A Proposed Treatment Algorhythm for
Stable Slipped Capital Femoral Epiphysis Deformity

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A practical algorhythm outlining the surgical approach to SCFE must take into consideration both the severity of the slip and the surgeon’s comfort level with the surgical techniques available. As both the slip pathomorphology and technical comfort levels of surgeons vary considerably, the algorhythm should provide alternative pathways in establishing immediate, and as appropriate, subsequent surgical treatment strategies.

SCFE pathology is defined by stability, duration and severity criteria.

1) Stability versus instability --- a patient with an unstable slip is defined as unable to ambulate even with crutches. A patient with a stable slip can ambulate. A patient with an unstable slip typically presents more like a displaced acute femoral neck fracture (severe pain, marked discomfort, stress on any attempted hip exam and the patient simply cannot nor will attempt to ambulate even with crutches). Taking an accurate history and performing a careful exam helps to further distinguish a stable from a truly unstable slip. Stable slips can become acutely painful ("acute on chronic") from repetitive metaphyseal bump/acetabular rim pathological impingement. The patient may indeed refuse to walk because of this pain (such a slip deformity
is more severely impinged then it is a Loder unstable slip). The majority of presenting slips are not, as such, unstable but are stable for the point of the subsequent discussion.

2) Duration---the term “acute” slip has had two meanings. If a slip (stable or unstable) is of recent onset, a few weeks or less, we today refer to this as a slip of recent or “acute” onset---and if longer, a chronic slip. In the past, if a patient presented with a history of a slip and an acute recent worsening of symptoms (essentially now unstable), the slip was then referred to as an “acute slip”, sort of equating acute with unstable.5

3) Severity---for discussion purposes stable slip deformity can be classified as mild (< 30° posterior slip), moderate deformity (30° to < 60°), and severe deformity (≥ 60° slip).1,2,6,7,8 Two radiographs, an AP and usually a frog-leg lateral, often suffice in the initial assessment of slip deformity. Additional details can be obtained from a cross table lateral radiograph (in some patients not so easily obtained) which can better define the relative severity of posterior epiphysis slippage and the pathoanatomy of the anterior metaphyseal bump.9 In severe deformities, the bump may be best visualized on a Dunn lateral. Additionally, ultrasound has been used to study bump morphology.10
Assessment / Treatment of Stable Slips:

Correlating the radiographic slip deformity with an assessment of hip motion, both awake and asleep, is essential in planning optimal immediate surgical strategy. The motion on the affected hip is compared to the likely unaffected hip.\textsuperscript{1,2} If there is near 90° of flexion and minimal notable obligatory external rotation in flexion, i.e., motion is almost similar to the normal hip, stabilization in situ with one large cannulated, preferably fully threaded screw is indicated.\textsuperscript{11} If flexion is restricted, and/or some obligatory external rotation is noted, then the initial treatment in addition to stabilization might also include an anterolateral osteochondroplasty (Heyman procedure).\textsuperscript{1,2} The osteochondroplasty can be performed through an anterolateral (Watson Jones) approach\textsuperscript{12}, or a Smith Peterson approach\textsuperscript{1,2,13}, or arthroscopically.\textsuperscript{1,14,15} The goal is to present further slippage and, as possible, eliminate pathologic anterolateral bump impingement. Post operatively, follow-up assessment of both hip function and range of motion is essential. For some patients, the above treatment for a mild slip may well be definitive; for others, potential hip impingement secondary to femoral acetabular impingement might later become problematic.

Moderate stable slip deformities, arbitrarily 30 – <60° posterior epiphyseal slippage, have a marked variability in the potential to become more chronically problematic.\textsuperscript{1} Again, the assessment of motion, both awake and asleep, is critical in the initial decision-making as to the optimal surgical strategy. If there is restriction in flexion but let’s say > 75° (and not excessive
obligatory external rotation) the slip is first stabilized in situ, again typically one screw. As the epiphysis slips more and more posteriorly, resist the temptation to start the screw insertion too far anterior on the femoral neck. Anteriorly prominent screw heads have a real potential for painfully impinging against the anterolateral rim of the labrum/acetabulum.\textsuperscript{16} Again, it may be appropriate to consider performing an attenuated and/or torn, and there is a real potential for the ragged sharp edge of the metaphyseal ridge of bone to subsequently damage the labral chondral complex.\textsuperscript{1,2,4,17,18} It takes some practice to satisfactorily perform an osteochondroplasty through either a Watson Jones, Smith Peterson, or arthroscopic approach.\textsuperscript{1,2,13,14,15} Bumpectomies are relatively more effective for acute versus chronic, sharper versus rounded, and smaller versus larger anterolateral metaphyseal bony deformities. Larger bumps distort the local anatomy making it more difficult to perform an adequate anterior or anterolateral capsulotomy. The labrum must not be cut. The presence of the lateral retinaculum just posterior to the midline of the base of the trochanter with the critical vessels extending to the femoral head limit the ability to safely resect the more superior lateral portion of particularly large bumps.\textsuperscript{1} At times, manual joint distraction helps in visualizing the junction of the metaphyseal bump with the anterolateral edge of the posteriorly displaced epiphysis. Following in situ stabilization of moderate deformity (with or without a bumpectomy), again long-term follow-up is critical to assess function, range of motion, and possible need for timely additional reconstructive surgery.\textsuperscript{1,2}
The algorithm for surgical treatment for a more notably deformed moderate slips is similar for the severe slips ($\geq 60^\circ$ posterior slip). The slip deformity will typically restrict passive flexion to less than $75^\circ$ with often marked obligatory external rotation in flexion. Passive abduction would be limited to less than $20^\circ$. An external gait deformity will be present. For these most problematic pathomorphological stable slip deformities, the initial options are:

1) In situ stabilization --- now→then later, an anticipated early reconstructive procedure of choice:

   • PFO at ITT level, mostly flexion & IR, often slight valgus ± osteochondroplasty -- without or with SHD$^{1,2,8,19,20}$
   • Osteochondroplasty with SHD$^{1,2,8,19,20}$
   • SHD and modified Dunn$^{1,2,4,18-22}$

2) SHD and modified Dunn --- now

3) Stabilize (maybe) --- now and for the longstanding chronic slip (PFO at ITT level, flex, IR & often slight valgus) ± osteochondroplasty ---now, with or without SHD.

There is no doubt that the most direct way to obtain optimal anatomical correction of a severe stable slip deformity is with a modified Dunn, that is per trans trochanteric surgical hip dislocation and open reduction of the severely displaced epiphysis. There is, however, an additional inherent risk of potential AVN with this approach. The modified Dunn should be
performed only when there is existing known technical abilities, in particular, safely performing a surgical hip dislocation in the treatment of slip capital/femoral epiphysis deformities.\textsuperscript{1,2,4}

For most surgeons, the treatment of choice for these severe deformities will be initial screw stabilization with subsequent early surgical consideration of residual deformity to minimize the unnecessary occurrence of early (often problematic) degenerative pathology of the involved hip. Surgical strategies could include care per your institution and/or referral if other options should be considered in the optimal correction of the deformity. A big factor in consideration of additional surgery includes the functional demands of the patient and the patient’s habitus. More active patients, particularly in athletic activities, tend to destroy an impinged hip sooner than later. These patients are also better candidates for more major hip joint reconstruction. In contrast, morbidly obese patients are not necessarily good surgical candidates (or candidates at all) and unfortunately also make very little demands of their hip joint.
Pathologic Hip Morphology in Cerebral Palsy

**Epidemiology:** Cerebral palsy is a non-progressive disorder of the central nervous system with variable impairment of motor function. Hip dysplasia in CP is the second most common musculoskeletal deformity, second only to equinus.[1] The incidence and severity of hip dysplasia disease is directly proportional to the extent of neuromuscular impairment most often quantified by gross motor classification system (GMFCS). For example, 3-7% of CP patients who walk (GMFCS I-III) develop hip dysplasia as opposed to 70% of CP patients who do not walk (GMFCS IV-V).[2] Classification of hip dysplasia in CP patients combines acetabular and femoral morphologic changes in association with migration of the femoral head from the acetabulum and is described cumulatively in the cerebral palsy hip classification system (CPHCS).[3]

**Natural History:** Early improper development of the proximal femur and the acetabulum results in early instability (4-12 years of age). In milder cases, increased tone in later stages of development (often associated with hydrocephalus or shunt dysfunction) can also result in late subluxation or dislocation.[1] As the majority of hip pathology occurs in the more impaired CP patients (GMFCS IV-V) the report of long term outcomes has been infrequently cited as this patient population is more likely to sustain early death and less likely to have the capacity to localize pain.[4, 5] From reports available on long term outcome it is estimated that approximately 50% of patients with CP related hip subluxation or dislocation of the hip have hip pain after skeletal maturity.

**Soft Tissue Pathology:** Pathologic muscular forces (tone and balance) across the hip during development are thought to be the primary contributing factors to pathomorphology of the femur and acetabulum leading to hip instability. Increased tone in the hip flexors and adductors overpower the relatively weaker hip extensors and abductors leading eventually in a shift of the center of rotation of the hip from the femoral head to the lesser trochanter.[1, 2, 6, 7] Secondary to this characteristic muscle imbalance in combination with abnormal proximal femoral and acetabular development the majority of instability occurs in the posterior, lateral and superior direction.

**Proximal Femoral pathomorphology:** Change in femoral geometry is directly proportional to severity of neuromuscular disease (GMFCS level). The pathologic morphology has been reported by multiple observers and most commonly consists of increased femoral neck valgus and anteversion.[1, 2, 6, 7] Other pathologies may include deformation of the femoral head secondary to persistent pressure by the labrum, rim of the acetabulum or the abductors in the subluxed or dislocated hip and coxa valga from growth disturbances of the proximal femoral physis.

**Acetabular Morphologic Changes:** Pathologic development of the acetabulum is directly proportional to pathologic morphology of the proximal femur and therefore the severity of the neuromuscular disease (GMFCS).[1-3, 6, 7] Acetabular dysplasia occur secondary to alteration of muscular forces and migration of the center of rotation of the femoral head from the center of rotation of the acetabulum. These changes cumulatively result in primarily posterior and lateral deficiencies.
## Pathologic Hip Morphology in Down syndrome

**Epidemiology:** Trisomy 21, first described in 1866, is the most common chromosomal abnormality with an incidence of 1 in 795 live births. Life expectancy of a 1 year old child with Down syndrome is between 43-55 years. Hip instability reported to occur in 1.3% to 7.0% during childhood. Hip pathology increases to 28% of adult patients.[8]

**Natural History:** Down syndrome patients who develop hip dysplasia initially have stable hips before walking age. Progressive instability develops typically around 7-8 years old with subluxation potentially leading to fixed dislocation and/or osteoarthritis. Down syndrome patients with problematic hip pathology are more likely to become non-ambulatory with use of wheelchair.[8]

**Soft Tissue Pathology:** Capsular insufficiency, ligamentous laxity and hypotonia are the main contributing factors in Down syndrome hip dysplasia.[8] These connective tissue pathologies lead to altered dynamics of hip motion during development resulting primarily in a deficient posterior wall of the acetabulum (relative acetabular retroversion).[9-11]

**Proximal Femoral Morphology:** The pathomorphology of the proximal femur in Down syndrome remains a debate. The proximal femur in Down’s syndrome has been described as having only a slight increase of anteversion and near-normal neck-shaft angle[12] whereas other studies report an more significant increase proximal femoral valgus [13].

**Acetabular Morphology:** In Down syndrome patients with hip instability the morphology of the acetabulum has most often described is a deficient posterior wall which results in a shallow, relatively retroverted acetabulum.[8, 9, 11] In a study of Downs syndrome age and sex matched controls, the average acetabular version of the Down syndrome patient with subluxation or dislocation was significantly more retroverted than non-dysplastic normal children and patients with idiopathic hip dysplasia.[10]
Pathologic Hip Morphology in Cerebral Palsy

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<th>GMFCS I</th>
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Prevailing morphologic changes in hips of patients with cerebral palsy: Severity of disease is predicted by gross motor function classification system (GMFCS). The combination of muscular forces results in increased femoral anteversion (FNA) and valgus femoral neck shaft angle (NSA) resulting in an increased migration percentage (MP). Reproduced with permission and copyright © of the British Editorial Society of Bone and Joint Surgery [2].

Pathologic Hip Morphology in Down syndrome

Prevailing morphologic changes in hips of patients with Down's syndrome: Severity of disease is dependent on the combination of ligamentous and capsular laxity and hypotonia with changes in the acetabular morphology. Deficient posterior wall results in relative retroversion and posterior instability.
References

Stable SCFE
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Radiographic deformity

Mild (<30°)
Assess ROM both awake & EUA

Near 90° flex, min ER deformity?

Yes
Pin in situ

No
Pin in situ +/- osteochondroplasty

F/U → observe function, ROM & radiographs

Asymptomatic

Symptomatic, ↓ flex, IR in flex, ↓ ABD, ER gait deformity

F/U for ? years

(Consider) Reconstructive surgery:
PFO (ITT) → ↑ Flex & IR
Osteochondroplasty → ↑ Flex
SHD → osteochondroplasty +/- PFO (ITT)

Moderate (<60°)
Assess ROM both awake & EUA

>75° flex w/o excessive ER deformity?

Yes
Flexion restricted & notable ER deformity

Surgical hip dislocation expertise?

No
Pin in situ (on occasion primary PFO & osteochondroplasty)

Consider early reconstructive surgery

Yes
PFO & osteochondroplasty or Consider referral for SHD → either mod Dunn or PFO & osteochondroplasty

Open physis: modified Dunn; Closed physis: PFO & osteochondroplasty

Remove hardware

Severe (≥60°)

Pin in situ

F/U for ? years

Symptomatic, ↓ flex, IR in flex, ↓ ABD, ER gait deformity

F/U for ? years

Yes

Remove hardware
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12. Wenger DR. Personal communication.

13. Zaltz I. Personal communication, experience.


