OMC JOURNAL CLUB

7 APRIL 2010

LOGAN MITCHELL

WHEN AND HOW TO STRENGTHEN THE SUPERIOR OBLIQUE MUSCLE

SAUNDERS RA

JAAPOS 2009;13:430-437

BACKGROUND



- Author from Charleston, South Carolina
 - ~100 citations, PEDIG investigator
 - 5 previous articles on SO surgery
- Article = Costenbader lecture
 First presented at AAPOS 2009



DESCRIPTION



- Aim
 - Describe history of superior oblique strengthening procedures, and methods of titrating this
 - To determine any relationship between congenital onset and tendon laxity at time of surgery
- Retrospective chart review



INTRODUCTION



- Outline of historical reluctance to operate on SO
 - Complex anatomy and actions
 - Banister 1928 "out of the question"
- Early methods of SO strengthening
 - 1930's: plication
 - (loop of tendon sutured to sclera (Wheeler), or insertion (Foster), after *disinsertion* of SR)
 - (McLean): temporal approach no SR disinsertion
 - 1966 (Dyer): today's method
 - Single non-absorbable suture around fold of tendon





- All cases of SO tucking from 10 year period
 - 1980-85 and 2003-08
 - As notes missing from intervening years (!)
- Case definitions
 - SO palsy
 - Positive three-step test
 - Congenital case
 - "Early childhood" onset of strabismus or torticollis
 - OR later onset of above combined with facial assymmetry
 - Post-traumatic OR "undetermined" case
 - DID NOT USE LAX TENDON AS CLASSIFICATION





- Surgical technique
 - SO tendon isolated temporal to SR
 - Provisional tuck created with Bishop tucker
 - held with 5-0 Dacron suture
 - FDT performed (without globe retropulsed)
 - Adequate tuck when *first* resistance to elevation in adduction felt as inferior limbus passes imaginary line between medial and lateral canthi
 - Technique described by author in 1985
 - Tuck adjusted as needed
 - Final satisfactory FDT = -2 elevation in adduction









MODIFIED BISHOP TUCKER

- In 2005 author's group devised Bishop tucker with internal spring and scale
 - Allowing intraoperative measurement of tension created (0-200gm)
 - Globe held in depression to perform measurements
- Used on 12 "selected" patients
 - Both congenital and acquired
 - (not otherwise specified)



RESULTS



- 30 cases identified
 - 15 "congenital", 15 "post-traumatic" or "undetermined"
 - Average 20.4^ hyperdeviation in primary (range 2-55)
 - Second muscle operated on if required for deviation
 - N=23, including all of the "congenital" group
- Mean tuck performed
 - Congenital cases: 10.8mm (8-16mm)
 - Non-congenital cases: 7.8mm (4-12mm) - P = 0.002
- Modest correlation between tuck and deviation
 - R = 0.30 for primary and lateral gaze



RESULTS



- Tension measurements
 - Sample length-tension graphs provided
 - "Measurements reasonably repeatable when restesting performed"





- Increased tuck noted with congenital cases
 - But note large variation (up to 12mm in non-congenital cases
- Weak correlation between deviation and tuck required previously noted
 - When correlation noted, strongest for contralateral gaze measurements
 - Noted that Knapp was vague about in which gaze his > 20[^] rule applied
 - Author's conclusion that surgeon should aim to treat the contralateral gaze deviation and "the primary deviation should sort itself out"





- Previous guides at tuck titration ambiguous
 - Knapp: "Bishop tucker should be snug"
 - Scott: "cause a moderate Brown's"
 - Plager: "match the FDT of the normal fellow eye"
 - No data available on this method
- Conclusions
 - Congenital SO palsies (as per study classification) do have laxer SO tendons
 - Measuring tension of SO tendon induced by tuck may prove to be valuable way of titrating surgery





• Other points

- Author reports 3 over-corrections (iatrogenic Brown Syndrome) with SO tucking in his 30 year career (i.e. over and above study population)
 - Only one requiring take-down of SO tuck (which was included in this study)
- Most patients (not stated) in this study had a degree of limitation of elevation in adduction
 - Desired, often improves with time





- "When to operate"
 - One paragraph explains indications for SO strengthening in non-congenital cases
 - Acquired SO palsy (less common)
 - Marked underdepression in adduction
 - Torsional diplopia
 - Residual IO overaction post IO weakening (occasional)
 - Little data on this use



CRITICAL ANALYSIS



- Positive
 - Good case series operated on by one surgeon with consistent technique
 - Novel instrument devised
 - Giving previously unknown data regarding SO surgery
 - Expert, experienced opinion



CRITICAL ANALYSIS



- Data lacking
 - Post-operative measurements
- Case selection and classification issues
 - "Congenital" classification criteria debatable
 - How were 12 "modified tucker" cases selected?
- Potential measurement / technique issues
 - Variations in position globe held in during FDT
 - Scope for wide variation in tension measurements depending on globe position



CRITICAL ANALYSIS



- Negative points cont.
 - Analysis lacking
 - Length-tension curves
 - No grouped data presented
 - No comparison between congenital and acquired cases
 - » (?should be much larger in acquired cases whom author describes as having "normal tendons")



OCULAR TORSION: ROTATIONS AROUND THE "WHY" AXIS

KUSHNER BJ

JAAPOS 2004;8:1-12

BACKGROUND



- Burton Kushner
 - Director of Pediatric Ophthalmology, Wisconsin University
 - No introduction needed
 - 150+ publications
 - Founding Editor-in-Chief, JAAPOS
- Article = Costenbader Lecture
 - Presented at AAPOS 2003

DESCRIPTION



- "Traditional teaching is what you resort to when you don't actually know"
 - Amended from Kurt Adler
- Traditional teaching regarding superior oblique weakness and the Bielschowsky Head Tilt Test raises several inconsistencies
 - Addressed by literature review and "thought experiment"





TRADITIONAL TEACHING









- IO weakening for SO palsy should increase BHTT difference
 - It actually decreases ~5^





INCONSISTENCY #1(A)



- Unilateral SO tenectomy and IO extirpation for SO myokomia should give positive BHTT
 - It doesn't





LSO Palsy After Left IO Overaction

TABLE 1 Data on patients with SO palsy over the course of development of IO overaction

Data	Acute	Later	Paired
	(Mean ±	(Mean ±	Student <i>t</i>
	SD)	SD)	Test
Primary-position HT (PD)	8.6 ± 3.2	$14 \pm 4.9 \\ 3.4 \pm 0.53$	p = .0001
10 OA (0 to +4)	0.71 ± 0.49		p = .0008
Bielschowsky HTD (PD)	8.4 ± 2.6	21.3 ± 4.4	p < .0001

10 0A = inferior oblique overaction.

HT = hypertropia; HTD = head-tilt difference;

- BHTT should get less positive over time in SO palsy as IO overaction develops
 - It actually gets more positive
 - N=7 over 14 months





- BHTT should be just as diagnostic for vertical rectus palsies
 - It is not





- BHTT should be more positive in bilateral SO palsies as forces that cause it for each eye should be additive
 - It is actually usually much less positive





- Close observation of a globe in a tilting head should show a smooth intorsion/extorsion on ipsi/contralateral tilt respectively
 - Observation actually shows a series of cog-wheel like torsional movements (both in- and extorsional)



WHAT ACTUALLY HAPPENS?

- Does torsion occur on head tilt?
 - Yes, seen in 1786 (Hunter)!
 - But it does not completely correct for head tilt
 - Usually 50% of head tilt is corrected by torsion
 - "partial compensatory countertorsion"
- Close dynamic recordings of ocular torsion on head tilt have been made
 - Petrov and Zenkin 1973
 - Kushner and Kraft 1983



WHAT ACTUALLY HAPPENS?



- "Compensatory dynamic counterrolling"
- "Anti-compensatory torsional saccades"
- "Static countertorsion"



WHAT ACTUALLY HAPPENS? PART 2

- The anticompensatory torsional saccades are not 'seen' by the eye
 - Same suppression mechanism as for horizontal saccades
- Yet final anticompensatory torsional movement can be visualised
 - (after-images, Bagolini glasses)
 - Probably a different mechanism to saccade
 - Hypothesis: final anticompensatory torsional movement mediated by *relaxation* of SO (extorsion) and IO (intorsion)



INCONSISTENCIES REVISITED

- Remember
 - Interplay of dynamic and static compensatory torsional movements
 - IO muscle cannot raise the globe above the midline when SR disinserted
 - And same probably holds true for SO and depression
 - Contracture vs overaction



INCONSISTENCY #1 REVISITED



- IO weakening for SO palsy should increase BHTT difference
 - It actually decreases ~5^
- LIO is active in LSO palsy to produce anticompensatory torsional saccade (extorsion) on L head tilt
 - When overacting may overpower LIR increasing LHT
 - Post-surgery LIR is relatively unopposed on LHT thus decreasing BHTT positivity



INCONSISTENCY #4 REVISITED

- *BHTT should be more positive in bilateral SO palsies as forces that cause it for each eye should be additive*
 - It is actually usually much less positive
- The forces that cause the BHTT measurement are antagonistic for each eye (compensatory torsional movements vs anti-compensatory torsional saccades) and thus cancel each other out



WHY DOES TORSION OCCUR ON HEAD TILT?

- Dynamic counterrolling movement minimises retinal slip and subsequent peripheral visual degradation
- Anti-compensatory torsional saccades occur to preserve convergence and stereopsis
 - If 90 torsion occurred, convergence would need to be mediated by IR and SR
 - Too great a vertical disparity will not allow fusion



Convergence with Tilt: No Torsion Convergence with Tilt: Complete Torsion



Interestingly torsional movements are greater in lateral-eyed and elongated-fovea animals (eg rabbit)



ANALYSIS



- A great read!
- Expert, intelligent and logical thought
- Well-written
- A reply to Dr Jampel describes the technique to display the static ocular counterrolling oneself
 - Using retinal afterimage and Maddox rod or Bagolini lenses



PARETIC SIDE/NORMAL SIDE RATIOS OF CROSS-SECTIONAL AREAS OF THE SUPERIOR OBLIQUE MUSCLE VARY LARGELY IN IDIOPATHIC SUPERIOR OBLIQUE PALSY

UCHIYAMA E ET AL

AM J OPHTHALMOL 2010;149:508-512

DESCRIPTION



- Okayama University Medical School, Japan
- Aim
 - To search for a new definition of muscle hypoplasia in congenital or idiopathic SO palsy
- Retrospective case-control study



INTRODUCTION



- Genetic background of congenital SO palsy suspected
 - Familial cases
 - Muscle hypoplasia or aplasia
- Recent use of MRI to assess SO muscle preoperatively
 - Proposed use of muscle hypoplasia to classify palsy as congenital
 - But no standard definition of SO hypoplasia





- 98 charts reviewed
 - Patients diagnosed with congenital/idiopathic SO palsy at Okyama University Hospital 1999-2008
 - [NB: patients with `known'-cause acquired SO palsies not included]
- 50 patients had available MRI imaging
 - Varying study protocols/centres
 - Eyes closed during imaging
 - Mean age 30.9 years (range: 2-80)
- Coronal T1 slice nearest to globe-optic nerve junction photographed
 - SO and recti muscle areas measured x 5, averaged
 - Left:right ratios calculated for each muscle





- Controls
 - 45 patients having undergone orbital imaging for other reasons
 - Mean age 51.0 significantly older
 - Same EOM area calculations



RESULTS



- 5 patients (SO palsy group) with SO aplasia
- Mean CSA ratios (95% CI)
 - SO palsy group
 - SO: 0.66 (0.57-0.75)
 - SO excluding aplasia cases: 0.73 (0.66-0.80)
 - Recti: 1.00 (1.00-1.00)
 - Control group
 - SO: 1.00 (0.99-1.00)
 - Recti: 1.00 (1.00-1.00)





- Most studies classify SO hypoplasia if CSA < 50% of contralateral side
- This study shows large variation in paretic SO CSA ratios, but virtually none in control patients
- Thus potential definition of SO hypoplasia:
 - If CSA ratio paretic side:nonparetic side < 0.99

- Limitations of study
 - Control group from varied clinical settings, different age
 - Different imaging protocols, slice positions
 - Eyes closed during imaging not true coronal slices
- May lead to better classification of congenital SO palsy and provide better basis for genetic analysis of disease



ANALYSIS



- Possible merits in using acquired SO palsy cases as controls
 - To investigate utility in differentiating congenital vs acquired cases



MRI IMAGING OF FAMILIAL SUPERIOR OBLIQUE HYPOPLASIA

KIM JH AND HWANG J

BR J OPHTHALMOL 2010;94:346-50

DESCRIPTION



- Seoul National University
- Aim:
 - To document to familial occurrence of SO hypoplasia for the first time
- Case series



INTRODUCTION



- No previous MRI documentation of familial SO hypoplasia
- Study describes pedigree of 3 patients aged 1, 7 and 27 yrs old at time of study





- Ophthalmological and orbital MRI examination of 3 patients in pedigree
 - All with ipsilateral SO under- and IO overaction, torticollis and positive head tilt test
- 3T MRI imaging performed under standardised protocol



RESULTS



- Moderate to severe hypoplasia of SO (tendon and belly) identified in all 3 cases
 - All other muscles normal



SUPERIOR OBLIQUE PALSY IN CASE 1.







SUPERIOR OBLIQUE PALSY IN CASE 2.



Kim J H, Hwang J Br J Ophthalmol 2010;94:346-350



SUPERIOR OBLIQUE PALSY IN CASE 3



Kim J H, Hwang J Br J Ophthalmol 2010;94:346-350





- First description of MR imaging of familial SO hypoplasia
- AD inheritance previously proposed for congenital SO palsy
 - This pedigree may display AD inheritance with incomplete penetrance



A SURGICAL CASE TO DISCUSS

MASTER SD, AGE 6



HISTORY



- 6 yr old, underweight Indian boy
- Mother notes LET since age 3-4/12
- PMHx: MVA age 3 facial paralysis, facial fractures



EXAMINATION



- 10/08
 - Unable to assess VA
 - PCTN 16-18^ LXT, 6^ LHT
 - PCTD 25-30^ LXT, 6^ LHT
 - A pattern
 - Emmetropic, normal fundus



- 12/08
 - LXT ~50%
 - Equal, good VA
 - LHT worse on left gaze
 - RIO underaction
 - [possible R Brown syndrome]





• 1/09

- PCTN: AXT 40-45^
- PCTD: AXT 35-40^
- EOM: R Brown's
- Trial of +1.5DS lens to see if control improves
- 4/09
 - Glasses no help
 - Bilateral IO underaction noted
 - Again, A-pattern





• 6/09

- Still no amblyopia
 - (Lang consistently negative)
- Fuses with 12^ prism
- ?global developmental issues
- Referred to paediatric neurology
 - No issues





• 3/10

- No change, no amblyopia
- PCTN: 10-12^ LXT, 3^ LHT
- PCTD: 14^ LXT, 5^ LHT
- Bilateral IO underaction, SO overaction, IR underaction
- My examination
 - 12^ LHT, 25^ XT, >40^ on downgaze



PLAN?



- ? Diagnosis
 - ?Right / ?Bilateral Brown Syndrome
 - ?should be V-pattern
 - ?XT with Brown's
- ? Management
 - No amblyopia
 - No stereopsis
 - Variable measurements

- Surgery?
 - Would need FDT first
- ? Merit in observation
 - Cosmetic issue
 - Role of orbital imaging?



INCONSISTENCY #2 REVISITED

- BHTT should get less positive over time in SO palsy as IO overaction develops
 - It actually gets more positive
- LIO recruitment in LSO palsy on left head tilt required for anticompensatory torsional saccade (extorsion)
 - As LIO overacts will overpower LIR, giving LHT
 - ?should balance increased LIO action on right head tilt
 - (equalising BHTT)
 - But remember LIO won't elevate above midline when SR inhibited as on right head tilt



INCONSISTENCY #3 REVISITED

- BHTT should be just as diagnostic for vertical rectus palsies
 - It is not
- Consider LIR palsy
 - BHTT would require LIO to elevate globe in right head tilt to decrease L hypotropia
 - This elevation may not occur due to the weak elevating action of IO alone

