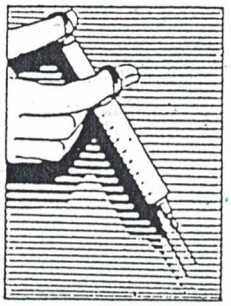

Use of Sclerosing Solution May Change Therapy in Vertebral Disk Problem

. . . by Earl H. Gedney, D.O.

TWO years of the use of sclerosing solution in stabilizing vertebral ligaments have convinced the writer of the probability that this is the therapy of choice in intervertebral disk degeneration: uncompensated. Until this therapy was promulgated and promoted, the recommended treatments for this condition had been exercises to improve the surrounding musculature, various and sundry manipulations, external supporting mechanisms and, in addition, resection of the disks with or without fusion. Heel lift therapy, and other therapy for leg length equalization has been brought into play and with a certain degree of success in this condition. These therapies have been shown quite efficacious in the sedentary and less rigorous occupations but leave something to be desired in the backs of laborers.

One of the most active, rigorous and laborious of occupations is that of woodsman. This therapy has been developed among such workers and is proving its efficacy. There have been no harmful sequelae. All cases, without exception, are enjoying some improvement and most patients are returning to their original occupation. Woodsmen are enjoying recognition of the Workmen's Compensation Commission, The Welfare Department of the State of Maine, and a number of insurance companies in our state as well. A number of physicians within accessible range are unhesitatingly referring patients for treatment.

We have all seen the patient who has been through examination for chronic backache, has been thoroughly "cliniced" and examined even with the aid of the myelogram, and who has been told by the examiners that, although he has a disk syndrome, surgery is not advised as yet. He still has his disability and must be wrapped up in some kind of support to fortify him against the daily rigors of life until such times as he must become much more acutely disabled —enough to justify grave operative procedures which still guarantee little more than a 70% satisfactory result to the patient. With present therapy, sclerosing in nature, it is hoped to take this patient out of the realm of surgical therapy. Results in these several cases seem to indicate that the therapy may even include the ruptured disk.

All diagnostic aids we can summon to identify developing lesions must be brought to bear. The chief complaint in the lumbar disk lesion

is helpful in that certain pain patterns are quite reliable, yet it must be kept constantly in mind that neurological variations do occur. Pain down the backs of the thigh and calf to the ankle, I have found, is significantly of L 4 origin although only borne out by absent reflex response at the medial hamstring tendon in 33% in this present group studied. However, it is corroborated with tenderness at L 4 spine in 77%, and with hypermobility in 83%. X-ray narrowing only proved the pain pattern in 61%—retrolisthesis as an X-ray positive sign has more recently come into the picture.

Fourth lumbar

Many authors feel L 5 disk is the most frequently involved one. This may be entirely true but from my studies it appears that L 4 is at least the most symptomatic one, whatever the reason. 95% in this series herewith reported had symptoms referable to L 4. 50% of them responded favorably to treatment directed to this segment alone, where it was felt that only this one disk was at fault. An additional 33% responded similarly when another disk was included in the treatment. The balance were felt to be either muscular syndromes or to be transposed neurologically, the prefixed or postfixed plexuses, and were treated at other segments than the fourth, although with good results. Test of treatment is thus seen to verify the constancy of the pain pattern.

Pain pattern behavior will vary also with pathology present and all the pathologies must be kept constantly before the examiner. Sprain of the psoas will compromise any of the nerves running through it. Facet insult with synovitis will produce pressure on the nerve trunk beneath it. Likewise inflammatory conditions causing swelling of the posterio spinal ligament may make pressure on the whole cauda causing symptoms bordering on paresis. The piriformis muscle and its relation to the sciatic nerve must also be remembered lest it be making pressure symptoms through traction, although the cause of this latter syndrome is often placed more centrally.

Should the pain involve the back of the thigh and leg and also continue on down into the foot or toes it can be felt to be more closely related to the fifth lumbar segment. Third lumbar segment pain is quite reliably portrayed by the patient when he says it is in his hips and he points to the area directly over the point of emission of the superior gluteal nerve. L 3 may also extend into the front of the knee. Adductor pain is a good indicator of trouble in the vicinity of L 2. Groin pain may be referred from L 1 but also may be coming from the psoas tendon.

In acute severe segmental sprains none of the ordinary signs can be taken too specifically to damn any given segment. The reaction is so severe and protective splinting is so broadcast that multiple spinal areas may be thought to be involved. In this situation reflex arcs are most helpful and much more constant than in the more chronic conditions. Their usefulness is exemplified in the case of one Mr. E., woodsman, who had been treated successfully at L 4 and 5 only to sustain a severe lumbar sprain later. He presented himself lamenting that he was

"right back where he started." However, he was shown from the case records that while on prior occasion his Achilles (L 5) and medial hamstring (L 4) responses were absent, they were now present, but the patellars (L 3) were now absent indicating perhaps that enough degeneration had taken place at L 3 to allow for loss of stability. Hence the sprain. Apparently this was the case since it responded to sclerosing treatment with alacrity.

It may become highly desirous to learn exact data on given segments with reference to their hypermobility, especially during an acute syndrome which does not abate sufficiently under conservative treatment for accurate analysis. General anesthesia should be employed without hesitation since it is in the area of hypermobility that the most effective gains are made in the control of these conditions, acute or chronic.

Calcified ligaments

This hypermobility is due to lack of stability in the supportive elements surrounding the vertebrae. Should the contained intervertebral disk material on one or either side of a vertebra degenerate from whatever cause, its annulus and nucleus will lose substance. As it loses substance it follows, as the day the night, that it will soon lose space. This loss of space approximates the superimposed vertebrae and unless the annular ligaments surrounding the disk also lose length, we have a potentially lax supportive element. Proof that this shortening and thickening does occur is shown by X-ray when these ligaments are seen to calcify extensively, even to the extent of bridging the entire gap across the disk. Ligaments so calcified efficiently limit motion.

Corresponding annular ligaments which retain their original length during disk degeneration can offer little assistance to the joint involved. Under direct vision during exposure in disk surgery these can be seen to wrinkle when the vertebra is mobilized via the spinous process as described by Dandy. This is the only constant sign I have been able to elicit. It would seem to indicate a structure with diminished underlying substance, else it would not wrinkle nor if it were attached to a disk in the norm, could it wrinkle.

In some intervertebral joints, however, we see the space decrease gradually over the years and multiple joints become involved without serious symptoms. Greater and greater stability frequently accompanies these changes and no hypermobility nor tenderness can be elicited over the involved segments. These joints have compensated their loss of space and decreased their ligament length by whatever process. They are regarded as compensated. They cause little pain or disability.

When this does not occur and structures other than ligaments must supply the stabilizing functions of the vertebral segment, it has failed to compensate for the process undergone. It has failed to repair its damaged elements or to build compensating ones and eventually when put to test, will fail the organism it is supporting. These joints receive the just label: uncompensated. They are symptomatic.

(Continued on Page 34)

(*Continued from Page* 13)

Inordinate muscular fatigue may become increasingly more evident as decompensation progresses. This is frequently brought out in the complaint syndrome. The patient may be able to walk only short distances or stand for short intervals before the pain of the chief complaint or this inordinate fatigue sets in. The fatigue may have its explanation in the increased tonic activity of the psoas and its antagonists, the sacro spinalis group, necessary to keep the superimposed vertebrae atop their slippery perch. The pain recurrence on standing may also be due to this same mechanism or it may also be due to a posterior spinal ligament bulging into the spinal canal. Inordinate fatigue may be easily overlooked by the patient in relating this complaint, but as the treatment begins to mend the broken compensation, he notices an immediate and steady return of his physical prowess. He will be the first to mention that he feels better than he has for years.

Some of these people have no surcease in slumber. They must wear their supporting belts and braces even at night in spite of hard beds or bed boards. One patient recently had his first full night's sleep in eight years. He has involvement of all the lumbar disks and the twelfth dorsal, shown at X-ray. He is progresing very nicely but is not included in the group being reported.

In this series of nineteen cases who have finished treatment, it is possible that one case may need more treatment. The others have finished and all have returned to their normal occupations and are able to do a normal allotment of work. 33% have been so certified, when necessary, to agencies and insurance companies.

Selection of the segment or segments toward which to divert our attention will depend on the sum total information gleaned from the examination. The chief complaint will almost always incriminate one of three segments as recorded above. Frequently it is necessary to treat all three and they may all be involved: the segment of chief complaint and those on either side as L 4 in relation to L 5 and L 3. Hypermobility has been shown the most constant finding in relation to the chief complaint followed by tenderness. Constant findings being obviously the most reliable, it is safe to select the area to be treated on this basis. Never discount the chief complaint syndrome as related above. The chief complaint is the constant factor and the operator will do well to remember this. For example, if the patient complains in the areas sup-

plied roughly by L 3, then his trouble is at L 3 with the known variations.

A good illustration of this is Mr. B., age 42, with chief complaint of pain in L 4 and L 3 zones. L 3, 4 and 5 were tender and hypermobile —left Achilles reflex absent. It was thought best to treat L 4 and 5 first and despite ten weeks of treatment to these segments there was no relief at L 3 until treatment was begun there. This had its use experimentally but almost lost the case for us.

Technic

How to treat these ailing segments presents several problems also. The actual technic has become quite stereotyped since it is only necessary to put the Sylnasol into the annular ligaments surrounding the disk space. This has been described on previous occasion but to repeat: a point is taken opposite the center of the spinous process of the vertebrae above and 1.5 inches away from it. A 3.5 inch, number 19 needle attached to a 2 c.c. syringe is carried down through the tissues, dropping a total of 2 c.c. of anesthetic as far as the space between the transverse processes. Any anesthetic below this point may contact the corresponding nerve and produce some loss of control for varying periods. This is embarrassing but not for long, since such small amounts are used that it is readily absorbed by the body in a few minutes. This is followed immediately with a short bevel needle of same size and length carrying the Sylnasol. The direction is downward, about 10 degrees medial and 15 degrees caudad.

Considerable experimental trial work has been done to ascertain the correct amount of Sylnasol to use. Cautiously we started with 0.25 c.c. and progressed to as much as 2.0 c.c. bilaterally into the capsule on other cases. As the dosage was increased there was, on occasion, much greater reaction and more attendant disability than desirable in a therapy advertised as ambulant. Eventually it was found that 0.25 c.c., 5 minums, of Sylnasol properly placed into the ligament produced satisfactorily rapid results. In this dosage should any spill occur into the joint, between the slips of the psoas or alongside the corresponding nerve as it travels over the disk space, the quantity will be so minute as to cause minimal irritation.

As well as abatement of symptoms and improvement of functions, your test of progress during treatment is the decreasing tenderness and limitation of hypermobility of the vertebra above. Should this not proceed apace despite ample thickness of the lateral parts of the capsule as palpated with the needle, then it is possible that other parts of the capsule may need treatment. The more posterior elements of the capsule may be reached by turning the needle a little more medially and travel medial to the nerve trunks instead of laterally as in the approach to the lateral ligaments.

The applied neuroanatomy varies at each of the segments as the emanating nerves add to the lumbar and sacral plexuses. For example, at L 4 we find L 4, 3 and 2 from within out. As the blunt point needle approaches this field of

nerves the patient advises the operator by prearranged signal that he feels an electric-like stimulus in the lower extremity. He is asked where he feels it and from the information supplied, the operator knows approximately where his needle point is and where to go from there.

The lead edge of the blunt point is kept medial as the needle approaches the capsule so that it might better insinuate itself between its fibers. Otherwise, especially after several applications of the sclerosing solution have caused it to become tougher, it sheds the needle very readily. The needle is kept blunt-ended but sharp. It is embarrassing to get the needle in contact and not to be able to pierce the capsule.

The vertebral bodies may be abnormally rotated and alter the relations sufficiently to make the technic of injection very difficult. Derotation is helped considerably by turning the head in the pathway of desired motion when the patient is prone over the table break waiting for treatment.

The number of treatments to be given may depend on the reaction of the patient who may desire to discontinue them with alleviation of symptoms. Ideally, however, it should depend on the return to normal mobility in the segment treated. The function of the sclerosing solutions is to cause localized inflammation which on healing causes scar tissue with its thickening and contracture. This should be continued to the point of stability. Nothing is to be gained by over treatment. In this group with varied dosages when treating a single disk at L 4, from three to ten treatments were necessary with an average of 6.4. A single lesion at L 3 took nine treatments. One L 5 took four and another eight. In paired lesions at L 4 and L 5, L 4 averaged seven and L 5 two treatments. In this connection it must be remembered that the spinal ligaments are actually continuous from the occiput down to the coccyx and the reaction may overflow from one segment to another, necessitating less treatment in adjoining disk ligaments. This ligament continuity is brilliantly demonstrated by the Halliday Spine being prepared at P.C.O. by Dr. Angus G. Cathie.

To summarize, an attempt has been made to show the following:

Symptoms occur in disk degeneration without demonstrable herniation due to failure of ligaments to shorten and stabilize the joint.

It has been previously shown that ligaments may be thickened and shortened with sclerosing solution.

A practical method for permeating the vertebral ligaments with sclerosing solution has been presented.

This method has been used on the living subject with encouraging clinical results. Bangor, Me.

REFERENCES

BARR, JOSEPH S.—*Journal Bone and Joint Surgery*, July 1951.

"Retrolisthesis," *Surgery, Gynecology & Obstetrics*, August, 1950.

"Vertebral Retroposition," *International Abstracts of Surgery*, September 1951.

Piersol "Human Anatomy," 9th Ed. Revised.

GEDNEY, EARL H.— The Osteopathic Profession, September, 1951.

EYELESHYMER and SHOEMAKER, "A Cross Section Anatomy," Appleton-Century-Crofts, Inc.

(This paper was read before the Eastern Osteopathic Assn. Convention, April 5, 1952)