed is determined by the width of the spinal defect, with one study suggesting that an area of skin deficit larger than 20-25 square centimeters is an indication for plastic surgical reconstruction [56]. In such cases, a variety of cutaneous and myocutaneous flaps have been designed to ensure adequate protection to the neural structures. They are discussed in Chapter 15.

Tissue expansion has been proposed to enable sufficient skin harvesting for MMC closure [57, 58], particularly in cases of delayed referral and closure. This has less applicability to newborns with MMC [59].

The various modifications of both cutaneous and myocutaneous flaps that can be found in the literature suggest that an ideal solution to the problem of skin coverage in MMC surgery is still lacking. Moreover, there is no general agreement on the true necessity for these sophisticated techniques. In general, neurosurgeons tend to perform direct skin closure, whereas pediatric surgeons are more prone to utilize plastic/reconstructive techniques. Some experienced pediatric neurosurgeons [31] refute the necessity for plastic/reconstructive techniques even in large defects. Our personal experience is in favor of this point of view, as only two out of more than 600 cases operated over a 30-year period required plastic surgery.

## Postoperative Course

After completing skin closure, the wound is cleansed again and covered with sterile gauze. The anus and perineal area are kept separated from the wound dressing by the interposition of an adhesive plastic drape that limits the contact of the wound with urine or fecal material. The newborn is usually observed in the neonatal ICU for the first 1-2 postoperative days, for apnea and/or any other sign of brainstem

dysfunction. The child is maintained prone with the lower back slightly elevated above the level of the head to reduce the risk of CSF leak from the wound. Should CSF pool beneath the wound or leak through the suture, immediate management of hydrocephalus should be undertaken. Prophylactic intravenous antibiotics are given either for the first 24 hours postoperatively or for a longer time (in our experience 5 days or more if there is significant risk of infection). The wound dressing is changed every 48 hours, or anytime it is soiled. If non-absorbable sutures have been used we remove these on the tenth postoperative day, a little later than is usual for other neurosurgical procedures.

Operative mortality is practically absent while morbidity may be significant [41, 42]. The most frequent complication is wound breakdown usually secondary to CSF leak, which is known to be an adverse factor for wound healing [49]. Conversely, wound infection is a much rarer complication of MMC repair, occurring in less than 2% of procedures [42]. It is managed by changing wound dressing and intravenous antibiotics. The most severe, though rare, complication of MMC surgery is meningitis with sepsis, which remains the main cause of death in these newborn children. Intravenous antibiotics are the treatment of choice, and ultimately CSF shunt removal if already implanted.

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