Answers to Audience Questions - WSPOS Worldwide Webinar 3:



Brown Syndrome - My Way



Burt Kushner (BK), Paolo Nucci (PN), Pauline Santiago (PS), Sumita Agarkar (SA), David Granet (DG), Kenneth Wright (KW)

1. Partial tenotomy of superior oblique posterior fibres – opinion?

BK: Not for Brown Syndrome where I think the entire tendon (anterior and posterior) is tight. OK for minimal weakening of the SO vertical fibers where you want to retain torsional action.

PN: I reserve this procedure to the conditions in which a mild to moderate superior oblique overaction is associated with A pattern strabismus without ocular intorsion

PS: I reserve partial 3/4 tenectomy described by Art Rosenbaum for significant A-pattern associated with significant superior oblique overaction. This supposedly preserves the anterior torsional fibers. There are however reports for example by Velez et al (JAAPOS 2006) who used this for Brown syndrome with small hypotropia in primary and reported good results.

SA: Doesn't work very well for Brown syndrome

DG: Not for Brown Syndrome. The proximal fibers can be shaved as shown.

2. What is the incidence of Brown Syndrome in your practice?

BK: Relatively low and about the same as Duane Syndrome. Can't give firm number as am now retired from regular practice

PN: 1 over 200 strabismus I see, but my place is a tertiary referral clinic

PS: Extremely rare.

SA: May be 10-12 new cases a year but then ours is a referral care hospital and there are 6 Pediatric ophthalmologists. It is a rare entity

DG: I will guess and say about 1 a month.

3. If congenital Brown's is a dysinnervation syndrome or cong fibrosis syndrome, why do some cases resolve? I've personally seen two cases resolve in my 25+ year career

BK: If it is indeed dysinnervation I would not expect resolution. There appear to be multiple different causes for Brown

PN: I am not convinced at all that Brown syndrome is a dysinnervation problem, despite some ccdds can simulate an anomalous V pattern and recall Browns Sy

PS: Brown syndrome just describes the constellation of findings of deficiency of elevation in adduction, hypotropia especially in the gaze opposite the involved eye, and overaction of the superior oblique. The causes are probably most

likely multifactorial: 1. Truly congenital dysinnervation syndrome that will most likely not resolve; 2. Anatomic variations in the trochlea; 3. A tendon sheath problem; 4. An inelastic tendon; 5. A "bursitis" as Dr. Nucci discussed. That is why some resolve and some don't, some respond to steroid injections, some to exaggerated forced duction and some to surgery.

SA: I believe Browns syndrome is just a morphological description of a pattern of limited motility. It results from several mechanisms all involving abnormal trochlea- tendon interaction or anatomical variations hence there is a subset which becomes better. I do not believe it is a dysinnervation syndrome

DG: I believe Brown Syndrome is a final common pathway for an inelastic tendon, short tendon, small trochlea or dysinnervation. Given the multiple causes it is not a surprise the same approach does not always work, nor that the outcomes vary across the board.

4. Can we use absorbable suture for split tendon elongation?

BK: I personally always use non-absorbable for the SO tendon. But I know others who use Vicryl for Harada Ito and get good results. So perhaps it would be OK. But I would not try it personally

PN: It's risky, 5-0 Mersilene is an easy and not so expensive suture to use

PS: I am not sure as I am a recent convert of the split-tendon elongation and excited to try it. Presumably the usual absorbable sutures will absorb in 2 weeks thereabouts? The patients are too few (unlike our regular rectus muscle procedures where studies have been done) to know if both ends of the muscles have combined into a unit after the suture absorbs. Also your superior oblique tendon may be tight and if sutures absorb, may convert into a superior oblique tenectomy...and will actually cause the dreaded superior oblique palsy. I would prefer to use the non-absorbable suture.

SA: Non absorbable suture would be more secure

DG: Don't know. I use non-absorbable.

5. Dr. Kushner, it was excellent talk; how do you grade the split tendon procedure?

BK: For most cases I do about 6 mm. If mild, I would do about 4 and if really severe about 8. Keep in mind the total lengthening will be about 1 mm less than the distance between hemostats, because of the approximately ½ mm of tendon that you need to leave at the sutured end so it does not pull out.

PN: 6 mm.

DG: I do about 6 mm., repeat fds and see if it needs to change. I aim for slight under-correction.

6. If the reason of the Brown's syndrome is a short tendon, is elongating the tendon the only way to treat it?

BK: There are probably multiple causes of Brown.

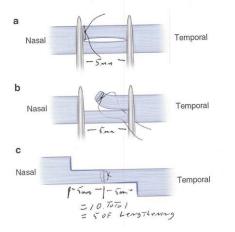
PN: I do not think is a shorter tendon, my opinion is that can be caused by an alteration in the course inside the Trochlea or a difficult telescopic movement of the muscle fibers in the tendon. See an old article by Helveston (Fig. 4) published in the great Paul Romano's Journal *Binocular Vision & Strabismus 1999; vol 14:15-26*

PS: It is believed to be one of the mechanisms but is NOT the only mechanism (See answer to question 3). If it is a shortened tendon, I agree, lengthening it, either with the spacer technique or the split tendon lengthening technique should work... And it does work for many patients with Brown syndrome.

SA: It could be several things but elongating tendon-by way of tenotomy, splitting or a spacer seems to work, at least to a certain degree, in patients with Brown's syndrome

7. For the split tendon lengthening, the tendon lengthens by twice the distance between the hemostats. Is that correct?

BK: No, it lengthens by just the distance between. One of the ½ tendon slips does not represent "lengthening." If you draw it out and measure on paper, you will see. In this figure, the tendon is split 5 mm. The total length of the connected 2 half tendon slips at the end of the procedure is 10 mm, for a lengthening of 5 mm.



PN: If you put the hemostats 4 mm apart you obtain a 4 mm elongation (I do standard 6mm)

PS: From what Dr. Kushner discussed, if you separate your hemostats by 5 mm, you lengthen the tendon by 5 mm. Remember there is a pre-existing length of the tendon.

SA: No

8. Dr. Kushner: what is the distance between the superior rectus and the temporal hemostat?

BK: I put the temp hemostat just adjacent to the SR

9. Shaving of the SO is indicated for Brown's without deviation in PP. You have to pull the tendon and shave the tendon that is before the trochlea. Is that correct?

DG: That is correct.

10. The shaving technique is effective in short tendons?

PS: I have not used the shaving technique. But presumably it seems to work by 2 mechanisms I can think of: 1. A partial tenectomy, reducing the superior oblique overaction; 2. Thinning the muscle allows it to scaffold in and out of a tight (or swollen?) Trochlea. Not quite sure about lengthening effect of the procedure, perhaps Dr. Granet can answer this better.

DG: I believe Brown Syndrome is a final common pathway for an inelastic tendon, short tendon, small trochlea or dysinnervation. Given the multiple causes it is not a surprise the same approach does not always work, nor that the outcomes vary across the board.

11. Someone should emphasize the importance of using Guyton exaggerated FD after the tenotomy before the spacer, chicken stitch, tenotomy, etc. To document complete tenotomy since even a single fibre of posterior tendon that is missed prevents any benefit from the procedure!

BK: Agree

PN: Useful technique that put the SO under stress (decrease the angle of reflection) just retropulsing the globe: useful in perceive the "bump or jump". After releasing the muscle, you should see no "bump/jump" anymore

PS: Dr. Granet discussed forced duction testing and he showed it, and how sometimes it can be therapeutic for Brown syndrome. Every case starts with an exaggerated forced duction test.

SA: Agree

DG: I use it in Brown Syndrome and in most strabismus!

12. Many said, an indication to operate in Congenital non-committent strabismus is deviation in PP. I think we should add to it (with lost binocularity and no compensatory AHP)

BK: Agree. But in almost all Brown they do adopt a head posture to fuse, unless there are other factors like anisometropic amblyopia.

PN: of course yes, but I must confess that I hardly remember a patient with Brown syndrome without stereocuity

SA: My indication to operate would be hypotropia of 15 PD or more and a significant Anomalous head posture

DG: Sure, but this is not the major issue.

13. I want to repeat Dr. Kushner's question - up to what duration of disease would you consider intratrochlear steroids; for example, if a child comes with onset 2 years prior to your appointment with only observation done in between, would you consider intratrochlear steroids?

BK: I know I have had success 6 months after onset, but also wonder about others.

SA: I would at least try a course of steroid or safer, an injection, before proceeding to surgery. Effect of injection is pretty much seen in 3-4 weeks It's worth waiting 3 weeks more if you could potentially avoid surgery

DG: On CT if there appears to still be inflammation I would inject.

14. How often have you seen an intermittent Brown's syndrome?

BK: Only a few times and always with a click syndrome.

- PN: Not many, I can say less than 10, but for sure is an entity that exists
- PS: Have not seen one, but can imagine it is possible.
- SA: I haven't seen in my practice

DG: A handful

15. How long does the effect of the steroid last before it wanes & the symptom returns?

BK: It varies. I have treated 14 patients. In 12 I ultimately got longterm resolution. The only 2 that were not successful had anatomic issues e.g. A Marfan's with dislocated trochleas. Of the 12 successes, one only needed one injection, 6 had one recurrence and needed a 2nd injection, and 5 needed 3 injections. Typically, the first one lasts a month or so if it recurs, the second one lasts longer, either indefinitely or 3-6 months, and none had a recurrence after a third injection.

SA: Adults often have waxing and waning course specially if associated if arthritis or SLE but they often respond to NSAIDs also. In children I haven't had need to give a second injection.

16. What is your approach for canine tooth brown syndrome?

BK: Hard to fix. The only thing I think is possible is a tenectomy of the SO to fix the restrictive Brown and a simultaneous large myectomy of the IO to treat the SO palsy. Success is a function of how badly and where it is scarred.

PS: Observe if no primary position deviation. Excise as much scar tissue if possible.

DG: Tenectomy and IO myectomy can work fairly well.

17. Can the panel speak about SO recession?

BK: Recession following the normal course of the SO has the problem I mentioned. It puts the insertion anterior to the equator and will cause the inverse of the IO anterior elevation syndrome – specifically a restriction of depression in adduction.

PN: I think it is an awkward procedure: difficult to manage the lateral tendon, difficult to reposition it and also you can create an unwanted adhesion with the fibers of the SR

PS: Always wondered about how SO Recession is done properly. Dr. Kushner has discussed it somewhat during the webinar, mentioning that SO recession may have an effect akin to anterior transposition converting it also to an elevator, thus the need to put the muscles a little bit more posterior than its path. I personally have not done it.

SA: I think it was answered by Dr Kushner in the webinar I personally don't have experience with SO recessions. SO recession will change the insertion to nasal and anterior which will change the action of superior oblique. It leads to limitation depression which is problematic as it affects the downgaze

DG: I like this procedure. As mentioned in the webinar one must also recess the insertion or the SO becomes an elevator. I put the SO on an intraoperative adjustable suture, repeat the fds and set it where I am just under corrected. Then I tie it off in the OR before finishing the case.

18. For Dr Kushner: which part of tendon do you keep attached to sclera. Ant or posterior?

BK: Both ant and posterior are still attached to the sclera after the procedure, because the insertion is not disturbed, and has both anterior and posterior fibers. I do not think it matters if it is the anterior or posterior fibers are the ones that are tenotomised at the nasal end of the split

19. Which suture do you use for SO split tendon lengthening? Is there chance of suture slipping?

BK: I use 6-0 dacron or mersilene. Suture needs to be tied securely in tendon halves to prevent slippage.

PN: 5-0 Mersilene. I do not think can happen

- PS: I prefer to use Dacron 5-0 or Polyester 5-0 but the needle is still too large
- SA: A non-absorbable suture would be better

DG: Hard to get 6-0 so I often end up with 5-0 non-absorbable.

20. What's the risk of rupture and losing the tendon in split tendon elongation?

BK: Low. The hemostats should hold the tendon securely and they are not released until after the sutures are passed thru both halves.

PN: never happened to me

SA: Unlikely as you are splitting and working with only half the tendon though it can be a difficult procedure

21. Dr. Parks showed that SO sheath is a technical artefact, in reality it does not exist. I would like to know the opinion of the panel.

BK: I agree with Parks

PN: See an old article by Helveston (Fig. 4) published in the great Paul Romano's Journal *Binocular Vision & Strabismus 1999; vol 14:15-26,* describes nicely the physiology and the Anatomy of the SO

PS: combine with #3

22. Dr. Kushner, I am concerned if the hemostats could damage the tendon and cause atrophy of the segment in between. Your comment?

BK: I have never worried about that. The tendon is actually tougher tissue than muscle, and we clamp muscles for resections and myectomies.

23. What's the panel's take on silicon band expanders compared to chicken sutures?

PS: I prefer the silicon band spacer (Wright) over the suture spacer (Suh et al) over the "chicken" sutures (Knapp), but certainly all can be used for Brown Syndrome. Banked sclera can also be used as a spacer.

SA: I prefer to use chicken sutures

DG: I live close to Los Angeles and anyone where the spacer was an issue or wanted I sent to Ken Wright! I think that procedure requires experience, so I took advantage of his proximity.

24. What needle (gauge) you use to inject betamethasone?

- PN: 28 G needle
- PS: Gauge 27-30
- SA: 30 gauge or 27 gauge

DG: 27 G

25. I have performed the "Chicken suture" technique for a few cases and always have noticed it takes several months to work (it is not an immediate result). Is that the opinion of the panel for this or the other surgical procedures?

BK: Limited experience with chicken suture. Do not think that the case with split tendon

PS: May be postoperative inflammation.

SA: Yes, I agree

26. Does anyone use traction suture after surgery?

BK: No. I think this was advocated more at a time when people were just operating on the so-called sheath, which did not really free up forced ductions.

PS: I don't.

SA: No

DG: No

27. Dr Kushner: what to do when your surgery doesn't work?

BK: I have not had a case where this failed in Brown Synd. But if it did, I would have to go back and see what the problem is, and do what is needed to free the forced ductions.

SA: You can go back and do a tenotomy I reckon

28. Have you converted your elongation procedure to SO tenectomy because of difficulty in dealing with thin half tendon?

BK: Have not had to.

SA: That could be an option

DG: Once.

29. When would it be recommended to use the technique described by Dr. Kushner?

BK: I use it for all brown needing surgery, or for any SO OA that is more than mild (+3 or +4)

PN: this is my preferred technique now

SA: It's a personal choice and comfort, all techniques work. Dr Kushner's technique has advantage of not using any artificial spacer and reduces risk of SOP but it is difficult

DG: Surgeon facility with a procedure matters.

30. Dr. Kushner, does the split tendon lengthening gets adhered to the globe again? That was a concern.

BK: I have not seen it adhere

PN: It seems unlikely

31. Maybe did not observe much risk of SOP with SO tenotomy / tenectomy itself too?

BK: Not sure what you are asking. SOP typically occurs with SO tenectomy or tenotomy, but can be avoided with simultaneous IO weakening

PN: I think that the figure of 80% reported by Dr Wright is a bit higher with respect to my personal experience that anyway account for 40-50%, and pushed me toward lengthening procedures

PS: We have described a series of patients with "Grave complications following superior oblique tenotomy/tenectomy for Brown syndrome. J AAPOS. 1997 Mar;1(1):8-15

DG: In that setting the IO must be approached.

32. What can we do if we slipped or cut one half (this happened to me previously)?

BK: I am not sure what you are describing or with which procedure

PN: Both the oblique muscles do not retract; they are the easiest to retrieve

PS: Not sure I understand the question... If you cut half the tendon... That's just half the procedure of Dr. Kushner's split tendon lengthening and should be salvageable?

SA: you could convert it to tenotomy or chicken suture

33. Dr. Kushner: Do you open the tendon sheath first to get access to the SO tendon to place the suture?

BK: No

34. Microscope?

BK: high powered loupes. 3.6x or 4.5x

PN: Always for me, but I do not press the residents if they are more comfortable with the loupe

DG: Loupes.

35. Have you had a recurrent restriction after several weeks of successful resolution? I have had this in about 20% of my lenghtenings.

BK: I have not

PS: In my limited series, have not had to redo, extend spacer, or take down a spacer. I follow Dr. Ken Wright's table.

SA: I have had this in about 20% of my lengthening. I haven't done split tendon lengthening but yes scarring post surgery could lead to recurrence

36. How do we decide how many mm. to elongate? Would it be based on the degree of motility limitation?

BK: For most cases I do about 6 mm. If mild, I would do about 4 and if really severe about 8. Keep in mind the total lengthening will be about 1 mm less than the distance between hemostats, because of the approximately ½ mm of tendon that you need to leave at the sutured end so it does not pull out

PN: No grading, if surgery is needed I elongate standard 6 mm

SO Overaction	A pattern (PD)	Length of spacer (mm)
+1	20-30	4
+2	30-40	5
+3	40-50	6
+4	>50	7

PS: Based on Ken Wright's recommendation, I showed this table:

SA: 6-8 mm should be enough

37. I was in Toronto during Dr Crawford's last year of life 1989-1990. Indeed, he described simultaneous IO weakening with SO tenoectomy / tenotomy, but eventually stopped since there was always further surgery for the SO palsy.

PN: Yes, Bob, I totally agree. I tried the simultaneous procedure but I had immediately the paradoxical symptoms of SOP palsy

DG: Agree, that's why these other procedures were developed.

38. I have done many with Chicken suture and they work well if not too long or too short.

SA: That's my preferred way too

39. Please can panel make very clear which cases would be conservative e.g. If orthodontic and stereo in primary gaze - conservative, despite poor cosmesis in elevation?

BK: If ortho and fusing in primary I would be conservative

PN: I am quite "tranchant" in this surgery, indication for me is if the Anomalous Head Posture is relevant and strongly influence daily activities. I cannot recall cases of Binocular Vision problems that indicate surgery in this group of patient. They are strangely protected and even amblyopia is quite rare.

PS: Indications for surgery in Brown syndrome: Primary position hypotropia, Severe downshoot on lateral gazes, anomalous head posture; loss of a prior anomalous head posture indicative of amblyopia and/or suppression. Any patient that does not meet criteria, not operated on. All patients should have the benefit of LOOONG observation period.

SA: I would operate only if there is significant head tilt or chin elevation or a hypotropia of more than 15 pd. Cosmesis in certain gazes should not be decision making factor

DG: Patience! Discuss repeatedly with the family.

40. Do the panel feel that the risk of SOP is equally high with Tenotomy as well? Somehow in our experience we don't see so high incidence of SOP post-operatively.

BK: Don't understand question. Equally as high with tenotomy as with what other procedure? If you mean tenectomy, it is not as high with tenotomy (i.e. Worse in tenectomy)

PN: I think that the figure of 80% reported by Dr Wright is a bit higher with respect to my personal experience that anyway account for 40-50%, and pushed me toward lengthening procedure

PS: I think every time you perform surgery on the superior oblique there is a risk of creating iatrogenic superior oblique palsy (unless you perform a tuck). This is greatest on SO tenotomy and tenectomy as we described in publication: "Grave complications following superior oblique tenotomy/tenectomy for Brown syndrome. J AAPOS. 1997 Mar;1(1):8-15

Less in tenotomy, worse in tenectomy, but grave complications nonetheless.

SA: Somehow in our experience we don't see so high incidence of SOP post-operatively. Risk of SOP exists and should be discussed with patients and family before planning tenotomy percentages don't matter

DG: Likely the tenotomy means recession as the tendon scars back down.

41. In Iowa we were taught something called intrasheath tenotomy. Any opinion on this?

BK: I think the sheath is an artefact. It does not matter if you open the "sheath" or not.

PS: It's just performing tenotomy inside the "sheath." It is still a tenotomy and one needs to be wary of SO palsy.

42. What are possible complications of tenotomy?

BK: SO palsy

PN: After a while, and not immediately, they can present the SOP palsy symptoms (1 out of 2 patients in my previous experience), with immediate worsening of the reading performance. Tough to resolve if the patient is older than 8 yrs.

PS. We reported this... It is bad superior oblique palsy that is difficult to correct because you can no longer retrieve the tendon. "Grave complications following superior oblique tenotomy/tenectomy for Brown syndrome. J AAPOS. 1997 Mar;1(1):8-15

SA: Superior oblique palsy requiring further surgery is the worst complication apart from undercorrection

43. Do you think there is any difference in terms of results if tenotomy is performed close or far from the trochlea? I think so.

BK: Closer to the trochlea, the more the effect. Helveston showed that the SO attaches to the globe all along the path of the reflected tendon via indirect connections. The closer to the trochlea you cut, the more of these attachments are taken out of action.

PN: It seems more effective closer to the trochlea, but no comparative experience

PS: I would not do tenotomy much less a tenectomy. "Grave complications following superior oblique tenotomy / tenectomy for Brown syndrome. J AAPOS. 1997 Mar;1(1):8-15

DG: See BK's answer.

44. What is your experience with tenotomy and IO weakening as advocated by Dr. Parks?

BK: In general, good; but some had a hyper of the affected eye in downgaze later on, necessitating further surgery usually on the contralateral IR

PN: I tried the simultaneous procedure but I had immediately the paradoxical symptoms of SOP palsy

PS: Certainly if the patient comes in after a prior tenotomy, and I see a superior oblique palsy, my first procedure will most likely be an IO weakening procedure. I will not do a full tenotomy or tenectomy for Brown syndrome.

SA: I would not do a simultaneous surgery because not all patients with tenotomy develop SO palsy

DG: I have used a Z myotomy of the IO in that setting. Can do more.

45. What are the different approaches to isolate the muscles?

PS: I open temporal to the superior rectus but extend circumferentially nasally to access the superior oblique tendon nasally. There is a different opening for a posterior superior oblique tenectomy I use for A-pattern deviations. These superior oblique procedures are described in our textbook Clinical Strabismus Management in the chapter Superior Oblique Procedures. The book is freely available and downloadable on books.google.com.

46. Do you not think triamcinolone is better than dexamethasone that is long acting?

PN: If you think there is an inflammatory reason, one shot of steroid is enough, unless you do not consider the bursa an area of chronic bursitis (as in JIA), in this case systemic treatment is more appropriate with respect a long acting agent ... But this is my humble, possibly wrong, logic.

PS: Triamcinolone is long-acting and will probably reduce the number of times it will recur, but if you get secondary steroid-induced glaucoma it will be difficult to excise your depot, and you will have a very difficult glaucoma to manage.

SA: Yes; it is longer acting

DG: Sure - there are several choices.

47. Do anyone advocate both SO and IO surgery at the same sitting for Brown syndrome?

BK: Not any longer though I did it for a while. See question 55

PN: I tried the simultaneous procedure but I had immediately the paradoxical symptoms of SOP palsy

PS: I don't. It is not necessary with the spacer procedure, following Dr. Ken Wright's algorithm.

SA: No

DG: I evolved away from that.

48. Dr. Nucci, I abandoned the SO tenotomy after seeing cases of severe SO palsy. What is your experience?

PN: I think that the figure of 80% reported by Dr. Wright is a bit higher with respect to my personal experience that anyway account for 40-50%, and pushed me toward lengthening procedure

49. How do we manage Brown's syndrome?

PS: (1) Observe (2) Medical management with intratrochlear injection of steroids (3) Surgery for recalcitrant cases; I use silicone spacer, may use suture spacer, chicken suture, split-tendon lengthening

50. Dr. Granet: Do you suggest to give perilesional steroids to all patients both for forced ductions and tenotomy?

DG: Yes.

51. Dr. David Granet: How many years you have observed the child following the relief from forced diction test? Any recurrence?

DG: A decade. I injected steroids a few times. He's done well.

52. In patients with intermittent brown's syndrome, what specifically will you look for in MRI Orbits & what specific investigations?

BK: Nodules or edema

PS: I would imagine you are looking for a "fibrosis" of the SO, or maybe a shortening of the tendon. But I do not request MR imaging of the orbits for Brown syndrome, and rely heavily on clinical measurements and forced duction testing.

DG: I scan for acquired Brown Syndrome more frequently. Edema and inflammation can be seen.

53. Did he have reduced depression in adduction Po?

SA: In the post op video depression was full

DG: In the video I showed, and in real life depression was maintained.

54. Usually Cong. Brown Syndrome does not resolve with FDT.

DG: I have n=2. Most do not resolve this way.

55. Do you do exaggerated FDT in congenital or acquired cases? How many times do you do the fdt before concluding it works or not?

PS: Exaggerated forced duction testing should probably be done in ALL patients with Brown syndrome regardless if congenital or acquired. Certainly for young patients who will not cooperate with forced duction testing in the clinic, all forced duction tests should be repeated just prior to surgery.

SA: I think you would know right away if it works because FDT would become negative

DG: I do them pre and post op in all.

56. Does the condition recur after FT?

DG: I injected steroids to prevent recurrence.

57. Can forced duction tried in all of Brown cases?

BK: they should be done for diagnostic reasons

- PS: Certainly. All cases should start with forced duction testing.
- SA: Yes, why not? FDT is a part of surgery anyway

DG: It should be a part of every surgery.

58. Could you show again how to inject trochlea?

PS: The webinar is posted on WSPOS's YouTube and Facebook pages.

59. Dr. Granet, when you do fdt, do you catch at 12 and 6 o'clock? What forceps do you use? Does anticlockwise movement suggest tight so palsy?

DG: I use Thorpes forceps. They handle conjunctiva better. The range of FD is wide. Some are much worse than others.

60. Do you perform the traction under full General Anesthesia or under Mask anesthesia?

DG: Expecting it to fail I use GA.

SA: GA in Children

61. Which cases we can do FDT only treatment for brown's?

DG: When it works! All cases should have exaggerated fds.

62. Do you inject steroid after exaggerated forced ductions?

DG: As mentioned, yes.

63. Why did the pupil dilate after the FDT Dr. Granet?

DG: We dilated the eye to look in!

SA: probably due to stretching of inferior oblique

64. Is steroid given in all patients with Brown's?

SA: No mostly on acquired cases with suspected inflammatory pathology

65. If there is a marked chin up posture, would you still wait for surgery?

BK: No

PS: Yes, at least a 1 to 2-year period for observation alone, but explain to parents some take 5-10 years. Chin-up posture means there is fusion and amblyopia less likely too

SA: Significant persistent head posture is an indication for surgery

66. What about injection of long acting steroid + short acting?

BK: That is my preference.

SA: no experience

67. Can u do a repeat injection?

BK: Yes. I have done up to 3

SA: yes, if symptoms recur

DG: Yes.

68. How much time after onset of acute browns can we take up patient for injection?

BK: I think once your work up is done there is no reason to wait, unless you try oral meds first, which in my hands has not worked. I have probably injected as soon as 3-4 weeks after onset

SA: Injection is effective in cases of acquired Brown syndrome with inflammation, so investigations like imaging and blood tests may be required before injection. If that's the pathology, you should not hold the injection

DG: I have used systemic anti-inflammatories if there is a delay getting to injection.

69. How many times can you give steroid injections if you don't see improvement?

BK: Usually if they do not respond to the first injection they do not to subsequent, but I have seen that on occasion. Usually repeat injection is for recurrence.

DG: What Burt said.

SA: I have given only once in all my cases

70. Can one fill trochlea by palpation?

BK: Yes

SA: yes, and you don't have to give in trochlea you can inject in trochlear region keeping needle away from globe

71. I would like to know how Dr. Wright feels about steroid injections in acquired, inflammatory Brown syndrome?

KW: Yes, I use local steroid injection in patients with acquired Brown Syndrome. Especially those cases where there is discomfort. Inject in the office in the area of the trochlea.

72. Which cases to give intratrochlear injection and which to give oral steroids?

BK: If I decide to use steroids, I always prefer the local route (injection)

PN: If you think there is an inflammatory reason, one shot of steroid is enough, unless you do not consider the bursa an area of chronic bursitis (as in JIA), in this case systemic treatment is more appropriate with respect a long acting agent ... But this is my humble, possibly wrong, logic.

PS: I will probably not give oral steroids, and if trying intratrochlear injection only the short acting dexamethasone, betamethasone, or methylprednisolone, NOT triamcinolone

SA: Acquired Browns with inflammation are candidates for steroids. Both oral and injection works. Injection is safer as it avoids systemic side effects of steroids but needs short GA in kids. Local diverse effects like elevated iop must be monitored

DG: Direct injection is my preference but will use oral if there's a delay.

73. After injection, do we give a course of systemic / oral steroids also?

BK: No

PS: I wouldn't.

SA: No

DG: No

74. How much steroid is injected?

BK: 1 cc.

PN: 1 cc. of Dexamethasone or Betamethasone

SA: 4 mg (1 cc.) Of betamethasone or Triamcinolone is in dose 40 mgr. (1 cc.)

DG: Total volume of about 1cc

75. What special instructions when you order for CT / MRI?

BK: I work with one specific radiologist with an interest in the EOM's. He has a protocol he uses if I just tell him I want the SO tendon visualized

SA: If suspecting Cysticercus it must have brain scans also to see any neurocysticercosis

DG: We have an orbital protocol for thin cuts etc.

76. Do you always order image on those patients? Even in congenital cases?

BK: No

SA: No not in congenital but always in acquired cases

DG: In acquired.

77. Albendazole in muscle cysts: duration of therapy?

SA: 15 mg/ kg wt. Plus oral steroid for at least 4 weeks. Oral steroids are essential as dying larvae can induce a severe inflammatory response

78. Congenital Browns associated with CNS abnormalities e.g. Corpus callosum agenesis or abnormal (mostly which I have seen); When do we operate? And do the results in these cases differ? I mean should our expectations be humbler of the post-operative outcome? In terms on ahp improvement etc.?

BK: Not sure I have experience with that

79. What simple practical tips can one use to differentiate brown syndrome from superior oblique palsy?

BK: With Brown there is a hypo of the affected eye on adduction and elevation in adduction, and with SOP there is a hyper.

PS:		
Parameters	SO Palsy	Brown Syndrome
Elevation in adduction	Usually overacting IO	Deficient
Vertical deviation	Hypertropia of involved eye	Hypotropia of involved eye
Superior oblique	Underacting	Overacting
Inferior oblique	Overacting	Underacting

80. What simple practical tips can one use to differentiate brown syndrome from inferior oblique palsy?

SA: Important differentiating point would be presence of an A-pattern / arrow-pattern in inferior oblique palsy vs Vpattern in Browns. Brown syndrome will have normal Superior oblique action and positive forced duction test while inferior oblique palsy maybe associated with superior oblique overaction and a negative force duction