HANDS-ON

Editor Jim Meadows

Editorial: Truth in Manual Therapy

A complaint commonly heard from the researchers of physical therapy is that clinical instructors frequently make claims for which there is no evidence. Often there is some truth to this but more usually the term evidence is being used in a form that is convenient for the complainant. Partly from this complaint comes the need for evidence based practice. On the surface this seems a reasonable requirement for all practice but the term "evidence based" needs to inspected in detail before clinicians give up 90% of their practice because there is no criterion or other high level evidence for the practice. First the clinician is not to blame fault for the paucity of high evidence supporting their practice. It is the job of the researchers to prove practice and the fact that there is so little validation of our practice together with good sensitivity and specificity numbers for our tests or experimental proof for out treatments is an indictment of how poorly our clinical-research teaming works in North America as compared with Australia. It not that those tests or treatments used by clinicians are not valid, but that they are just not validated nor invalidated. In these cases (the majority) other forms of evidence must be used. This evidence may take the form of construct, consensus, case studies, even opinion and while these forms may not be the strongest evidence there is they are frequently the only evidence there is. If we lived in a perfect world, the clinicians would go to the researchers with a question that followed an observation and the researchers would do their best to answer it. The clinician would then take the research and incorporate it into their practice. This not being the perfect world generally the clinician, especially the newer graduate must go to the experienced clinician and the instructors in manual therapy for the best evidence available. It is then that we run into the problem we started with, claims to truth that are not only unsubstantiated by research but more frequently have never been researched in the first place. The clinician is now at fault for stating as a truth what may be backed by lower levels of evidence but is rarely the truth. Even when there is experimental proof of a test or treatment this merely states a fact and not the truth. Truth in the philosophical sense and fact are not necessarily the same. The best examples of this dichotomy comes from the hardest of sciences, physics. Relativity and quantum mechanics are the two most proven theories extant, neither has failed to agree with obser-

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Courses in the Next Month

Nov 1-4	Supervised Clinical Practice	Detroit, MI
Nov 12-14	Acute MVA Patient	Boise, ID
Nov 26-28	Chronic MVA Patient	Saskatoon, SK
Dec 10-12	Cervical Spine	Baltimore, MD

For further information on courses contact jim@swodeam.com

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Inside this issue:

A theory is not accepted because of its merits, it is accepted because the authoritative figures opposing it die off.

Paraphrased from a giant in physics whose name escapes me at the moment.

Editorial Continued

vation and both have been proven to about 12 decimal places. Yet the underlying "truth" to each theory is not known. Physicists use models that incorporate the facts of these theories to form a framework that allows them to image the universe which allows them the best concitions to do their work. For quantum theories there are many models of how the universe work at a sub-atomic level none of which are of necessity compatible with any of the others. The point of these models is not to tell the truth but to explain the facts in an organized and rational manner. A good model incorporates all or most of the facts, a weaker model less facts and a downright bad model no facts and should properly not be called a model but fantasy . When a model fails to incorporate new facts as they are discovered then it is modified or replaced until the new version does so explain them.

Instructors of manual therapy would do well to follow this example and make it clear that what they are describing is a model and not the truth. This way we do not look silly and more importantly the specialty does not loose credibility when our "truths" turn out to be lies. One example of this in manual therapy can be seen with the explanation of how manipulation works (whether it does or not can be debated at another time). The two main explanations or models are the mechanical and the neurophysiological. The former simply holds that manipulation is effective by moving a joint that is fixed (and the exact mechanism of fixation is a variable part of that model) at one end of its range and eliminating the abnormal stress put upon a pain sensitive tissue. The neurophysiological model explains manipulation's efficacy by having the sudden mechanoreceptor input into the spinal cord segment and more central regions cause pain modulation and muscle tone relaxation and so decrease or eliminate pain and increase range of motion. In all probability there is an element of "truth" in both of the models but providing that the user understands that the adopted model is simply a working framework that helps visualize what is going on it does not really matter which model is chosen. You pays your money and you makes your choice and the truth may never be known.

Letters and Comments

None as yet.

Swodeam Courses 2004

2004	Course	Location
Jan 9-11	L2 Lower (A)	St Louis, MO
Jan 16-18	L2 Lower (B)	St Louis, MO
Jan 23-24	L2 Upper (A)	Houston, TX
Feb 6-9	L2 Upper (A)	Houston, TX
Feb 13-15	The Acute MVA Patient	Washington, DC
Feb 20-22	Lower Limb	Baltimore
Feb 27-29	L 3 Upper (A)	Madison, WI
Mar 2-4	Clinical Practice	Dallas, TX
Mar 5-7	L3 Upper (A)	Dallas, TX
Mar 12-14	Peripheral Manipulation	Colorado Springs, CO
Mar 19-21	L3 Upper (B)	Milwaukee, WI
Mar 26-28	L3 Upper (A)	Quebec City, PQ
Apr 2-4	L3 Upper (B)	Quebec City, PQ
Apr 16-18	L3 Upper (B)	Dallas, TX
Apr 23-25	The Chronic MVA Patient	Ithaca, NY
Apr30-May 3	Spinal Manipulation (A)	Boston (MA)
May 7-9	Spinal Manipulation (A)	Fremont, CA
May 14-16	Spinal Manipulation (B)	Fremont, CA
May 21-23	L2 Lower (A)	Portland, OR
May 29-31	Acute MVA	Quebec
Jun 4-6	Peripheral Manipulation	Ottawa, ON
Jun 11-13	Spinal Manipulation(C)	Fremont, CA
Jun 25-27	Spinal Manipulation (B)	Boston, MA
Jul 16-18	L3 Upper (A)	St Louis, MO
Aug 13-15	L3 Upper (B)	St Louis, MO
Sep 10-12	The Acute MVA Patient	Tacoma, WA
Sep 17-19	The Acute MVA Patient	Everett, WA
Sep 24-26	The Chronic MVA Patient	Ottawa, ON
Oct 1-3	The Chronic MVA Patient	Edmonton, AB
Oct 8-10	Lumbar Thrust	San Diego, CA
Oct 15-17	L3 Lower (Lower Quad)	Quebec City, PQ
Oct 22-24	L3 Lower (Lower Quad) 2	Quebec City, PQ
Oct 29-31	L3 Lower (A)	Detroit, MI
Nov 1-4	Clinical Placement	Detroit, MI
Nov 5-7	L3 Lower (A)	Detroit, MI
Nov 12-14	The Acute MVA	Boise, ID
Nov 26-28	Chronic MVA	Saskatoon, SK
Dec 10-12	Cervical Spine	Baltimore

All courses unless specifically stated are combinations of lecture and lab, usually about 50/50. Each course is organized by a local coordinator and for contact to that person please email Jim Meadows at jim@swodeam.com

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For further information on courses contact jim@swodeam.com

Alar Ligament Injuries. Part 2: The Art

Jim Meadows

Alar ligament injury is a relatively common occurrence in post-whiplash victims with frequently both ligaments being injured. The numbers of the grade 1 injury might be exaggerated by non-whiplash causes but the grade 2 and 3 injuries seem to be the result of whiplash. The injury can be visualized by appropriate MRI technique but will be missed if the technique is inappropriate[8].

Insufficiency of the alar ligament will produce and increase the average contralateral rotation at the atlanto-axial joint by up to 30% or almost 11 degrees[5]. Mechanical dysfunction of this region, either due to alar ligament, odontoid process or transverse ligament insufficiency, has been shown to be a factor in the production of vertigo and associated symptoms possibly by occlusion of the vertebral artery or by disturbance of afferent input to the vestibular nuclei[1,2,7,11,12].

Partial or complete tears of the alar ligament, generally, are not an immediate serious danger to the patient's life and a less drastic approach can be taken than when fracture or atlantoaxial rotatory fixation (AARF) is supected. Treatment can be continued but should not be such that it might transform a grade 2 to a grade 3 tear or exacerbate symptoms ascribable to damage to this ligament. The physician should be informed and MRIs[3,8,9] or as second choice CTs[4,5,11] should be ordered or where these are not easily accessed, side flexion and rotation X-rays of the cranio-vertebral joint may demonstrate the instability[4].

Clinical Test

The alar ligament has been found to be tightened by contralateral rotation and contralateral side flexion of the atlanto-axial joint[6]. This biomechanical knowledge is used to test the ligament. Two clinical tests are typically used to determine the sufficiency of this ligament.

Kinetic Test

The test can be carried out in sitting or lying, in this article the lying test will be described but the principles are the same. The patient is supine and the therapist palpates the spinous process of C2 and then side flexes the head around an axis roughly through the nose. There is movement of the spinous process to the side opposite the side flexion. This movement is the result of rotation of the axis putatively due to the tension generated in the contralateral alar ligament. A positive test is one where there is a delay in the onset of movement. The test has pros and cons like most tests. The main pro is that it can be carried out in the acute patient without stress the region. The con is that it is not a stress test and it is unlikely to reproduce symptoms and if the rectus captitus posterior major is hypertonic or fibrotic a false negative may result as this muscle runs between the same two bones in the same plane and direction.

Stress Test

This test may also be carried out in sitting or lying and again the lying position will be described. The patient is supine and the therapist stabilizes the spinous process of C2 by bringing the pad of one thumb against the side of the spinous process ensuring that the soft tissue is fully compressed against the bone. The index finger wraps around the other side of the process to complete the stabilization. Gripping the top of the head the

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therapist side flexes it away from the stabilizing thumb around the axis through the nose. There should be no movement and no symtptoms. A positive test is for instability is one that reproduces the patient's pain and causes movement to occur. A consideration must be made to allow for variations in the orientation of the ligament. The vast majority of these ligaments are orientated laterally or posterolaterally[6,10]. This means that if the head is flexed at the craniovertebral joints the posterior glide of the occiput will tighten up the majority of orientations. If movement is felt then the test should be carried out in the extended position to ensure that it is not a false positive due to the variation.

The patient is laid supine; the second cervical vertebra is fixed by gripping the spinous process and the lamina in a wide pinch grip. The head and atlas is then side flexed around the coronal axis for the atlanto-axial joint. This side flexion results in an attempt by the axis to ipsilaterally rotate. Rotation is prevented by the fixation of the axis if the alar ligament in intact. If there is any laxity in the ligament some rotation will result until the laxity is taken up. This motion is appreciated by the examiner and considered as an indicator of instability. In addition to an excessive motion occurring, symptoms ascribable to disorders of the balance mechanism are noted.

Treatment

Treatment consists of the usual stability therapy but the mainstay is a hard collar worn for at least 8 weeks by the patient. The patient should be informed that recovery will take a protracted period and this may be important for medico-legal reasons.

Case History

A 28-year-old woman who was driving the car ahead in rear end collision ten years early suffered immediate severe pain that remained acutely painful for months after the accident. At 18 years of age she had to quit university due to the pain and since then had held many jobs over the following ten years losing each quickly as she had to take time off work due to exacerbations of pain caused by minor triggers.

She had been extensively treated with physical therapy, chiropractic, acunpuncture, nerve root ablation therapy, multiple anesthetic injections into various zygopophyseal joints and pretty much everyting else you could think of. Nothing gave her more than very temporary improvement.

I saw her as a consult and she complained of continuous right suboccipital, occipital pain that was easily exacerbated to intense levels and was accompanied by Type 2 (pre-syncope) dizziness that took sometimes weeks to settle down. At the time I saw her she was between episodes of severe pain but she still complained mild to moderate occipital pain.

On examination she had full range movement with pain on flexion and left rotation. Isometric contractions of the cervical muscles increased her pain. There was no neurological symptoms or deficit. On biomechanical segmental testing there was a hypomobility of the right uncovertebral joint into inferomedial gliding. This was not felt to significant to her pain and dysfunction due to the minor nature of its character. The alar ligament kinetic test was negative and permission was asked to stress test it with the patient being informed that a severe flare up was likely afterwards. Permission granted the ligaments were stress tested and the test for the right ligament demonstrated obvious instability and reproduced her pain exactly.

The patient was asked to call the next day to let me know how her symptoms were. They were considerably worse and she felt this was as a bad an exacerbation has she had recently suffered and that it would likely take two to three weeks to settle down. She was seen one month later and put into a hard Philaladelphia) collar. She

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felt immediate relief of the background pain. She was instructed to wear it at all times including at night. She called two days later and said that essentially she was pain free. She was told to wear it for 8 weeks and see me then and at that time the collar was removed. The pain recurred immediately.

Surgery is unlikely to be performed because there is no threat to her life or neurological status. CT and/or MRI was suggested to her physician as investigative procedures but were not carried out during the time I had contact with the patient. As minor isometric contractions were painful stabilization therapy was not an option although it was tried unsuccessfully as was manipulation of the hypomobile segment with the same result. The patient was told to wear the collar as she chose but that physical therapy had little to offer. It was not even an option to refer her for different treatment as she had tried pretty much everything.

1. Albertus S. Cervical vertebral problems as a cause of variations in the nystagmographic R-factor. Acta Otolaryngol 1984;97:27-32.
2. Bogduk N. Cervical Causes of Headache and DIzziness. In Boyling J, Palastanga N eds. Grieve's Modern Manual Therapy. 2 ed. Edinburgh: Churchill Livingston, 1994:317-31.
3. Briem D, Linhart W, Dickmann C, et al. [Injuries of the alar ligaments in children and adolescents]. Unfallchirurg 2002;105:555-9.
4. Dvorak J. [Functional roentgen diagnosis of the upper cervical spine]. Orthopade 1991;20:121-6.
5. Dvorak J, Panjabi M, Gerber M, et al. CT-functional diagnostics of the rotatory instability of upper cervical spine. 1. An experimental study on cadavers. Spine 1987;12:197-205.
6. Dvorak J, Panjabi MM. Functional anatomy of the alar ligaments. Spine 1987;12:183-9.
7. Galm R, Rittmeister M, Schmitt E. Vertigo in patients with cervical spine dysfunction. Eur Spine J 1998;7:55-8.
8. Krakenes J, Kaale BR, Moen G, et al. MRI assessment of the alar ligaments in the late stage of whiplash injury--a study of structural abnormalities and observer agreement. Neuroradiology 2002;44:617-24.
9. Muhle C, Brossmann J, Biederer J, et al. [Alar ligaments: radiological aspects in the diagnosis of patients with whiplash injuries]. Rofo 2002;174:416-22.
10. Panjabi MM, Oxland TR, Parks EH. Quantitative anatomy of cervical spine ligaments. Part I. Upper cervical spine. J Spinal Disord 1991;4:270-6.
11. Urso S, Pacciani E, Ascani E, et al. [Static-dynamic computerized tomography in the diagnosis of traumatic lesions of alar ligaments. Preliminary results]. Radiol Med (Torino) 1994;88:736-41.
12. Wrisley DM, Sparto PJ, Whitney SL, et al. Cervicogenic dizziness: a review of diagnosis and treatment. J Orthop Sports Phys Ther 2000;30:755-66.

Technique Tips

Kinetic Testing of the Alar Ligament

If you test the ligament with the patient supine do not use the hands to side flex the head as this results in the fingers moving on the spinous process and may confuse your kinesthesia. It is actually easier to do the test by moving your hips from side to side while the patient's head is against you stomach and at the same time doing it this way maintains the relationship between your fingers and the spinous process of C2.

Quizzes for Fun. Solutions

Septembers Word Jumble Solution

M	F	S	I	S	H	U	N	S	S
O	E	S	O	P	H	A	G	U	S
S	S	E	I	P	V	W	G	R	O
O	F	D	I	I	T	V	L	T	U
B	U	R	N	S	C	O	I	O	N
E	L	I	I	I	C	R	P	T	D
R	T	C	O	F	F	U	S	H	B
B	R	O	N	O	S	E	S	I	Y
E	A	C	A	R	P	A	L	G	W
L	F	T	A	M	C	A	D	H	Y

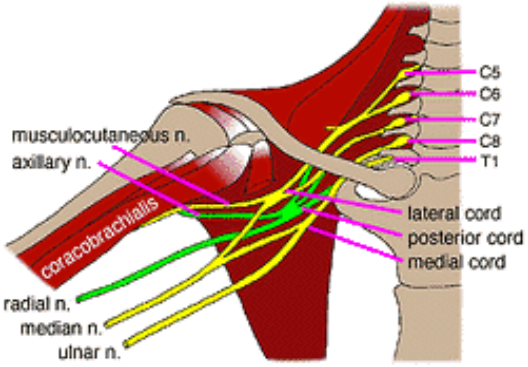
Answer the following:

1. The vertebral artery is in four parts names from inferior to superior as:

A. Osteal, transverse, intracranial, sub-occiptial
B. Transverse, sub-occipital, osteal, intracranial
C. Osteal, transverse, sub-occipital, intracranial
D. Primus, secondus, tertius, quartus

2. From medial to lateral the brachial plexus is divided into:

A. Roots, spinal nerve, division, trunk, cord, branches
B. Spinal nerve, roots, trunk, division, cord, branches
C. Roots, spinal nerve, cord, trunk, division, branches
D. Roots, spinal nerve, trunk, cord, division, branches



http://depts.washington.edu/anesth/regional/brachialplexusanatomy.html

Which of the following definitions for S1 nerve root pain is best::

A. Any pain that runs down the back of the leg in the S1 dermatome
B. Sciatica
C. Lancinating pain that runs down the back of the leg in the S1 dermatome
D. Aching in the back of the leg in the S1 dermatome that is made worse by lumbar movements and sitting

Which of the following statements is correct:

A. Lancinating (zinging or electrical) pain is never caused by non-neurological sources
B. Aching pain can result from nerve root irritation if it is in the appropriate dermatome
C. Compression of the nerve root without damage or severe inflammation may cause pain
D. The nerve root may cause aching pain if it is damaged.

While there are nociceptors in the covering of the nerves they do not seem to be sensitive to compression or traction (clinical and experiemental studies have shown this) and when these forces are applied parasthesia and deficit occurs but not pain. To be painful they must be damaged or possibly seriously inflamed. Then the pain is typical neuropathic and brought on by irritation of the nerve tissue. Neuropathic pain is either lancinating (zinging, electrical flashes) or causalagic (burning, itching pain). The exception is the dorsal root gangion which can produce neuropathic pain by simple mechanical deformation and the CNS particularly the spinothalamic tract.

Clinical Anatomy of the Lumbar Spine and Sacrum, Bogduk, N. Churchill Livingston, 1997

Quizzes For Fun

Word Jumble

A	X	I	S	T	R	A	X	B	E
B	R	R	C	A	W	F	L	E	X
D	O	I	A	Y	S	O	I	F	T
U	Y	S	N	P	P	E	P	R	E
C	S	E	N	S	A	T	I	O	N
E	S	T	I	T	S	A	D	A	D
N	P	E	N	A	M	L	L	B	Y
S	A	R	G	B	E	E	E	E	A
T	I	R	F	L	S	T	A	N	D
E	N	Y	G	E	N	U	G	D	E

A. Answer the following about research:

1. What is the difference between a theory and a hypothesis
2. True or false; validity is can only established by experiment
3. What is the difference between sensitivity and specificity
4. What are descriptive statistics

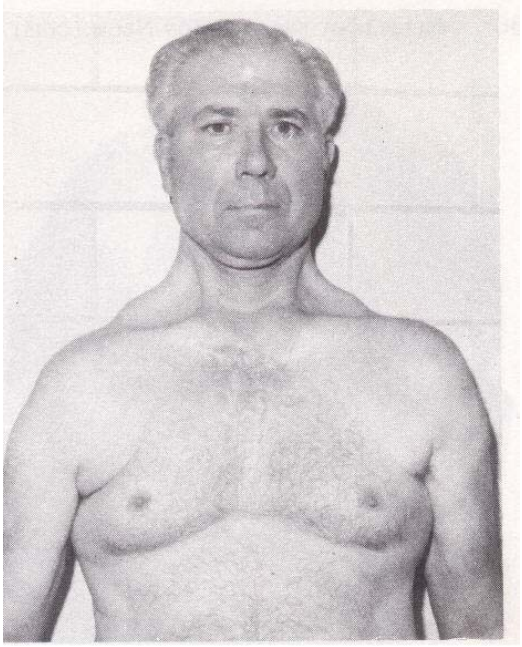
B. Answer the following about anatomy:

1. What are Muller's muscles
2. Where are the utricle and sacule and what are their functions
3. With which artery does the posterior communicating artery of the brain communicate
4. What is the function of the Edinger-Westphal nucleus

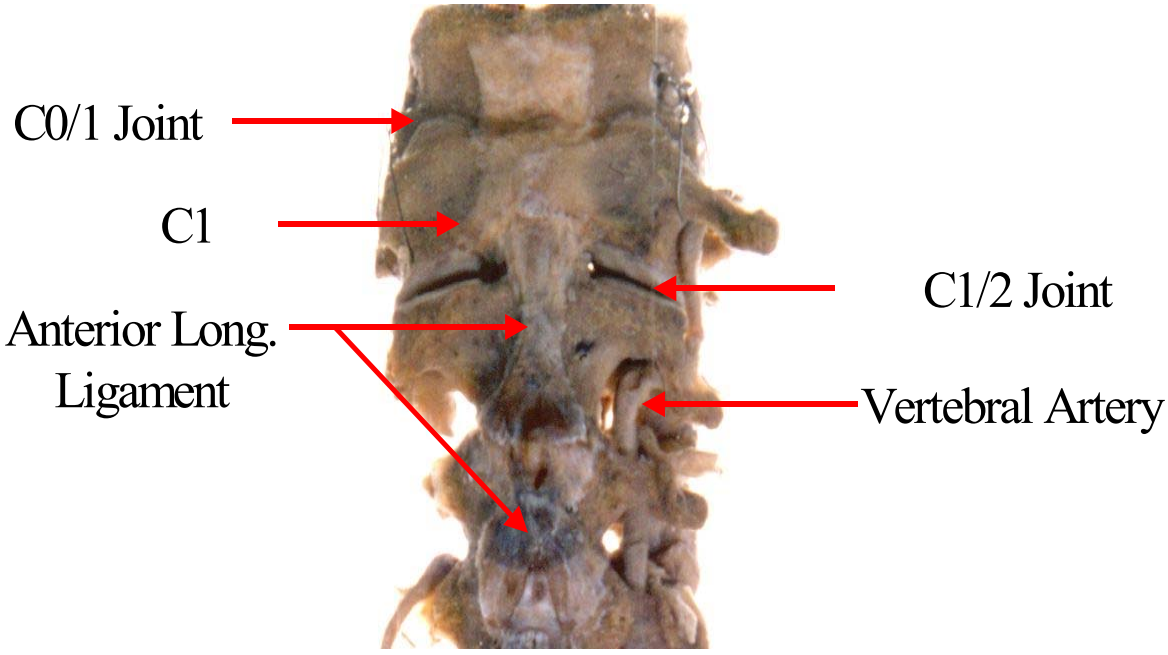
C. Answer the following about pathology:

1. With what condition is paralysis of Muller's muscles commonly associate
2. What is type 1, 2 and 3 dizziness
3. What is the name of the distribution system that shunts blood between the hind and fore brain
4. Define miosis

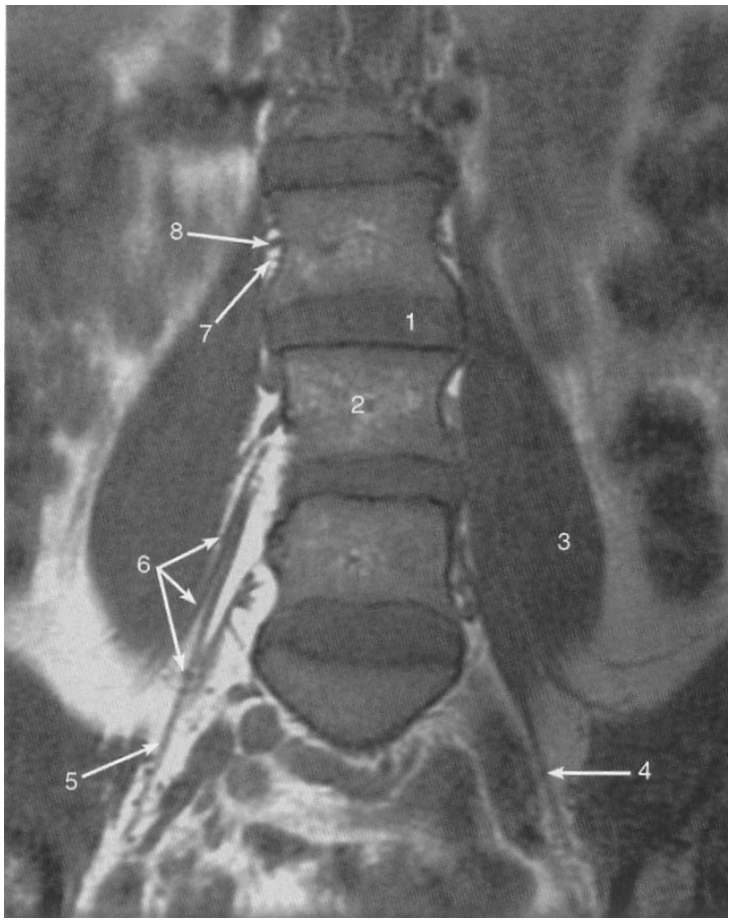
D. What is wrong with this man?



Name That Structure
September's Answer



Name That Structure



Don't curse the darkness, light a candle.

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Video Tapes Digital Conversion Update

The video tapes series Manual Therapy by Jim Meadows is being converted to DVD format and should be ready for sale in a month or so. There are 50% discounts available for past buyers of the video tapes and for past students of my courses and 30% discounts for subscribers to this newsletter and to NAIOMT students. The estimated full price of the complete set of DVDs is $700 Canadian or US depending in which country you reside.

For further information on the content of the video see my web site.