ABSTRACT

Title:

ELECTROPHYSIOLOGY VERSUS CLINICAL OUTCOMES OF TETHERED CORD SURGERY ASSISTED WITH INTRA-OPERATIVE MEP AND SSEP

Authors

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Introduction:

The tethered cord syndrome (TCS) is an uncommon disease caused by an abnormal stretching of the spinal cord, reported incidence of 0.05 to 0.25 per births untethering of the tight filum terminale. Spinal dysraphism is associated with tethered cord, which includes malformations like myelomeningocele, lipomyelomeningocele, tight filum terminale, split cord malformation, and dermoid sinus. Once the symptoms have developed, the patient has a terthered cord syndrome (TCS). These symptoms may either be neurological, urological, orthopedic, or pain.

The goal of surgery is to detether the cord by disconnecting its aberrant tightening attachments, and to relieve the cord from continuous stretching forces. Tethering structures may consist lipoma, fibrous bands, non-functional nervous tissue, or a

dermoid sinus tract. Peroperatively it can be difficult to distinguish between functional neural tissue and these non-functional tethering structures. Permanent neurological complications were reported in about 4.5% of patients, and this was high as 10.9% when transient complications were taken into account. Intraoperative neurophysiological monitoring (IONM) may help to protect the functional nervous tissue and prevent postoperative neurological deficits. There is no evident consensus on the exact methods of multimodal (M) IONM that should be used during surgery for TCS.

The use of intraoperative neurophysiological monitoring (IONM) may contribute to the safety in tethered cord surgery. We present a series of 15 patients with a wide variety of morphology of spinal dysraphism. All patients were operated for reasons of a tethered cord with the preoperative use of IONM. The usage of IONM in the different age groups and its potential contribution to the safety of the procedure are investigated.

This will further aid in prognosticating the neurological outcome of patients undergoing TC surgery.

This study also helps us in evaluating the importance of IONM in tethered cord surgery.

Objective:

The objective of this study was to determine if intra-operative neurophysiological monitoring is helpful in identifying which patients will have worsening symptoms after surgery.

It is also to observe the motor response thresholds before and after de-tethering, helping in early identification of the motor response via neurophysiological monitoring in comparison to clinical motor scoring with qualitative prediction of SSEP (somatosensory evoked potential) and MEP (motor evoked potential).

Material & methods:

This was a prospective study involving patients with tethered cord associated with lipomyelomeningocele of age 6 months to 12 years old. This study is conducted in Hospital Queen Elizabeth Sabah from Jun 2011 to October 2013. The follow-up was done at the third month or sixth month from the date of discharge of the recruited patients, which ended on October 2013. The candidates that had underwent tethered cord release were included in the study, however those of age less than 6 months and older than 12 years of age and those children who has tethering of cord post myelomeningocele repair was excluded in the recruitement.

Parents needed to sign a consent form which has been prepared in either English or Bahasa Malaysia. To be aware of the usage of the monitoring device intraoperatively and knowing that the device has no side effects to the spinal cord neither has it

anything to do with change in the surgical treament it merely aids in identification of nerve tissue during the surgery

Free running EMG of selected lower limb muscles ; with the aid of subdemal needle electrodes inserted in the following muscles quadriceps (L2-4), tibialis anterior (L4-5), gastrocnemius (S1-2), additionally needles are also inserted at the external sphinter . These needles were inserted after patient anesthetized and secured with tape.

Two needle electrodes were placed on the head subcutaneously at the anatomical location C3 and C4 (EEG 10 -20 system). TES – MEP recordings were done prior to and during the course of surgical intervention for detection of functional neural elements .The monopolar probe is used applying voltage pulses of (50 – 100) mA prior to and after detethering.

The anesthetic technique preferably used was TIVA (total intravenous anesthesia) with remifentanil 0.2 – 0.5 microgram/kg/min infusion and 2 % propofol infusion. Neuromuscular blocking agents were not used after induction. However there were two cases that we could not proceed with TIVA inview of one case having renal disease stage 3 and the other patient

Patients were subjected for nerve conduction study prior to the operation and post operatively after 3 months or 6 months . Post operatively post op retethering was followed up with MRI spine after 3 – 6 months.

Data were recorded using a data collection sheet. The results of preoperative NCS intraoperative SSEP and MEP and post opererative 3 month and or 6 months NCS were recorded. Data were entered into SPSS software. Non parametric test was used for analysis because of small number of patients.

Results

A total of 15 patients in this study, age ranging from 6 months to 12 years old (mean, 5 years). Motor improvement occurred in 3 patients (14.3%) and improvement in bladder function in 2 patients (28.6%). No postoperative neurological worsening occurred. Follow up available for 15 patients ranging from three to six months post operative. Intraoperative MEP and SSEP done was inconsistent and with the MEP of 5 patients showed descriptively that there improvement in amplitude of the waveform and reduced in latency of conduction , however , there was no statistical significant correlation with SSEP changes and clinical outcome, the motor (p value =1.000) and bladder function improvement (p value= 0.500).The post operative nerve conduction test , urodynamic study and MRI lumbar was done , there was improvement in conduction amplitude and latency in 3 patients with correlation to clinical outcome however there was no statistical significance.

Conclusion:

NIOM during TCS surgery can be used not only to aid with safety of surgery but in prognosticating on the postoperative outcome of the patient. This will eventually help in reducing operative morbidity , thus giving a likely favourable outcome.

Dr. Pulivendhan Sellamuthu: Supervisor

Professor Dato’ Dr. Jafri Malin Abdullah: Co-supervisor

CHAPTER 1: INTRODUCTION

1.1 DEVELOPMENT OF SPINAL CORD

The process of spinal cord formation is driven by growth factors produced by notochord The development of notochord happens during the 16 – 17 days of development. It is formed by mesoblastic cells migrating rostrally to Hensen's node between ectoderm and endoderm. The notochord has always been also known as the embryonic inducer.

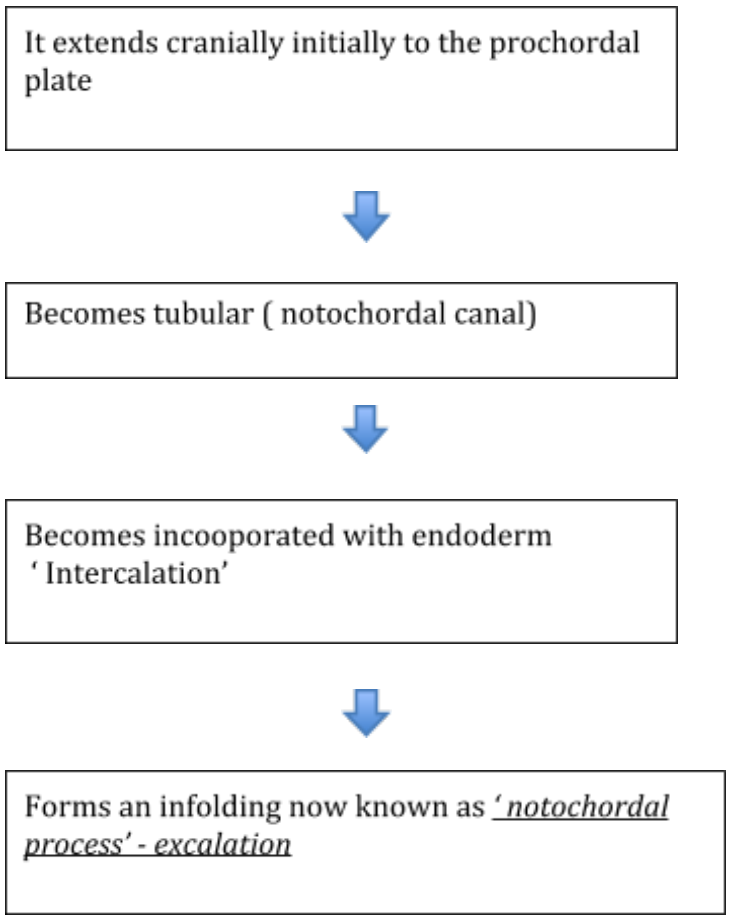

Figure 1: Flow chart showing the processes of notochord formation

Neural tube is formed via process call neurulation. There are 2 types

i) primary neurulation: it's a process of brain and spinal cord formation up to L1 – disorders that arise during this period represent the open spinal dyrspism
ii) secondary neurulation: process of spinal cord distal to L1 via canalization and regression – disorders during this period gives rise to closed spinal dysraphism

The process takes place when ectoderm thickens and forms neural plate , then there will be a longitudinal depression that develop[s in the neural plate . The depression is known as neural groove and the elevated sides are called the neural folds. These neural folds fused in the midline to form the ' neural tube'.

The neural; tube detaches from the cutaneous ectoderm and lie dorsal to notochord. There are 2 openings , the anterior pore that closes at the middle of the 4^{th} week and the posterior pore that closes at the end of the 4^{th} week at the level of L1 and/ L2

The caudal neural tube (including the tip of the conus medullaris caudal to the second sacral segmant , as well as the filum terminale) arises by secondary neurulation from the caudal cell mass or end bud (the remnants of the Hensen node and priitive streak) at the caudal pole of the embryo.

The ascent of the conus medullaris is where the conus will change position with respect to surrounding verebral column . This ascent begins at period of gestation of day 42 and continues throughout embryogenesis and perhaps even into the postnatal period.

Ascent occurs in 2 mechanisms . The first occurring between day 42 and 54, this is known as ‘ retrogressive differentiation’ , this is when the caudal end of the spinal cord becomes thinner and develops a rudimentary marginal and no matle zone. (Mark S dias)

Beyond day 54 retrogressive differentiation has ceased and elongating ascend of the spinal cord will be more slowly. The rate of ascent is steeper between gestational weeks 12 and 20 and slows down after term. According to Barson the conus medullaris at birth lies opposite the L2/3 disk space and ascends to its final level opposite the L1/2 disk space by 2 months postnatal.
(Batson AJ et al 1970). However several subsequent radiographic studies have demonstrated that the conus already lies opposite the L1/2 disk space at term. Therefore , any conus medullaris that is positioned caudal to the midbody of the second lumbar segment should be considered radiographically tethered (Kessler H et al 2007)

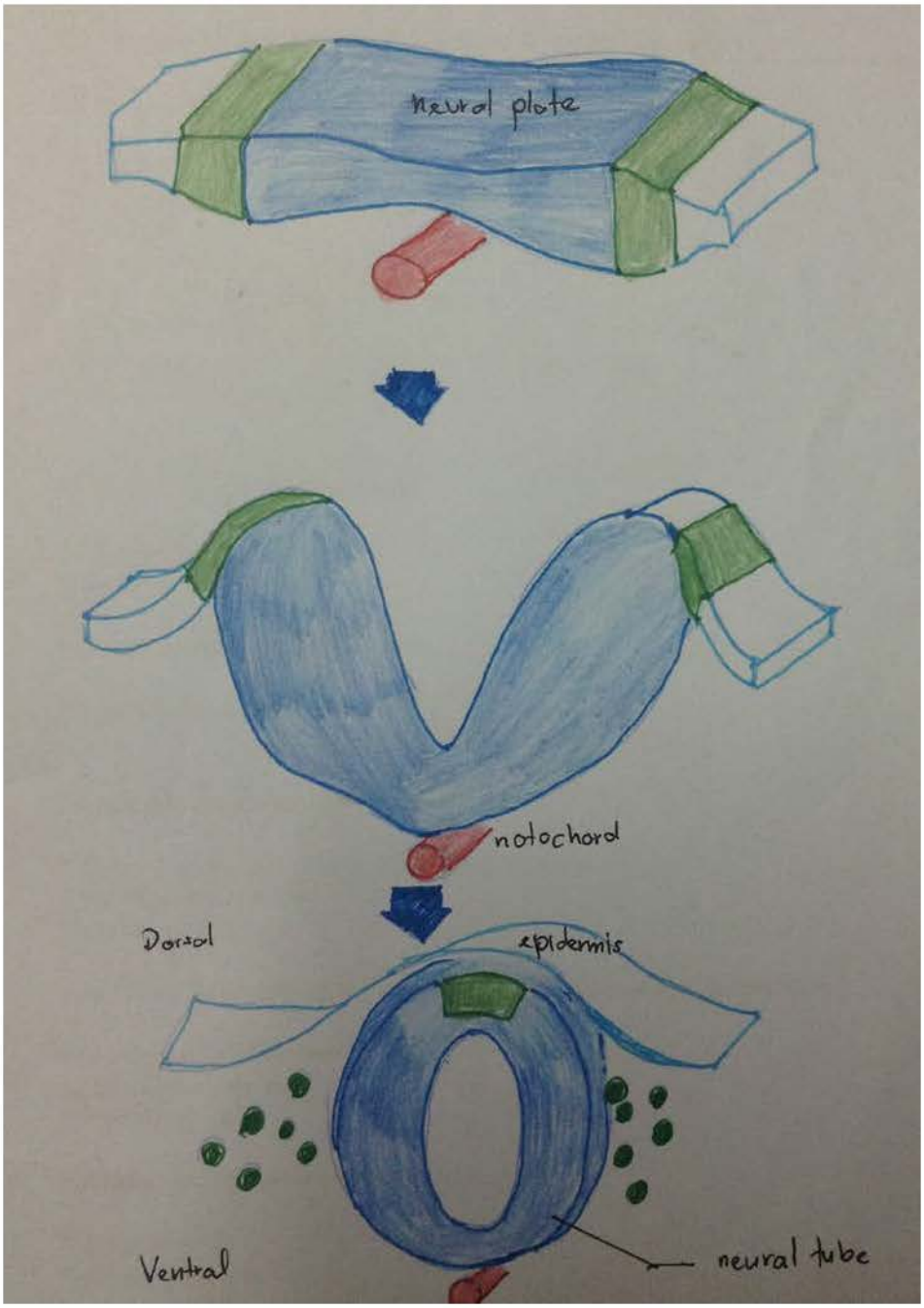
neural plate
notochord
Dorsal
epidermis
Ventral
neural tube

Figure1.1 : figure depicting the embryological process of spinal cord development (adapted from lectures of embryology by Dr. Brian E. Staveley Department of Biology , Memorial University of Newfoundland)

Table 1: Classification addapted from' tethered cord syndrome in children and adults – by Shokei Yamada

Embryogenic Classification of dysraphic spinal malformation

Disordered midline axial integration during gastrulation

*Split cord malformations
*combined spina bifida
*neurenteric cysts
*some myelomeningoceles
* some cervical myelomeningoceles
* hemimyelomeningoceles
* some examples of caudal agenesis and klippel- Feil
* complex dysraphic malformations

Localized failure of neuralation

* Myelomeningocele
* Anencephaly

Premature ectodermaldysjunction

* Lipomyelomeningocele

Incomplete ectodermal dysjunction

* Dermal sinus tracts, dermoid/epidermoid tumours
* meningocele manqué
* meningocele

Disordered formation of the caudal cell mass or secondary neuralation

* terminal spinal lipomas
* fatty filum terminale
* myelocystoceles

Postneurulation disorders (not discussed)

* Encephaloces
* Chiari II malformation

1.2 EMBRYOGENESIS OF TETHERED CORD SYNDROME

When neural tube has completed its fusion , the ectoderm detaches on either side via a process called dysjunction. If however the dysfunction occurs before the neural tube closure is complete, or if the closure is faulty , mesenchymal cells gain access to the central canal of the neural tube .

The mesenchymal cells then differentiate into fatty tissue to form a lipomyelomeningocele (Pang D et al 1993). This fatty tissue is extending from the conus to the subcutaneous plane underlying an intact skin.This embryogenesis explaines the manifestations of lipomyelomenngocele .

An error in the canalization of the caudal cell mass may be another explanation for the origin of a lipomyelomeningocele. According to Marin – Padilla (Pang D 2005) dural schisis is the basic defect resulting in the various forms of tethering with a short thick filum terminale occurring as a secondary event. Occult spinal dysraphism with terthered cord may be associated with anorectal malformations. (Dawson Fong ;2006)

The proposed embryogenesis involves faulty seperation of neuroectoderm from cutaneous ectoderm at the time of dysjunction (Walker AE Brain 1934)

1.3 DEVELOPMENTAL ANATOMY OF THE SPINAL CORD

The spinal cord in comparison to the brain lies freer within the vertebral canal. The dura mater is composed of dense connective tissue with few elastic elements derived from paraxial mesoderm. (Sensenig EC Contrib Embryol 1951) It is separated from the vertebral internal periosteum by the epidural space, which contains fat cells, blood vessels, and loose connective tissue.

The spinal cord needs to be completely free from the vertebral column during development because the rates of growth of the two structures are different. Early in development, the caudal region of the spinal cord undergoes a progressive upward displacement or retrogression relative to the caudal vertebral column. (Streeter GL et al 1919).

The conus medullaris , which is initially at the the coccygeal level in the 30mm embryo, ascends through the S4 level in the 67mm embryo, to the level L3 level by birth (40 weeks conceptional age) and to the adult L1-2 by 49 to 50 weeks conceptional age (Barson et al 1970)

The subarachnoid space elongates progressively to accommodate the elongating spinal nerve roots and the filum terminale. The filum terminale must also be elongated because

the cord retains its original coccygeal attachment through this structure. Early dural schisis (below L3) through which the spinal cord comes in direct contact with the subcutaneous tissue lends to tethering of the spinal cord to this tissue.

Later, subcutaneous adipose tissue penetrates and expands into the intraspinal space (Marin-padilla 1985). This results in a low conus medullaris and a short, thick filum terminale. It is possible that the adipose tissue is stimulated by its direct contact with neural elements and the abundant arachnoidal vascularity through the dural schisis

1.4 ETIOLOGY & PREVALENCE

The causes are multifactorial with both genetic and environmental factors playing a role.

Risk factors have been identified for specific types of dysraphic lesions:

- Folic acid deficiency (whether caused by dietary deficiency, folic acid antagonists, or genetic abnormalities of folate metabolism), has been clearly associated with open spinal dysraphism (myelomeningocele), and the benefit of folic acid supplementation has been established (Betteridge KJ 2001) However, no studies have specifically addressed whether there is a relationship between folic acid deficiency and closed spinal dysraphism (Michelson DJ 2004)
- Maternal diabetes has been associated with increased incidence of the syndrome of caudal regression, but the exact mechanism remains unclear Estin D. Cohen , 1995).
- Maternal exposure to certain medications has also been associated with spinal dysraphism in the fetus. As an example, maternal exposure to valproate or carbamazepine has been associated with spina bifida in their offspring, both in

humans and rodent models (Ceylan S 2001). The human studies generally focused on open rather than closed spinal dysraphism.

Data from Malaysian National Neonatal Registry (Boon NY et al J. 2013) revealed in his study that the prevalence of neural tube defects was 0.42/1000 live births, being highest among the indigenous people of Sarawak (1.09/1000 live births) & lowest among Malaysian Chinese descent (0.09/1000 live births)In this study (Boon NY et al) the majority of mortality was seen in anencephaly (94.5%) encephalocele (45.8%) & lowest in spina bifida (9.5%).

1.4 SYMPTOMALOGY & ASSESSMENT

Cutaneous manifestations are a common presentation in patients with terthered cord. For example patients with lipomyelomeningocele will commonly have a fatty mass . Other cutaneous manifestations are like hemangioma or dimple could be present. Some may present with hairy patch or area of thin atrophic skin .

Patients with previous history of myelomeningocele repair who develop secondary tethering will obviously have a previous surgical scar .Patients with a tethered cord present with progressive motor or sensory deficit in the lower limbs. They may come with gait disturbance secondary to foot deformities such as pes cavus or an equines deformity.

The patients could develop trophic ulcers of their foot due to sensory loss.Scoliosis alone or in combination with other problems are common in patients with tethered cord. Untethering the spinal cord in a patient with mild scoliosis can frequently prevent

progression of or even improve the scoliosis and thus avoid the need for instrumentation.

Neurogenic bladder is a common presentation in patients with tethered cord. Back pain and root pain may occur in patients with a tethered cord , may be seen in patients with secondary tethering after repair of lipomyelomeningocele or myelomeningocele . The pain may radiate along dermatomes.

Sudden and irreversible deterioration neurological function is possible especially in childhood during growth spurts and also in adult life as a result of trauma and flexion movements of the spine. (James M drake and Harold J Hoffman 1981)

The initial assessment of the newborn is crucial. Visual examination of the lumbosacral region may reveal a hair tuft, a dermal sinus, a lipomatous mass, a midline nevus, or hypertrichosis, which may be the only suggestion of embryonic failure of differentiation between the midline ectoderm and mesoderm occurring in the first trimester of pregnancy.

Cutaneous manifestations occur in 40% of patients with TCS. Pes cavus , and equinovarus skeletal abnormalities may be the presenting feature feature of TCS.

Prenatal screening for neurological abnormalities is based on ultrasound performed routinely or oriented by maternal alpha fetoprotein(AFP) screening. It should be performed around around 12, 22 and 32 weeks. Maternal serum screen can detect up to 80% of spina bifida and 90 % of anencephaly. (Boop et al 1992)

Sonography may identify up to 90& of myelomeningoceles. Maternal serum screening for chromosomal abnormalities is increasingly being used.Radiological assemment should be be done as early as possible . MRI is the investigation of choice to study the neural tissue abnormalities and assess the severity of hydrocephalus . (Brunberg JA et al 1988).

1.4.1 MRI IN TERTHERED CORD

MRI is the investigation of choice to study the neural tissue abnormalities and also to assess the severity of hydrocephalus and Chiari malformation.There are important comparisions that need to be made anatomically thro imaging and two features need to be determined for supporting the diagnosisof tethered spinal cord, 1) position of he normal cord and 2) filum terminale

1) position of the normal

During in utero life, the bony spine grows faster than spinal cord, thus the spinal cord affectively ‘ascends’ in the spinal canal. The ascension occurs rapidly between 8 and 25 weeks of gestation, with the conus generally being located opposite L2 at birth. (Kaufman BA et al 2004).

It reaches adult level at age of 2 months postnatally. (Barson AJ et al 1970) .The conus does not ascend further during childhood and it terminatesnear the L1-2 disk space in majority of normal individuals. A conus level terminating at or below L3 -4 is abnormal (except possibly in premature infants and full term newborns), and the significance of an L3 conus level must be determined by means other than its position.

2) Filum terminale

Controversy still remains as to how thick the filum may be and still be 'normal'. Some have said that filum thickening occurs by definition when it is wider than 2mm (Naidich 1986).A fatty filum can be an incidental finding and is not considered diagnostic of tethered cord (Mc Lendon RE et al 1988) because it is reportedly present in 5.8% of the normal population on postmortem examination. (Emery JL et al 1969). The fat appears as low attenuation (dark) on CT and bright (or white) on T1 weighted MRI sequences.

Cord terthering can be associated with spinal bifida occulta in the following conditions:

- Low Cord Termination: Cord tethering is often assumed when the conus is below the normal L2-3 level. (Page LK ; 1985). In some patients with tethered cord show normally positioned conus in MRI. In such case , it might be appropriate to obtain additional prone and supine images to determine whether the cord shows normal movement, in normal cases the spinal cord exhibits anteroposterior and craniocaudal movement when the patient has changed from supine to prone position. (Figure 1.3)

- Thick filum : Cord tethering is usually associated with shorthened, thickened filum terminale. The thickened, shortened filum seeks the shortest distance between the tethering site and the lowest pair of dentate ligaments and may tent the dura posteriorly resulting in a triangular shape of the thecal sac. This is well appreciated with MRI(Yamada S, et al 2001). The thick filum has higher intensity signal than CSF on relatively T1 or proton density weighted MRI scans.

- Myelomeningocele : these are herniations of meninges and spinal cord through a defect in the bony spinal canal, placode is elevated from the dorsal surface of the body, with an underlying expansion of the subarachnoid space. There is a fusiform widening of the interpedicular distances spanning several verterbral bodies and outward rotation of the axes of the pedicle and laminae toward the coronal plane. Kyphoscolisiosis is present 30 -35% of the time due to severe segmentation anomalies and 65% of the time due to neuromuscular imbalance.

- Lipomyelomeningocele: Here there is posterior herniation of meningocele, spinal cord and lipoma into the subcutaneous tissues. According to Naidich and associates (Naidich TP et al 1983) as the lipomyelomeningocele herniates through the canal, (Figure 1.2) its superior aspect is notched by a fibrovascular band. This band is continous with the periosteum, joins the laminae of the most cephalic vertebra with widely bifid laminae, and appears to

tether the neural tissue and meningocele because they relax after surgical transection of the band.

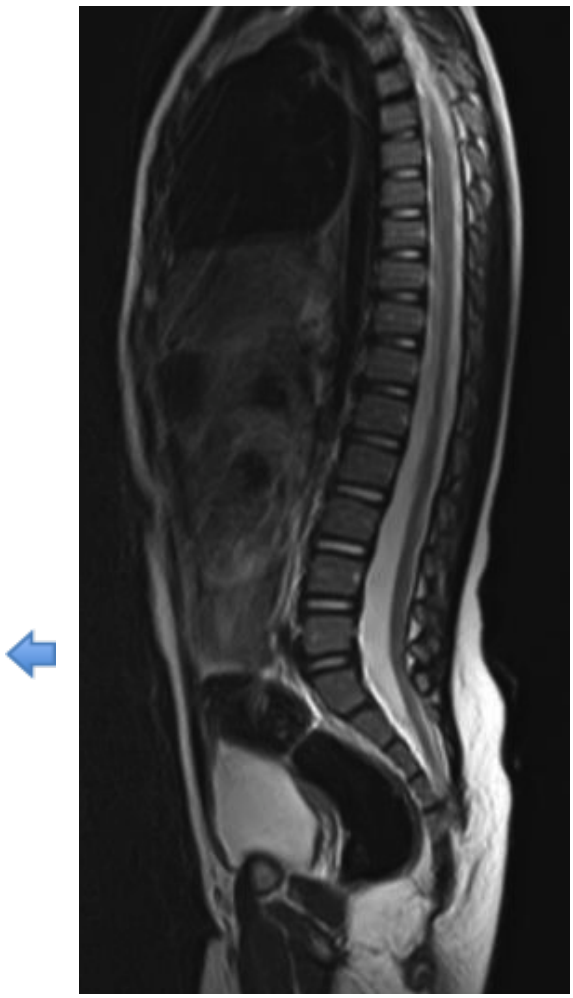

Figure 1.2 T2 weighted sagital MRI of a patient in this study preoperatively showing the low lying conus at the level of L4 and the high signal fat extending through a defect in the spine. The cord is tethered.

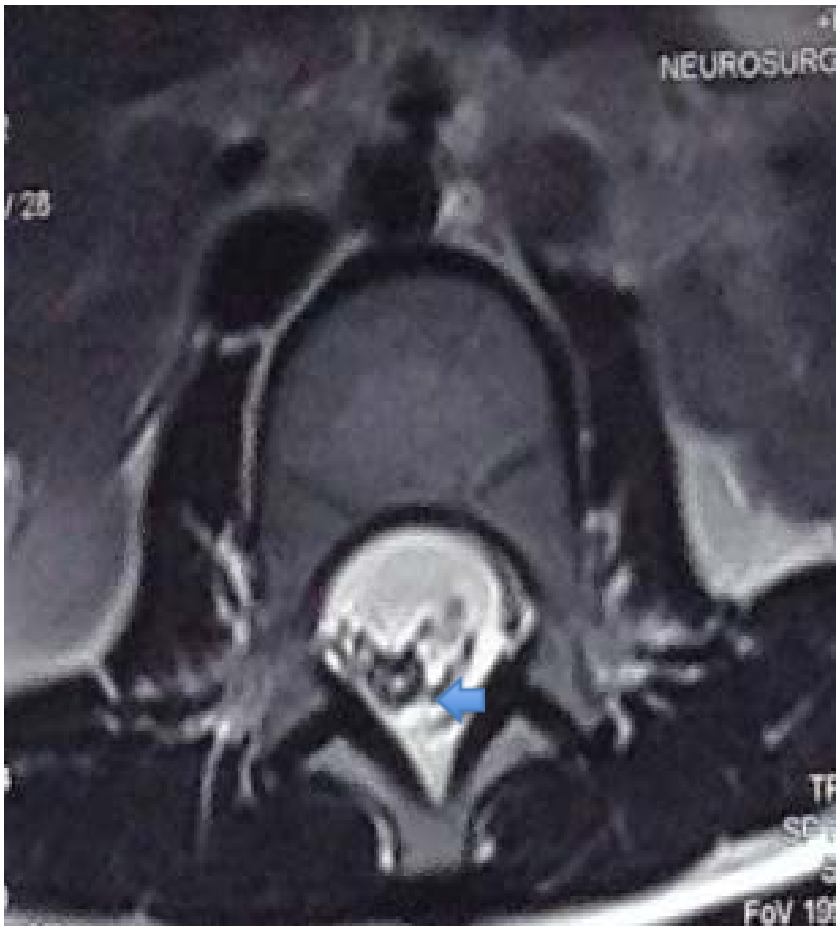

Figure 1.3 Axial T2 weighted image of a patient in this study showing tethered cord at the level of L3 – 4.

CHAPTER 2: LITERATURE REVIEW

2.1 Historical Background of Terthered Cord Surgery

Lichtenstein a neuropathologist in 1940 is the first to coin the term tethered cord , proposing that tethering of the spinal cord may cause paraplegia and herniation of the brainstem althought it was not a hypothesis accepted .

In 1976 Hoffman et al adopted the term tethered spinal cord in a report on 31 patients presenting with incontinence and motor and sensory deficits in the lower limbs.These symptoms subsidedafter sectioning of a thickened filum terminale (Hoffman HJ, 1976) which indicated that the neurological lesion was in the lumbosacral cord.

Barson et al 1970 noted that the spinal cord does not ascend significantly after birth.Spine grows most rapidly during embryogenesis and after puberty (during teenage growth spurts), while symptoms of tethering most often are observed in early childhood (age 3-10 years).

Yamada et al in 1981 demonstrated impairment of oxidative metabolism in the lumbosacral cord before surgery and recovery from the impairments after surgery. Yamada et al examined the mitochondrial oxidative metabolic changes in the spinal cord before and after subjecting it to stretching. Using reflection spectrophotometry, they monitored in vivo changes in the reduction: oxidation (redox) ratio of cytochrome a, a_3 in animal models and in human tethered spinal cords.

Yamada found a marked metabolic and electrophysiological susceptibility of the lumbosacral cord subjected to hypoxic conditions, especially under traction with hypoxic stress this is supported by the associated evoked potential changes.

The goal of surgery is to detether the cord by disconnecting its aberrant tightening attachments, and to relieve the cord from continous stretching forces. Kang et al found that cord tethering caused a reduction of regional spinal cord blood flow in the distal spinal cord close to the site of tethering. The reduction in regional spinal cord blood flow (rSCBF) became progressively worse over the weeks following the tethering. Untethering of the cord led to an increase in the rSCBF if the untethering occurred by 2 weeks after tethering.

The entity of lipomyelomeningocele was first described by Johnson in 1857 (Johnson A et al 1857) . However it was not until 1950 that Bassett (Bassett RC et al 1950) stressed on the deterioration in function in patients with a lipomyelomeningocele and emphasized on the value of prophylactic surgery.

However the role of surgery remained controversial until recently. Matson
(Matson DD et al 1969) mentioned the importance of cosmetic repair to be included in the surgical management.
In 1953 Garceau (Garceau GJ et al 1953) who demonstrated improvement of 3 patients following sectioning of thick filum terminale which was discovered at laminectomy. In 1976 Hoffman and collugues, reviewed their outcome with patients who had spinal cord tethering or thick filum .

2.2 PATHOPHYSIOLOGY OF TETHERED CORD SYNDROME

Two decades ago, the usual explanation for the neurological deficit associated with tethered cord syndrome was the effect of traction in preventing the ascent of the spinal cord within the spinal canal during growth. However (Barson , 1970) pointed out in 1970 that the spinal cord does not ascend significantly after birth. The incongruity in observations is due to the fact theat the spine grows most rapidly during embryogenesis and after puberty (during teenage growth spurts) , whereas symptoms of tethering are most often observed in early childhood (age 3 to 10 years).

Yamada et al examined the mitochondrial oxidative metabolic changes in the spinal cord before and after subjecting it to stretching. Using reflection spectrophometry, they monitored in vivo changes in the reduction:oxidation (redox) ratio of cytochrome a,a3 in animal models and in human tethered spinal cords . Yamada et al found a marked metabolic and electrophysiological suscepbility of the lumbosacral neuronal dysfunstion and that this dysfunction is possibly due to impairmant of mitochondrial oxidative metabolism.

Most pediatric neurosurgeons now believe that the chronic stretch on the cord produced by tethering is an essential part of the problem but that superimposedinsults such as acute flexion episodes or cord hypoxia accentuate symptoms to become more prominent.

Kang et al tethered and untethered the cords of immature kittens and studied the effects of these manipulations on regional spinal cord blood flow, clinical features, and SSEPs.

They (Kang et al) found that cord tethering caused decreases in regional spinal cord blood flow in the distal spinal cord close to the site of tethering. The decreases in regional spinal cord blood flow (rSCBF) became progressively worse over the weeks following the tethering.

Untethering of the cord led to an increase in the rGCBF if the untethering occurred by 2 weeks after tethering. Delaying the untethering until 8 weeks caused failure of the rSCBF to recover the normal level. Changes in the evoked potential occurred when rSCBF fell below 14ml/100g/min. The decrease in rSCBF had occurred by 2 weeks of tethering.

2.3 SURGICAL INTERVENTION FOR TETHERED CORD SYNDROME

The principles of surgery is for complete untethering of the spinal cord and proper dural reconstruction with adequate CSF space around the spinal cord to prevent retethering.

Surgical techniques are discussed differently when tethered cord syndrome is associated with myelomeningocele and lipomyelomeningocele

Yamada et al divided MMC into 3 types based on surgical anatomy

i) Type 1: spinal cord buckled, travels inside the meningocele sac, and its neural placode surfaces along the dome of the sac . The placode is covered by a thin layer of poorly developed epithelial tissue but not by meningeal lining. The caudal part of the spinal cord often enters the spinal canal with defective laminae, its end the filum is either loosely or densely adhesive to the surrounding tissue.

Surgical technique(Shokei Yamada 2004)

An elliptical incision surrounds the myelomeningocle.(Figure 2.1) The bukled spinal cord reaches the dome of the MMC where the placode is exposed. The spinal cord ends

in the sacral canal with either loose or dense adhesion to the surrounding connective tissue. It is important to release the tethering of the cord. After laminectomy at one level above the spina bifida, a midline dural incision is made to expose the cephalic end of the neural placode is ontinous to the central canal. The trimmed edge of the meningocele sac is shown underneath the arachnoid in the normal spinal canal. The arachnoid and the dura are everted away from the placode. The pia mater has been closed with 8-0 nylon sutures. The figure shows sutures being plaed through the arachnoid (dura is indicated by large arrows and the arachnoid by small arrows in.

The central placode has sunk to the anterior spinal canal after cerebrospinal fluid (CSF) draining,(Figure 2.2) and the trimmed edge of the meningocele sac borders the placode (white arrows). The everted arachnoid has a broad base and its lateral extension has been isolated from subcuticular tissue (black arrows). The opening of the central canal (open arrow) and the midline groove of the neural placode are clearly seen. CSF draining from the central canal percolates through the thin membrane over the placode, causing constant wetting of the placode surface. The nerve roots directing cephalad (to the right) and exiting through the arachnoid and dura are identified.Transection of the MMC shows the arachnoid membrane extending laterally to undermine the skin. The meningocele sac is fused with the arachnoid except for the area of membrane directly attached to the placode edge.

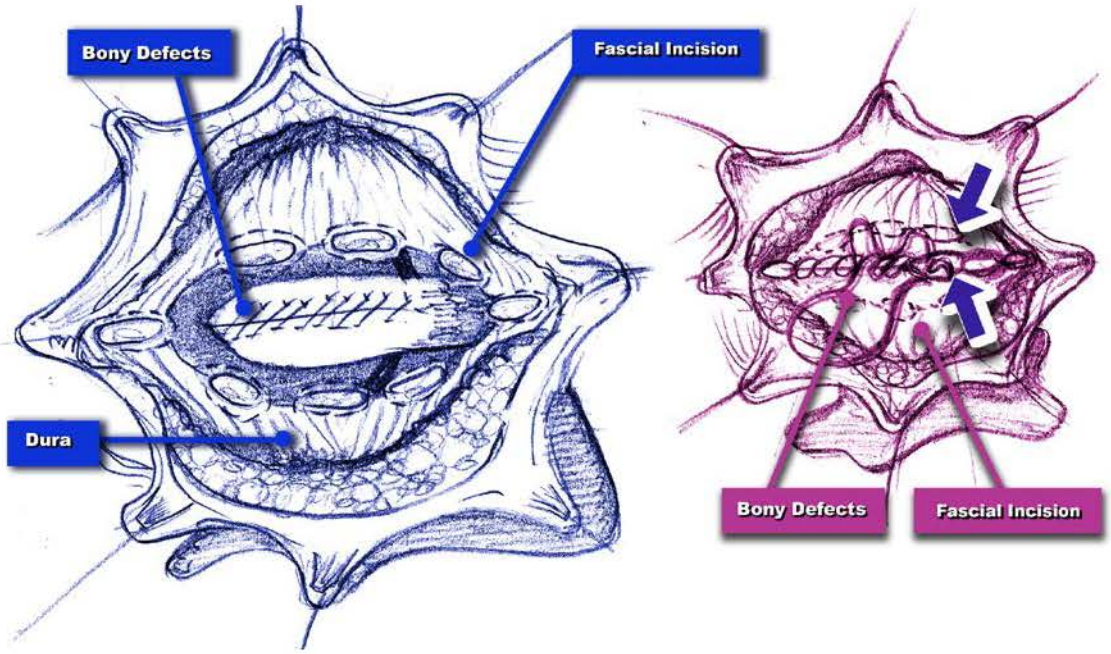

Figure 2.1 The surgical technique adapted from illustrations by shokei Yamada in " tethered cord syndrome in children and adults"