Spinal Dysraphism

Andrew Jea MD MHA FAAP
Professor and Chief
Section of Pediatric Neurosurgery
Riley Hospital for Children
Department of Neurosurgery
Indiana University School of Medicine
Goodman Campbell Brain & Spine

Disclosures
None
Anomalies of Notochord Formation

- **Diastematomyelia**
  - Spinal cord divided vertically into two hemicords, each with its own central canal and surrounding pia
  - Disorder may result from splitting around an adhesion between the endoderm and ectoderm
  - Split notochord may influence formation of two neural tubes and two subsequent hemicords with associated segmentation anomalies of the vertebrae at the site of diastematomyelia
  - Females more commonly affected than males

- **Cutaneous stigmata in 2/3 of cases**
  - Nevus
  - Hypertrichosis
  - Lipomas
  - Dimples
  - Hemangiomas
  - Symptoms related to cord tethering

- **Conus is low-lying in 75% of cases**

- **Hydromyelia found in 50% of cases**

- **Two forms of diastematomyelia by Pang classification**
  - Type I: two hemicords surrounded by individual dural tubes (40%)
  - Type II: two hemicords surrounded by a single dural sheath (60%)

- **Type of spur (bony or fibrous) probably determined by amount of trapped mesenchyme in cleft (red arrow)**

- **Diastematomyelia accounts for 5% of congenital scoliosis and associated with 30-40% of myelomeningocele**
Anomalies of Notochord Formation

- **Split Notochord Syndrome**
  - Persistent connection between the endoderm and ectoderm results in splitting or deviation of the notochord
  - Neuroenteric fistula communicates intestinal cavity to dorsal skin in the midline
  - Fistulae may traverse prevertebral soft tissues, vertebral bodies, the spinal canal and its contents, and the posterior elements
  - Usually present as newborns
  - Neuroenteric sinus has an opening to the skin surface and is a remnant of the posterior portion of the tract

- **Neuroenteric cysts** are trapped remnants of the middle portion of the tract found in the intraspinal or paraspinal compartments
  - Usually present between 20 and 40 years as radicular pain that may progress to myelopathy
  - Most commonly occur at the cervicothoracic junction

- **Neuroenteric diverticulum** is a tubular diverticulum arising from bowel and represents a remnant of the anterior portion of the tract

Anomalies of Premature Dysjunction

- **Spinal Lipomas**
  - Three types of spinal lipomas
    - Intramedullary lipomas (IM): premature dysjunction
      - Completely closed by dura
    - Most commonly occur in the cervical and thoracic spine
      - Present with slow, ascending monoparesis or paraparesis, spasticity, cutaneous sensory loss, and defective deep sensation
    - Lumbosacral lipomas may present with flaccid paralysis of legs and sphincter dysfunction
      - Chapman classification: dorsal, terminal/caudal, or transitional
Anomalies of Premature Dysjunction

• Spinal Lipomas
  • Three types of spinal lipomas
    • Lipomyelomeningoceles / lipomyeloceles (84%): premature dysjunction
    • Lipomyelomeningoceles: lipomas attached to the dorsal surface of the neural placode through a dural defect
    • Herniation of malformed neural elements due to expansion of ventral subarachnoid space
    • Lipomyeloceles: lipomas attached to cord and subcutaneous tissue through a dural defect but with normal-sized ventral subarachnoid space
  • Fibrolipomas of filum terminale (12%): abnormality of retrogressive differentiation

Anomalies of Premature Dysjunction

• Spinal Lipomas
  • Conservative versus prophylactic surgery for “asymptomatic cases”
  • Natural history of spinal lipomas of conus
    • 53 asymptomatic children followed up to 9 years (average 4.4 years)
    • Conservative cohort compared to historical cohort
    • Risk of neurological deterioration at 9 years
      • 33% for conservative group
      • 46% for surgically treated group
      • No significant difference in risk of neurological deterioration for those treated conservatively versus those who underwent early surgery

Anomalies of Nondysjunction

• Dermal Sinus Tracts
  • Focal anomaly of nondysjunction
  • Thin, epithelium-lined channels that open on the skin posteriorly in a hyperpigmented patch or hairy nodule
  • Sinus tracts extend deep into subcutaneous tissues and possibly bone, reaching the spinal canal in 1/2 to 2/3 of cases
  • Sinuses may be attached to dura
  • Sinuses with intradural passage may end in subarachnoid space, conus medullaris, filum terminale, a nerve root, a fibrous nodule on the surface of the cord, or a dermoid / epidermoid cyst (50%)
  • May be a history of meningitis from extension of bacteria along the tract or from chemical irritation if the cyst ruptures
Anomalies of Nondysjunction

- Myelomeningocele
  - Diffuse anomaly of nondysjunction

Anomalies of Caudal Cell Mass

- Tight Filum Terminale Syndrome
- Incomplete retrogressive differentiation
- Complex of neurologic and orthopedic deformities
  - Scoliosis
  - Myelopathy
  - Bowel/bladder dysfunction
  - Orthopedic foot deformities (club-foot, talipes equinovalgus)
  - Red pain
  - Asymmetric leg/calf atrophy

Anomalies of Caudal Cell Mass

- Syndrome of Caudal Regression
  - Degree of maldevelopment often profound and the clinical defects severe
    - Sirenomelia
    - Cardiac anomalies
    - Anus atresia
    - Lower extremity paralysis
    - Neurogenic bladder

- Sacrococcygeal Teratomas
  - Congential tumors probably arise from primitive, multipotential cells along primitive streak
Myelomeningocele

Anatomy

• Groove in center of placode represents remnant of central canal (arrow)
• Spinal roots exit from anterior surface of placode
  • Ventral roots medial
  • Dorsal roots lateral

• Edge of placode fused with dystrophic epidermis and dermis laterally
• Collection of CSF in subarachnoid space (between dura and placode) pushing placode dorsally
• Dura fuses with defect in fascia laterally
• 85% myelomeningoceles in caudal thoracolumbar spine or more distal; 10% in thorax; 5% cervical
Anatomy

- Bony abnormalities
  - Absence of spinous process and laminae
  - Decrease in A-P diameter of vertebrae
  - Increase in interpedicular distance
  - Decrease in pedicle height
  - Large transverse processes

Almost all patients with myelomeningocele have associated Chiari II malformation
- Herniation of cerebellar vermis and brainstem into cervical spinal canal
- Medullary kinking
- Upward angling of cervical nerve roots
- Low-lying tentorium with small posterior fossa
- Widened foramen magnum

Anatomy

• Chiari II malformation
  • McLone’s hypothesis for sequential development of myelomeningocele and Chiari II malformation
    • In normal development, primitive central canal occludes transiently during period of rapid brain enlargement, after closure of posterior neuropore
    • In myelomeningocele, primitive central canal fails to occlude allowing continuous egress of CSF
      • Lack of distention of brain by CSF results in malformation of cranium and its contents - infratentorial and supratentorial

Anatomy

• Beaking of tectum
• Intrinsic brain stem nuclei changes
• Partial or complete dysgenesis of corpus callosum
• Polymicrogyria
• Large massa intermedia
• Gray matter heterotopias
• Luckenschadel = mesodermal skull abnormality

Anatomy

• Hydrocephalus
  • 80-90% myelomeningocele patients require treatment
• Hydrosyringomyelia
  • 40-80% myelomeningocele patients
History

• 1950s development of VA shunt for hydrocephalus led to more aggressive treatment of children with spina bifida

Epidemiology

• Prevalence declined due to prevention with folate supplements and pregnancy termination after prenatal diagnosis
  • Before 1980s, prevalence in US = 1 to 2 per 1000 live births
  • By 1989, prevalence declined to 0.6 per 1000
  • Recently, prevalence reported at 0.44 per 1000

Etiology

• Prevalence of spina bifida in US higher in whites than blacks or Asians, suggesting genetic basis for disorder
Etiology

- Anticonvulsants
  - Risk of NTD with maternal ingestion of carbamazepine or valproic acid = 1 to 2%
- Diabetes
  - Risk of NTD with maternal history of DM Type I = 1%
- Obesity
  - Pre-pregnancy obesity as independent risk factor for NTDs by Shaw et al

Prenatal Diagnosis

- Maternal Serum AFP
  - AFP levels peaks at 10-13 weeks, in amniotic fluid at 12-14 weeks, and in maternal serum at 28-32 weeks
  - Optimal time for sampling at 16-18 weeks
  - 79% fetuses with open NTDs (vs 3% normal fetuses) result in increased MSAFP by 2.5-fold compared to median
  - If initial and repeat MSAFP levels elevated, then proceed to fetal ultrasound... stable or declining MSAFP level not consistent with NTD

Prenatal Diagnosis

- Ultrasonography
  - High-resolution fetal ultrasonography at 16-20 weeks gestation as screening tool with near 100% sensitivity
  - Detects spinal defect as well as hydrocephalus and Chiari II malformation associated with spina bifida
  - "Lemon sign" = scalloping frontal bones on coronal views
  - "Banana sign" = abnormally shaped midbrain and elongated cerebellum in Chiari II malformation
Prognosis

- Survival
  - > 95% infants today with myelomeningocele survive 1st 2 years
  - Still 10-15% children with spina bifida die before age 6
    - Hydrocephalus
    - Renal failure
Prognosis

• Ambulatory Status
  • Standing requires L3 function, ambulation requires L4 and 5 function
  • 60% community ambulators with or without assistive devices before adolescence (only 50% after adolescence because of weight gain)... 15% household ambulators... 26% non-ambulators
  • Only 6-17% with normal urinary continence... 85% socially continent of urine with meds and intermittent catheterizations... 86% socially continent of feces

Prognosis

• IQ
  • 75-80% children with spina bifida have normal intelligence with aggressive management
  • Correlates with lower intelligence:
    • Higher myelomeningocele level
    • Degree of ventricular enlargement on prenatal u/s
    • Number of CNS infections, not number of shunt revisions

Perinatal Management

• L&D
  • Infants may suffer trauma to exposed neural placode during labor... elective C-section after lung maturity but before onset of labor may improve neurologic outcome
  • Contraindications for C-section
    • Fatal fetal chromosomal abnormalities
    • Severe hydrocephalus and < 1cm cortical mantle (and no cephalopelvic disproportion)
    • Fatal u/s with no knee or ankle movement
    • Flat or depressed placode
Perinatal Management

• L&D
  • Indications for C-section
  • Movement of knees or ankles
  • Protruding myelomeningocele sac

• Wound Care
  • Myelomeningocele covered with sterile saline-moistened gauze
    dressing and wrapped with plastic wrapping to minimize
    dehydration and contamination
  • Positioned prone or on side to avoid pressure on placode
  • Latex precautions taken

Perinatal Management

• Urological Exam
  • If no void, then intermittent catheterizations
  • If voiding present, then post-void residual checked to
    confirm adequate emptying of bladder
  • Renal U/S to rule out significant abnormalities of GU
    system, ie absence of a kidney

Perinatal Management

• Neurologic Exam
  • Document spinal level of defect, sensory level, and
    motor level
  • L1-3 = hip flexion with extended knees and clubfeet
  • L2-4 = knee extension with inverted feet but intact hip
    adduction and flexion
  • L5-S2 = hip adduction, knee extension, and knee flexion with
    dorsiflexed feet
  • Sacral = plantar flexion with rockerbottom feet
Perinatal Management

- HC, AF, and cranial sutures examined for signs of increased ICP suggestive of HCP... cranial u/s to document degree of HCP (sometimes ventricles will remain small until spinal defect closed)
- Observation for signs and symptoms of Chiari II malformation
  - Cranial nerve dysfunction
  - Central or obstructive apnea
  - Stridor
  - Opisthotonus

Myelomeningocele Repair

- Timing
  - Myelomeningocele repair usually performed within 48-72 hours after birth
    - delay after 72 hours may increase chances of meningitis or ventriculitis
- Hydrocephalus
  - VP shunt placement post-myelomeningocele repair reserved for:
    - Patients with clinically proven hydrocephalus
    - Patients with radiographically progressive ventriculomegaly
    - Patients with pseudomeningocele/CSF leak from lumbar wound

Myelomeningocele Repair

- Operative Technique
  - Goal: reconstruct neural tube and coverings to avoid meningitis and protect functional tissue in neural placode
  - Neural placode freed from surrounding junctional zone circumferentially
Myelomeningocele Repair

- Residual epidermal or dermal elements removed from periphery of neural placode to minimize formation of dermoid or lipoma
- Layers closed
  - pia
  - dura
  - thoracolumbar fascia
  - subcutaneous tissue
  - skin

Fetal Repair

- Based on premise that secondary neurologic damage occurs from exposure of the neural tube to caustic intrauterine environment
  - Heffez's "two hit" hypothesis
- Initial goal of fetal surgery was to reduce exposure of neural tissue to intrauterine environment

Fetal Repair

- Initial attempts with fetoscopic and open repair showed decreased hindbrain herniation and need for VP shunting but no significant improvement in distal neurologic function
  - Motor
  - Sensory
  - Bladder
- Considerable maternal morbidity and fetal prematurity led to moratorium on procedure
- Prospective, multicenter, randomized trial for Management of Myelomeningocele Study (MOMS) performed to address unclear cost/benefit ratio of fetal versus standard postnatal treatment
  - Role for fetal intervention
  - Establish indication for fetal surgery in non-lethal defects
Historical Perspectives

- 1994: Bruner attempts laparoscopic repair of spina bifida (4 cases performed before stopping)
- 1998: Tulipan reports open repair at 28-30 weeks gestation in 4 fetuses
  - All with absent hindbrain herniation at birth
  - 2 required ventricular shunts
- 253 open cases completed prior to the MOMS trial (177 at VUMC, 54 at CHOP, 12 at UCSF, 10 at UNC)

Am J Obstet Gynecol 1997;176:256-7
Pediatr Neurosurg 1998;29:274-8

Feb 2003 – Dec 2010
$22.5 million

MOMS
Management of Myelomeningocele Study

MOMS Study Population

1087 screened
530 excluded
299 referred to a center
75 excluded
183 randomized
92 postnatal repair
91 fetal repair

### MOMS Inclusion Criteria (maternal)

- Singleton pregnancy
- Gestational age at randomization of 19<sup>0/7</sup> to 25<sup>6/7</sup> weeks gestation
- Maternal age > 18 years
- Body mass index of < 35
- No previous uterine incision in the active uterine segment
- No risk factors for preterm birth (short cervix, history of previous preterm delivery)

### MOMS Inclusion Criteria (maternal)

- Insulin-dependent diabetes
- Infection with hepatitis B or C
- HIV infection
- Red cell alloimmunization
- Unwillingness to accept blood transfusions for religious or other reasons

### MOMS Inclusion Criteria (fetal)

- Myelomeningocele defect between levels T1 to S1
- No evidence of kyphosis
- No major fetal anomaly unrelated to the spina bifida
- Normal chromosomes by amniocentesis

MOMS Outcomes

- **Primary**
  - ✓ 12 months: death or need for cerebrospinal shunt placement
- **Secondary**
  - ✓ 30 months: composite score of Mental Developmental Index of the Bayley’s Scales of Infant Development II AND child’s motor function


MOMS Study Population

<table>
<thead>
<tr>
<th>Level of lesion on US</th>
<th>Fetal surgery</th>
<th>Postnatal surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic</td>
<td>4 (5%)</td>
<td>3 (4%)</td>
</tr>
<tr>
<td>L1 - L2</td>
<td>21 (27%)</td>
<td>10 (12%)</td>
</tr>
<tr>
<td>L3 - L4</td>
<td>30 (38%)</td>
<td>45 (56%)</td>
</tr>
<tr>
<td>L5 - S1</td>
<td>23 (29%)</td>
<td>22 (28%)</td>
</tr>
</tbody>
</table>


MOMS Outcomes (Maternal)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Fetal surgery</th>
<th>Postnatal surgery</th>
<th>RR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Membrane separation</td>
<td>26%</td>
<td>0%</td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Rupture of membranes</td>
<td>46%</td>
<td>6%</td>
<td>6.15X</td>
<td></td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>6%</td>
<td>0%</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>Abruption</td>
<td>6%</td>
<td>0%</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>3%</td>
<td>0%</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>Polyhydramnios</td>
<td>21%</td>
<td>4%</td>
<td>2.22X</td>
<td></td>
</tr>
<tr>
<td>Status of uterine incision</td>
<td></td>
<td></td>
<td></td>
<td>0.03</td>
</tr>
<tr>
<td>Very thin</td>
<td>29%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial separation</td>
<td>10%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### MOMS Outcomes (Neonatal)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Fetal surgery</th>
<th>Postnatal surgery</th>
<th>RR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gest Age (wks)</td>
<td>34.1 ± 3.1</td>
<td>37.3 ± 1.1</td>
<td></td>
<td>0.003</td>
</tr>
<tr>
<td>&lt; 30 wks</td>
<td>13%</td>
<td>0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 – 34 wks</td>
<td>33%</td>
<td>5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35 – 36 wks</td>
<td>33%</td>
<td>8%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birthweight (gms)</td>
<td>2383 ± 668</td>
<td>3039 ± 469</td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>RDS</td>
<td>21%</td>
<td>6%</td>
<td></td>
<td>3.32</td>
</tr>
<tr>
<td>NEC</td>
<td>1%</td>
<td>0%</td>
<td></td>
<td>0.49</td>
</tr>
<tr>
<td>PVL</td>
<td>5%</td>
<td>2%</td>
<td></td>
<td>0.44</td>
</tr>
<tr>
<td>Sepsis</td>
<td>1%</td>
<td>0%</td>
<td></td>
<td>0.20</td>
</tr>
</tbody>
</table>


### MOMS Outcomes (12 months)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Fetal surgery</th>
<th>Postnatal surgery</th>
<th>RR (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary outcome</td>
<td>68%</td>
<td>98%</td>
<td>0.70 (&lt;0.001)</td>
</tr>
<tr>
<td>Death</td>
<td>2%</td>
<td>5%</td>
<td></td>
</tr>
<tr>
<td>Shunt criteria met</td>
<td>65%</td>
<td>97%</td>
<td>0.48 (&lt;0.001)</td>
</tr>
<tr>
<td>Shunt placement</td>
<td>40%</td>
<td>82%</td>
<td></td>
</tr>
<tr>
<td>Any hindbrain herniation</td>
<td>64%</td>
<td>96%</td>
<td>0.67 (&lt;0.001)</td>
</tr>
<tr>
<td>Degree of herniation</td>
<td></td>
<td></td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Moderate</td>
<td>10%</td>
<td>45%</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>6%</td>
<td>22%</td>
<td></td>
</tr>
<tr>
<td>Brainstem kinking</td>
<td>20%</td>
<td>46%</td>
<td>0.42 (&lt;0.001)</td>
</tr>
<tr>
<td>Degree of kinking</td>
<td></td>
<td></td>
<td>p = 0.001</td>
</tr>
<tr>
<td>Moderate</td>
<td>10%</td>
<td>25%</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>4%</td>
<td>12%</td>
<td></td>
</tr>
</tbody>
</table>

*1 IUFD at 26 wks; 1 neonatal death after delivery at 23 weeks; 2 infants died in the postnatal surgery group after one year of age. 4 shunt placed w/o meeting criteria.

### MOMS Outcomes (30 months)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Fetal surgery</th>
<th>Postnatal surgery</th>
<th>RR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bayley MDI</td>
<td>89.7 ± 14.0</td>
<td>87.3 ± 18.4</td>
<td>0.53</td>
<td></td>
</tr>
<tr>
<td>∆ motor/anat level</td>
<td>0.58 ± 1.94</td>
<td>-0.69 ± 1.69</td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>&gt; 2 levels better</td>
<td>32%</td>
<td>12%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 level better</td>
<td>11%</td>
<td>9%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walking independent</td>
<td>42%</td>
<td>21%</td>
<td>2.01</td>
<td></td>
</tr>
<tr>
<td>WeeFIM score</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-care</td>
<td>20.5 ± 4.2</td>
<td>19.0 ± 4.2</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>Mobility</td>
<td>19.9 ± 6.4</td>
<td>16.5 ± 5.8</td>
<td></td>
<td>0.003</td>
</tr>
<tr>
<td>Cognitive</td>
<td>23.9 ± 5.2</td>
<td>24.1 ± 5.9</td>
<td></td>
<td>0.67</td>
</tr>
</tbody>
</table>
Postoperative Care

• Complications
  • 0% operative mortality
  • Superficial wound dehiscence most common complication after myelomeningocele repair
  • Incidence of wound infections 1-1.5%
    • Frequent fecal contamination of myelomeningocele defect
    • Subsequent meningitis with sepsis poses more difficult problem

Postoperative Care

• Hydrocephalus
  • Patient presenting with any deterioration has a shunt malfunction until proven otherwise

Postoperative Care

• Symptomatic Chiari II malformation -> place a shunt early or revise shunt
  • If symptoms continue to progress, then likely secondary to intrinsic brainstem abnormalities
  • Rare indications to perform Chiari II decompression, consisting of cervical laminectomies only
    • No suboccipital decompression
Postoperative Care

- Improved neurosurgical and urological care of children with myelomeningocele mean higher rates of survival into adulthood.
- More patients consequently progress through their growth spurts in setting of previously described anatomic abnormalities and present with spinal deformities.

Normal Spine Growth

- Graph showing normal spine growth with age.

Natural History

- Retrospective review of 250 cases at Birmingham Children’s Hospital from July 1977 to June 1978.

<table>
<thead>
<tr>
<th>Deformity</th>
<th>% Cases</th>
<th>% Cases ≥ 10 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scoliosis</td>
<td>54.0</td>
<td>82.5</td>
</tr>
<tr>
<td>Kyphosis</td>
<td>22.0</td>
<td>17.5</td>
</tr>
<tr>
<td>Lordosis</td>
<td>1.5</td>
<td>12.5</td>
</tr>
<tr>
<td>No deformity</td>
<td>31.0</td>
<td>10.0</td>
</tr>
</tbody>
</table>

> Scoliosis much more common than other deformities and its incidence increased with age.
> Conversely, if a child reached the age of 9 years without developing scoliosis he was unlikely to do so thereafter.

Incidence of scoliosis (%)

- Neurologic deficit:
  - Above T12: 83%
  - L1 to low sacral: 67%
  - None: 5%

- Bony defect:
  - Above T12: 73%
  - L1 to L4: 44%
  - L5 or below: 8%

> Ambulatory patients at lower risk for development of scoliosis.
Myelomeningocele and Spinal Deformity

- Clinical indications for deformity surgery
  - Decreased pulmonary function from restrictive lung disease
  - FVC and FEV1 < 40%
  - Difficulty walking, if ambulatory; or difficulty sitting, if wheelchair-bound
  - Difficulty with catheterizations because of body position
  - Intractable back pain
  - Decubiti ulcers over bony gibbus

Decubitus ulcer formation in wheelchair-bound patients

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Thank you