In utero repair of myelomeningocele (MMC) in humans spares distal neurologic function, reverses the hindbrain herniation component of the Chiari II malformation (ACM), and reduces the rate of postnatal shunt placement.

A study tested whether delayed in utero repair of such evolving experimental MMC lesions spares neurological function. In 12 sheep fetuses, a spina bifida-type lesion with exposure of the lumbar spinal cord was created at 75 days' gestation (full term, 150 days). Four weeks later, the developing MMC lesions were repaired in utero for seven fetuses (five fetuses died before this time). Of those that had repair, three were delivered near term by cesarean section, and four died in utero or were aborted. All survivors had healed skin wounds and near-normal neurological function. Despite mild paraparesis, they were able to stand, walk, and perform demanding motor tests. Sensory function of the hindlimbs was present clinically and confirmed electrophysiologically. No signs of incontinence were detected. Histologically, the exposed and then covered spinal cord showed significant deformation, but the anatomic hallmarks as well as the cytoarchitecture of the spinal cord essentially were preserved. These findings show that timely in utero repair of developing experimental MMC stops the otherwise ongoing process of spinal cord destruction and “rescues” neurological function by the time of birth. Because there is evidence that a similar secondary damage to the exposed neural tissue also occurs in human MMC, Meuli et al. propose that in utero repair of selected human fetuses might reduce the neurological disaster commonly encountered after birth [1)].

1)
Meuli M, Meuli-Simmen C, Yingling CD, Hutchins GM, Timmel GB, Harrison MR, Adzick NS. In utero repair of experimental myelomeningocele saves neurological function at birth. J Pediatr Surg. 1996 Mar;31(3):397-402. PubMed PMID: 8708911.