Abstract: The intervertebral disc is avascular. With aging, endplates become occluded by calcified layers. Diffusion of nutrients and oxygen into the disc diminishes. The disc degenerates, and pain ensues. Traction, hyperbaric chamber, nutritional supplements, vasodilator and/or ultrasound are proposed to enhance uptake of fluid, nutrients and oxygen to replenish sulfated glycosaminoglycans, restore swelling pressure, sustain compressive loads and alleviate back pain.
TREATING THE CAUSE OF BACK PAIN
WITH TRACTION WITHIN HYPERBARIC CHAMBER

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FIELD OF INVENTION

This invention relates to methods and devices to increase uptake of nutrients and oxygen into the intervertebral disc using traction, oxygen, a hyperbaric chamber, nutritional supplements and/or vasodilators to enhance biosynthesis of sulfated glycosaminoglycans and restore swelling pressure within the intervertebral disc.

BACKGROUND

Although the sources of low back pain are varied, in many cases the intervertebral disc is thought to play a central role. Degeneration of the disc initiates pain in other tissues by altering spinal mechanics and producing stress in surrounding tissues.

Intervertebral discs 100 absorb most of the compressive load of the spine, with the facet joints 129 sharing approximately 16% of the load, as depicted in Figure 1. The disc 100 consists of three distinct parts: the nucleus pulposus 128, the annular layers 316 and the cartilaginous endplates 105, as shown in Figures 1 and 2. The disc 100 maintains its structural properties largely through its ability to attract and retain water. A normal disc 100 contains 80% water. The nucleus pulposus 128 is particularly rich in water absorbing sulfated glycosaminoglycans, which create the swelling pressure to provide tensile stress within the collagen fibers of the annulus 316. The swelling pressure produced by high water content is crucial to support the annular layers 316 and sustain compressive loads.

In adults, the intervertebral disc 100 is avascular. Water, nutrients and oxygen 317 are transported from external blood vessels through endplate flow 326 and annular flow 325, as shown in Figure 3. Survival of the disc cells, as well as production of chondroitin and keratan sulfates, depend on endplate flow 326 and annular flow 325. However, annular flow 325 penetrates only up to 1 cm into the annular layers of the disc 100. An adult disc can be as large as 5 cm in diameter; hence endplate flows 326 through both the cranial and caudal endplates 105 are crucial for maintaining the health of the nucleus pulposus 128 and inner annular layers 316 of the disc 100.
Water gain and loss depend on the disc pressure. During the day, compressive loads cause the disc to lose 8-12% of the disc fluid to bodily circulation. In supine, compressive loads on the disc 100 are lifted, so the disc pressure is low. The water containing nutrients and oxygen 317 is driven inward from both endplate flow 326 and annular flow 325 by diffusion, convection and the water absorbing capability of sulfated glycosaminoglycans within the disc 100, as depicted in Figure 4. The water absorbing capability of sulfated glycosaminoglycans contributes to osmotic pressure in the disc 100. Disc 100 pressure increases from about 0.1 MPa at the beginning of sleep to about 0.24 MPa after eight hours of sleep. The water retained by the chondroitin and keratan sulfates creates the swelling pressure that provides tensile stress within the collagen fibers of the annulus 316, as shown in Figure 5. Chondroitin sulfate is the primary, and keratan sulfate the secondary, water absorbing sulfated glycosaminoglycans within the intervertebral disc, as shown in Figure 29, crucial for maintaining swelling pressure to support the annular layers 316 and sustain compressive loads.

Calcium pyrophosphate and hydroxyapatite are commonly found in the endplate 105 and nucleus pulposus 128. Calcified layers 108 begin to accumulate in the cartilaginous endplate 105 as early as 18 years of age, as shown in Figure 6 (Oda J, Tanaka H, Tsuzuki N: Intervertebral disc changes with aging of human cervical vertebra from the neonate to the eighties, Spine, 13(11), 1205-1211, 1988). The blood vessels and capillaries at the bone-cartilage interface are gradually occluded by the build-up of the calcified layers 108, which form into bone (Bernick S, Cailliet R: Vertebral endplate changes with aging of human vertebrae, Spine, 7(2) 97-102, 1982). Bone formation at the endplate 105 increases with age, restricting diffusion of nutrients 317, including sulfate, proline and oxygen, from entering into the nucleus pulposus 128. With only limited outer annular flow 325, the majority of the cells within the disc 100 are deprived of nutrients and oxygen (Urban JP, Smith S, Fairbank JC: Nutrition of the intervertebral disc, Spine, Dec 1;29(23):2700-9, 2004). As a result, production of the water absorbing sulfated glycosaminoglycans is significantly reduced, and the water content within the nucleus pulposus 128 decreases (Kitano T, Zerwekh J, Usui Y, Edwards M, Flicker P, Mooney V: Biochemical changes associated with the symptomatic human intervertebral disc, Clinical Orthopaedics and Related Research, 293, 372-377, 1993). During normal daily compressive loading on the spine, the reduced pressure within the
nucleus pulposus 128 can no longer distribute forces evenly against the circumference of the inner annulus 316 to keep the lamellae bulging outward. As a result, the inner lamellae sag inward while the outer annulus 316 continues to bulge outward, causing delamination 114 of the annular layers 316 and degeneration of the disc 100, as shown in Figure 7.

The nucleus pulposus 128 is thought to function as “air in a tire” to pressurize the disc 100. Normal swelling pressure effectively distributes the forces evenly along the circumference of the inner annulus 316 and keeps the lamellae bulging outward. With lack of swelling pressure, the degenerated disc 100 exhibits unstable movement, similar to a flat tire, as depicted in Figure 8. Approximately 20-30% of low-back-pain patients have been diagnosed as having spinal instability, as shown in Figure 9 (Weiler PJ, King GJ, Gertzbein SD: Analysis of sagittal plane instability of the lumbar spine, Spine, 15:1300-1306, 1990). The pain may originate from increased load and stress on the facet joints 129 and/or surrounding ligaments (Kirkaldy-Willis WH, Farfan HF: Instability of the lumbar spine, Clin Orthop, 165:110-123, 1982).

Shear stresses of the disc 100 are highest at the posteriolateral portions adjacent to the neuroforamen. With decreased swelling pressure, shear stresses cause the delamination 114, tearing and bulging of the annulus 316, as depicted in Figure 10. The nerve 194 is confined within the neuroforamen 121 between the disc 100 and the facet joint, vulnerable to impingement 329 by the bulging disc 100, as shown in Figure 11. Hence, chondroitin and keratan sulfates are crucial to retaining water for the swelling pressure to sustain compressive loads and maintain disc height.

The disc 100 is expandable or inflatable in the absence of compressive loads. A majority of astronauts suffer constant back pain in space, but not from instability or impingement. Their discs expand or thicken significantly, elongating the spine, the spinal cord, facet joint capsules, anterior and posterior longitudinal ligaments. The heights of astronauts increase between 4-7 cm in space (Hutchinson KJ, Watenpaugh DE, Murthy G, Convertino VA, Hargens AR: Back pain during 6 degree head-down tilt approximates that during actual micro gravity, Aviat Space Environ Med, Mar; 66(3):256-9, 1995). In the absence of gravity, the daily cycle of disc compression during the day and recovery during the night is missing. Water continues to accumulate beyond the normal recovered volume, leading to disc thickening. Micro-gravity would be an ideal treatment for spinal instability or spinal stenosis.
Unfortunately, the excess water within the discs is eliminated within days or weeks after the compressive loads resume. Spine lengthening is also observed when placing a person on a 6 degree head-down tilt bed.

Similarly, oxygen through endplate flow 326 is also significantly restricted by the calcified layer 108, and oxygen penetration from annular flow 325 is shallow. When oxygen partial pressure in the disc falls below 0.25 kPa, anaerobic production of lactic acid dramatically increases with increasing distance from the endplate. The pH within the disc falls as lactic acid concentration increases. Lactic acid diffuses through micro-tears of the annulus and irritates the richly innervated posterior longitudinal ligament, facet joint and/or nerve root. Studies indicate that lumbar pain correlates well with high lactate levels and low pH (Diamant, B, Karlsson, J, Nachemson, A: Correlation between lactate levels and pH of patients with lumbar rizopathies, Excerpta Medica, 24, 1195-1196, 1968). The mean pH of 23 symptomatic discs is significantly lower than the mean pH of normal discs (6.65±0.07 versus 7.14±0.04, p<0.0001, respectively). Acid concentration is three times higher in symptomatic discs than normal discs. In symptomatic discs with pH 6.65, the acid concentration within the disc is about 5.6 times the plasma level. In some preoperative symptomatic discs, nerve roots were found to be surrounded by dense fibrous scars and adhesions with remarkably low pH 5.7-6.30 (Nachemson, A: Intradiscal measurements of pH in patients with lumbar rizopathies, Acta Orthop Scand, 40, 23-43, 1969). The acid concentration within the disc was about 50 times the plasma level.

The majority of patients with low back pain cannot be given a precise pathoanatomical diagnosis. This type of pain is generally classified under “non-specific pain”. Back pain and sciatica can be recapitulated by maneuvers that do not affect the nerve root, such as intradiscal saline injection, discography, and compression of the posterior longitudinal ligaments. It is possible that some of the non-specific pain is caused by lactic acid irritation secreted from the disc. Injection into the disc can flush out the lactic acid. Maneuvering and compression can also drive out the irritating acid to produce non-specific pain. Currently, no intervention other than the discectomy can stop the production of lactic acid.

Biosynthesis of chondroitin sulfate requires oxygen. On the other hand, keratan sulfate can be biosynthesized under anaerobic condition. When oxygen concentration is dangerously low within the degenerative disc 100, production of keratan sulfate is favored over
chondroitin sulfate. Within the degenerated disc 100, the ratio between keratan and chondroitin significantly increases. Keratan sulfate has only one negative charge, but chondroitin sulfate has two negative charges in the repeating disaccharides, as shown in Figure 29. Water binding or retaining capacity is enhanced by the negative charges. Hence, even with similar molar equivalence, increased ratio between keratan sulfate and chondroitin sulfate results in lowered water retaining capacity within the disc 100 capped by calcified endplates 105.

In addition, the rate of sulfate uptake into the disc 100 is pH sensitive. The maximum rate of sulfate incorporation occurs at pH 7.2-6.9. Below pH 6.8, the rate falls steeply. At pH 6.3, the sulfate incorporation rate is only around 32-40% of the rate at pH 7.2-6.9. Thus, from the lack of oxygen within the disc 100, high lactic acid concentration can (1) slow down the rate of sulfate uptake to limit the production of sulfated glycosaminoglycans, (2) reduce the swelling pressure within the disc 100, and (3) irritate nerve endings to cause non-specific pain.

In conclusion, calcified endplates initiate a progression of degenerating factors upon the intervertebral disc 100. (A) Sulfate deficiency reduces chondroitin sulfate and keratan sulfate concentration within the disc. (B) Production of the highly water-absorbing chondroitin sulfate is further hindered under anaerobic condition. (C) Water loss from reduction of sulfated glycosaminoglycans lowers the swelling pressure within the nucleus pulposus, leading to inward bulging and delamination of the annulus, added load on facet joints, strain from segmental instability and nerve impingement from disc space narrowing or bulging. (D) Lactic acid production in anaerobic condition irritates nerves, causes pain and further slows down the rate of sulfate uptake into the disc, which leads to progressive disc degeneration and pain.

Since oxygen is vital for proper metabolism within the disc 100, increasing the amount of oxygen into the disc 100 can halt or even reverse disc degeneration to treat back pain. Air consists of approximately 80% nitrogen and 20% oxygen. The atmospheric pressure (ATA) is 1.0 or 14.7 pounds per square inch (PSI), where nitrogen partial pressure = 0.8 and oxygen partial pressure = 0.2. When air pressure increases from 1 ATA to 2 ATA in a hyperbaric chamber, the oxygen partial pressure becomes 0.4 and nitrogen partial pressure becomes 1.6. If 100% oxygen is used under 2 ATA in the hyperbaric chamber, the oxygen partial pressure becomes 2.0. Although the blood pressure measured within the hyperbaric pressure remains
approximately the same, the absolute pressure within the patient’s bodily circulation is approximately equal to the pressure within the hyperbaric chamber plus the blood pressure of the patient.

In addition, Henry’s Law states that when a liquid is exposed to a gas, some of the gas molecules will dissolve into it. The number of gas molecules that dissolve into the liquid depends on the mass of the liquid, the partial pressure and solubility constant of the gas. Henry’s Law can be described as: \( X_i = \frac{P_i}{K_i} \) where \( X_i \) = Mole of dissolved gas at a given temperature, \( P_i \) = Partial Pressure of the gas above the solution, and \( K_i \) = Solubility Constant of the gas. For helium, hydrogen and nitrogen dissolved in water, an essentially linear relation between \( X_i \) and \( P_i \) is observed for pressures up to 300 ATA. For oxygen solubility in water, Henry’s Law is moderately deviated at pressures around 100 ATA. However, solubility of oxygen in blood is enhanced by hemoglobin and myoglobin, which can be saturated. Hence, for oxygen solubility in blood, Henry’s Law is non-linear. However, an increase in oxygen partial pressure will still elevate oxygen concentration in bodily circulation to saturate the avascular disc.

SUMMARY OF INVENTION

Most back pain is caused by degeneration of the intervertebral disc. The disc is avascular. Diffusion of nutrients and oxygen through the endplates is crucial for survival of the cells within the disc. As the endplates become calcified, diffusion of nutrients and oxygen diminishes, leading to the loss of swelling pressure and capacity to sustain compressive loads. In this invention, a hyperbaric chamber is used to increase oxygen partial pressure to enhance diffusion and convective flows of oxygen into the deprived disc. In addition, traction is used within the hyperbaric chamber to lower the disc pressure, favoring and promoting oxygen uptake into the avascular disc. Building blocks of the sulfated glycosaminoglycans are ingested as nutritional supplements to increase concentration for diffusion into the disc. Moreover, vasodilators can also be used to increase blood flow, bringing nutrients and oxygen near the avascular disc to enhance uptake. As a result, biosynthesis of sulfated glycosaminoglycans increases to restore swelling pressure, sustain compressive loads and alleviate back pain.
REFERENCE NUMBER

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<td>287</td>
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<tr>
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<td>Calcified layer</td>
<td>288</td>
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<td>114</td>
<td>Annular delamination</td>
<td>289</td>
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<td>Nerve</td>
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<td>Neuroforamen</td>
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<td>Spinal cord</td>
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<td>Lower-half bed</td>
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DESCRIPTION OF THE DRAWINGS

Figure 1 shows a vertebral segment with an intervertebral disc 100 and facet joint 129 sharing the compressive load between vertebral bodies 159.

Figure 2 shows a healthy and well-hydrated disc 100 containing nucleus pulposus 128 and annulus 316. The nerve root 194 extends from the neuroforamen 121.

Figure 3 depicts diffusion of nutrients and oxygen 317 through endplate flows 326 and annular flows 325 into the avascular intervertebral disc 100.
Figure 4 depicts convective flows of nutrients and oxygen 317 into a relaxed disc 100 in the supine position.

Figure 5 depicts swelling pressure within the nucleus pulposus 128 providing tensile stress within the collagen fibers of the annulus 316 for sustaining compressive loads and stability.

Figure 6 depicts formation of calcified layers 108 over endplates 105, restricting endplate flow 326 of nutrients and oxygen 317 into the avascular disc 100.

Figure 7 depicts decreased glycosaminoglycans and swelling pressure within the disc 100, applying inadequate tensile stress to the annulus 316 and resulting in inward annular bulging and delamination 114.

Figure 8 depicts unstable movement of the vertebral segment caused by inadequate swelling pressure within the degenerated disc 100.

Figure 9 depicts segmental instability and stress on the facet joint 129.

Figure 10 depicts annular 316 rupture and bulging of a degenerated disc 100.

Figure 11 shows annular delamination 114, disc 100 bulging and nerve 194 impingement 329.

Figure 12 shows a patient in the supine position to lower disc pressure within a hyperbaric chamber 318.

Figure 13 depicts increased partial pressure of oxygen 317 within the disc 100 through endplate flow 326 and annular flow 325, resulting from increased partial pressure within the hyperbaric chamber 318.

Figure 14 shows a patient undergoing traction within the hyperbaric chamber 318.

Figure 15 shows a patient undergoing traction using a head-tilt traction bed 320.

Figure 16 depicts disc volume increase by traction to lower the disc pressure. Added pressure from the hyperbaric chamber intensifies the endplate flow 326 and annular flow 325 into the disc 100.

Figure 17 depicts increased swelling pressure within the disc 100 from newly produced glycosaminoglycans made with the replenished supply of nutrients and oxygen 317.

Figure 18 shows increased swelling pressure pushing the annulus 316 outward to support compressive loads on the disc 100.

Figure 19 shows traction to increase the height of a bulging disc 100 with weakened annular layers 316.
Figure 20 shows intensified annular convective flow 325 generated by the hyperbaric chamber 318 and traction causing the weakened annular 316 to bulge inward.

Figure 21 shows trapping of the outer annulus 316 in the inward bulging position after releasing the traction while maintaining pressure within the hyperbaric chamber.

Figure 22 shows nerve root 194 impingement 329 by bone spurs 328 and spinal stenosis.

Figure 23 depicts enhancement of convective flow 325 and endplate flow of nutrients and oxygen 317 using traction and a hyperbaric chamber to enhance biosynthesis of glycosaminoglycans and swelling pressure.

Figure 24 shows a patient with a head harness 321, upper body holder 285 and lower body holder 286 within the hyperbaric chamber 318.

Figure 25 shows a patient with head harness 321 on a head-tilt traction bed 320 for treating cervical, thoracic and lumbar discs.

Figure 26 depicts nutrients and oxygen 317 flow through an annular conduit 126 to nourish the disc cells under traction and/or hyperbaric pressure.

Figure 27 depicts nutrients and oxygen 317 flow through an inferior endplate conduit 126 to nourish the disc cells under traction and/or hyperbaric pressure.

Figure 28 depicts nutrients and oxygen 317 flow through a superior endplate conduit 126 to nourish the disc cells under traction and/or hyperbaric pressure.

Figure 29 shows molecular structures of repeating disaccharides in chondroitin sulfate and keratan sulfate.

Figure 30 depicts the structural arrangement of proteoglycans containing a hyaluronic acid as backbone, link protein and core protein linked to multiple chondroitin and keratan sulfates.

Figure 31 shows the molecular structure of repeating disaccharides in hyaluronic acid.

Figure 32 shows possible linkages of chondroitin sulfate and keratan sulfate to protein/collagen and hyaluronic acid.

Figure 33 shows a patient on a traction bed 320 within a hyperbaric chamber 318, partially submerged in a solution 330 and strapped with ultrasound transducers 331.

**DETAILED DESCRIPTION**

Nutrient and oxygen deficiency is generally accepted as a major cause of disc degeneration. As calcified layers 108 form over the endplates 105, diffusion of nutrients and oxygen diminishes. When external oxygen partial pressure increases, the oxygen partial
pressure within bodily circulation will also increase to supply the oxygen-deprived disc 100. Pure oxygen is commonly used in a hyperbaric chambers 318 at 2 ATA. The present invention, depicted in Figure 12, shows a back pain patient lying flat in a hyperbaric chamber 318 using a leg support 319 to minimize disc pressure for maximum oxygen uptake. Since diffusion flows from high to low concentration, increased oxygen partial pressure will intensify endplate flow 326 and annular flow 325 into the disc 100 as shown in Figure 13. If 100% oxygen is used at 2 ATA in the hyperbaric chamber 318, the oxygen partial pressure will become 2.0, ten times higher than the oxygen partial pressure in ambient air at 1 ATA.

Since the disc 100 contains mostly incompressible water, a small disc volume change will provide a huge pressure difference within the disc 100. During sleep, the spine is in the supine position with minimal compressive loads. In addition, surrounding muscles are relaxed, allowing the disc 100 to expand in volume. As the disc 100 expands or increases in volume, disc pressure drops significant. As a result, water flows inward through convection (from high to low pressure). Chondroitin and keratan sulfates within the disc 100 provide water absorbing and retaining capabilities to pressurize the disc 100 during sleep. In essence, creating a small volume increase leads to a large disc pressure drop, promoting a convective uptake of fluid into the disc 100.

Figure 14 shows a patient on a traction bed 320 within the hyperbaric chamber 318 being pulled by an upper holder 285 toward one direction 289 and by a lower holder 286 toward the opposite direction 290. The traction bed 320 can also be a head-down tilt bed 320 within the hyperbaric chamber 318, as shown in Figure 15. Traction is used to create a small disc volume increase. However, the elevated pressure within the hyperbaric chamber 318 also compresses or collapses the disc 100, which was under atmospheric pressure (1 ATA). The hyperbaric compressive force upon the disc 100 can be estimated by multiplying the surface area of the disc 100 by the hyperbaric pressure in addition to 1 ATA or 14.7 PSI. The total surface area of the lumbar disc 100 is about 5.3 inch². Multiple hyperbaric pressures in addition to 14.7 PSI are listed as examples in Table 1. The hyperbaric compressive force in pounds upon the disc 100 is estimated using 5.3 inch² multiplied by the hyperbaric pressure in PSI, shown in Table 1.
Table 1: Hyperbaric Compressive Force on Disc and Oxygen Partial Pressure

<table>
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<tr>
<th>Hyperbaric Pressure in ATA (in addition to 1 ATA)</th>
<th>Hyperbaric Pressure in PSI (in addition to 14.7 PSI)</th>
<th>Compressive Force from Hyperbaric Pressure (Pounds)</th>
<th>Oxygen Partial Pressure using 20% O₂</th>
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<td>0.3</td>
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<td>23.2</td>
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<td>1.5</td>
<td>22.1</td>
<td>116.0</td>
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From calculation in Table 1, only about 23.2 pounds of traction is required to overcome 0.3 ATA added pressure (1.3 ATA total) within the hyperbaric chamber 318. In physical therapy, traction applied to the spine is within the patient’s comfort level, typically ranging from 10% to 50% of body weight. Some therapists may apply traction up to 80% body weight. Traction initially distracts surrounding muscles and ligaments, then eventually the tensile forces reaches the disc 100. Unlike the relatively sealed disc 100, added pressure from the hyperbaric chamber 318 does not significantly affect tensile resistance in muscles and ligaments. Tensile forces required for distracting muscles and ligaments are approximately the same between added pressure and normal atmospheric pressure.

With additional 0.7 ATA, total 1.7 ATA, a moderate 55-65 pounds traction is used to distract muscles and ligaments. The tensile force will reach and adequately overcome the added pressure applied over the disc 100 to create a small disc volume increase and a drastic disc pressure decrease. As a result, convective endplate flow 326 and annular flow 325 carrying nutrients and oxygen 317 will rush into the disc 100, as shown in Figure 16.

According to Henry’s Law (although non-linear), the increased oxygen partial pressure by using 100% oxygen to pressurize a hyperbaric chamber 318 will increase oxygen partial pressure within the avascular disc 100 through endplate flow 326 and annular flow 325, as shown in Figure 16. With the new supply of oxygen and nutrients 317 from fluid uptake, biosynthesis of chondroitin sulfate is enhanced. In the presence of oxygen, anaerobic production of lactic acid is reduced, normalizing pH within the disc 100 and minimizing acid irritation. In addition, normalizing pH helps to enhance uptake of sulfate ions into the disc 100, essential for biosynthesis of sulfated glycosaminoglycan. With renewed production of sulfated glycosaminoglycans, additional water is retained and swelling pressure of the disc...
100 is restored or partially restored, as depicted in Figure 17. The restored swelling pressure within the nucleus pulposus 128 distributes forces against the circumference of the inner annulus 316 to keep the lamellae bulging outward and sustaining compressive loads, as shown in Figure 18.

Table 1 shows significant increase in oxygen partial pressure within the hyperbaric chamber, especially using 100% oxygen. The high oxygen partial pressure in bodily circulation favors uptake of oxygen into the oxygen-deprived disc 100 through diffusion. In addition, as the disc 100 is distracted by traction, disc volume increases while the disc pressure drastically decreases. By combining (1) traction to decrease disc pressure, (2) using high oxygen concentration, and (3) hyperbaric chamber 318 to elevate pressure within bodily circulation, oxygen rapidly flows from bodily circulation into the deprived disc 100 through convection and diffusion.

The combination of traction and hyperbaric pressure can also be useful to treat disc bulging. Shear stresses can cause delamination, weakening and tearing of the annulus 316, which leads to disc bulging. The bulging annulus 316 is weak and often floppy. In addition, bulging near the neuroforamen can impinge upon the nerve root 194, as shown in Figure 11. Traction is used to widen the disc space, as shown in Figure 19. The floppy or bulging annulus 316 is more sensitive to hyperbaric pressure increase than the healthy annulus. The widened disc space provides entry for the bulging annulus 316. In addition, the distracted or widened disc space creates disc pressure drop. As the hyperbaric pressure increases, shown as annular flow 325 in Figure 20, the bulging annulus 316 is pressed inward into the low-pressure nucleus pulposus 128. While maintaining the pressure or annular flow 325, traction is released to restore normal disc height and trap the annulus 316 in the inward bulging position, as shown in Figure 21. As a result, nerve impingement by the outward bulging annulus 316 is minimized. The time between traction, hyperbaric pressure and release of traction is preferred to be short to avoid pressure build up within the disc 100. In conjunction with traction, lateral bending away from the bulge may provide additional space for inward movement of the bulging annulus 316.

As disc space narrows, nerve 194 impingement 329 by bone spurs 328 is common for patients with spinal stenosis, as shown in Figure 22. With traction and increased oxygen partial pressure within the hyperbaric chamber 318, diffusion and convective endplate flow
326 and annular flow 325 of nutrients and oxygen 317 into the disc 100 are intensified, as depicted in Figures 14-18, 23. As a result, biosynthesis of sulfated glycosaminoglycans increases to restore or partially restore the swelling pressure within the disc 100 to halt the progression or even reverse spinal stenosis.

Calcified endplates 105 are also common in cervical segments. Figure 24 shows a patient using multiple tractions simultaneously. A head harness 321 pulled by a tensile machine 322, an upper body holder 285 and lower body holder 286 are used within the hyperbaric chamber 318. Similarly, Figure 25 shows a patient on a head-down tilt traction bed 320 using a head harness 321 to apply traction from the neck with foot holders 324 to anchor the lower part of the body. As a result, cervical, thoracic and lumbar discs 100 obtain fluid uptake with high oxygen partial pressure from the combined traction and hyperbaric treatment.

Conduits 126 were proposed by J. Yeung and T. Yeung, PCT/US2004/14368 on May 7, 2004 and J. Yeung, PCT/US2005/22749 on June 22, 2005 to re-establish the exchange of nutrients and waste between the avascular disc 100 and bodily circulation. After implantation of the conduit 126, hyperbaric chamber 318 and/or traction bed 320 can be used to enhance transport of nutrients and oxygen 317 into the disc 100. The hyperbaric chamber 318 intensifies pressure to drive convective flows. In addition, the hyperbaric chamber 318 can be pressurized with 100% oxygen to increase oxygen partial pressure through the conduit 126 to saturate the oxygen-deprived disc 100. Traction is used to lower disc pressure to favor convective flows into the degenerative disc 100. A combination of hyperbaric pressure and traction further intensifies uptake of nutrients and oxygen 317 through the conduits 126.

Figure 26 shows flow of nutrients and oxygen 317 through the annular conduit 126 enhanced by the hyperbaric chamber 318 and/or traction to nourish the cells within the deprived disc 100. Similarly, the hyperbaric chamber 318 or traction can be used to enhance flow of nutrients and oxygen 317 through endplate conduits 126, as shown in Figures 27 and 28.

Chondroitin sulfate and keratan sulfate contain repeating disaccharides with molecular structures shown in Figure 29. The repeating disaccharides of chondroitin sulfate are D-glucuronic acid and N-acetyl-6-sulfate-D-galactosamine. The repeating disaccharides of keratan sulfate are D-galactose and N-acetyl-6-sulfate-D-glucosamine.

Multiple chains of chondroitin and keratan sulfates commonly attach to a core protein and a link protein bonding to a long chain hyaluronic acid to form proteoglycans, as depicted in
Figure 30. Hyaluronic acid also contains repeating disaccharides, glucuronic acid and N-acetyl-D-glucosamine, as shown in Figure 31. The connections between chondroitin and keratan to core proteins contain a terminal unit linking to serine or threonine of the core protein, as shown in Figure 32, to form proteoglycans. The nucleus pulposus 128 is rich in proteoglycans essential for retaining water within the disc 100. In addition, chondroitin and keratan sulfates with a terminal unit can also link to hydroxylsine of collagen, as shown in Figure 32, a major component of the annulus 316. The terminal unit of the chondroitin or keratan sulfate can contain \( \beta \)-glucuronic acid (1-3) – \( \beta \)-galactose (1-3) – \( \beta \)-galactose (1-4) – \( \beta \)-xylose to link with the core protein. The hydroxyl-group, –OH, of serine, threonine or hydroxylsine from protein or collagen is the likely functional group linking to the carbohydrate of the terminal unit. \( \beta \)-xylose of the terminal unit is possibly the linkage connecting to the serine or threonine of the core protein, or connecting to hydroxylsine of collagen. Bonding between the carbohydrate and protein/collagen to form proteoglycan is essential for retaining water and building tensile strength of the intervertebral disc 100.

Silicon is also present with approximately one atom of silicon per 130-280 repeating units of the polysaccharides in cartilage and chondroitin sulfate. Silicon is a biological crosslink agent that forms side chains of polysaccharides or proteoglycans. The crosslinked matrix provides additional strength and resiliency in connective tissue, such as the cartilaginous endplate 105, annulus 316, nucleus pulpos 128 and/or hyaline cartilage in joints. Moreover, the crosslinked matrix of proteoglycans or polysaccharides contains negative charges to absorb and retain a significant amount of water essential for the nucleus pulposus 128 and the intervertebral disc 100. The silicon crosslink agent can be orthosilicic acid, Si(OH)\(_4\), reacting with hydroxyl groups of the carbohydrates to form ether linkages which may bridge between two carbohydrate chains as: \( R_1 – O – Si(OH)_2 – O – R_2 \) or \( R_1 – O – Si(OH)_2 – O – Si(OH)_2 – O – R_2 \). Silanol compound, \((R_4)_2 Si(OH)_2\), can also be a crosslink agent to form \((R_4)_2 Si(\overline{O – R_1})(\overline{O – R_2})\), where \( R_1 \) and \( R_2 \) are carbohydrate chains and \( R_4 \) can be a methoxy group, –OCH\(_3\), or other components of the silanol compound. In essence, the free hydroxyl group, –OH, of silicon is a possible crosslink site to a carbohydrate.

The free –OH group of silicon can also react with serine or threonine of core protein to form proteoglycans, \( R_1 – O – Si(OH)_2 – O – Serine – Protein \), or \( R_1 – O – Si(OH)_2 – O – Threonine – Protein \). When the free –OH group of silicon reacts with hydroxylsine, the
crosslink may bridge between polysaccharide and collagen, \( R_1 - O - Si(OH)_{2} - O - \) hydroxylysine – Collagen. These types of linkages between proteoglycans and collagen may be the crucial structures for the tensile strength and water retaining capability of the intervertebral disc 100.

Similar to silicon in connective tissue, boron may be the crosslink agent between collagen chains to form a strong bone matrix. Boron, in the form of boric acid, \( B(-OH)_3 \), can link to collagen fortified or bound with calcium to form bone matrix: Calcium\(_{\text{bound}}\) – Collagen – Hydroxylysine – O – B(OH) – O – Hydroxylysine – Collagen – Calcium\(_{\text{bound}}\). Hydroxyproline in collagen can also link to boron as well. Boric acid is toxic, even lethal, in amounts of grams. In milligram quantities, boric acid is used as an anti-bacterial and anti-fungal food preservative. Other boron compounds, such as calcium borogluconate, can be used to build bone.

The severity of calcified formation 108 at the endplate105 increases with age. In a cadaveric study, more than half of elderly L4-5 specimens showed separations or gaps between the discs 100 and the adjacent vertebral bodies 159. The separation indicates disconnection between the disc 100 and the adjacent vertebral body 159. At the curved lower lumbar levels, such as L4-5 and L5-S1, separation or disconnection between the disc 100 and vertebral body 159 is particularly serious. Shear stress forces at these levels are intense. Separation between disc 100 and vertebral body 159 can lead to spondylolisthesis - forward slippage of vertebral body 159 from the disc 100 below. Spondylolisthesis often impinges upon nerve roots 194 and even the spinal cord 123, a serious condition requiring surgical intervention.

Boron and/or silicon can play a vital role in the connection between bone and connective tissue, including the cartilaginous endplate 105, disc 100 and tendons. A possible linkage between bone and connective tissue can be: Calcium\(_{\text{bound}}\) – Collagen – Hydroxylysine – O – B(OH) – O – Serine – Protein – Threonine – O – Si(OH)_2 – O – R\(_1\), where the section containing Calcium\(_{\text{bound}}\) – Collagen – Hydroxylysine – O – B(OH) – O – is bone, and the section containing – Serine – Protein – Threonine – O – Si(OH)_2 – O – R\(_1\) is connective tissue, such as cartilaginous endplate 105, annulus 316 or nucleus pulposus 128. Strong bonds or connections between the vertebral body 159 and endplate 105 or disc 100 can prevent spondylolisthesis.
Since diffusion of nutrients, from high to low concentration, is vital for survival of the avascular intervertebral disc 100, ingestion of nutritional supplements may significantly increase molar uptake of the nutrients into the degenerative disc 100. Chondroitin and keratan sulfates are too large to penetrate through the endplate 105 or outer annulus 316. However, the building blocks of chondroitin sulfate, keratan sulfate, proteoglycans and collagen can be ingested as a nutritional supplement to elevate their concentrations in the blood stream to enhance uptake into the avascular disc 100 through diffusion. The basic glycosaminoglycan building blocks of as nutritional supplement for biosynthesizing chondroitin sulfate, keratan sulfate and hyaluronic acid include: sodium sulfate, potassium sulfate, glucuronic acid, glucose, galactosamine, galactose and glucosamine. The building blocks as nutritional supplement for linking or crosslinking molecules include: xylose, serine, threonine, hydroxylsine, hydroxyproline, orthosilicic acid, silanol compound, boron compound, boric acid and calcium borogluconate. These linking elements are the connectors essential for structural formation of large and interconnecting biomolecules. The building blocks for core protein and link protein are the amino acids: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan. In addition to the building blocks, catalytic compounds, such as thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid, are essential in biosynthesizing proteoglycans. For cellular mitosis within the discs 100, adenine, guanine, thymine and cytosine are required for biosynthesizing deoxyribonucleic acid, DNA. For protein synthesis, uracil, ribose and sodium phosphate are required for ribonucleic acid, RNA, transcripted from DNA. In essence, elevated concentrations of nutrients 317 from the nutritional supplements by ingestion, injection or intravenous administering into bodily circulation can enhance uptake of nutrients 317 into the avascular disc 100 through diffusion to treat disc degeneration and back pain.

The use of traction or traction bed 320 can increase the uptake of oxygen and nutritional supplements 317 into the disc 100 through convection. The intervertebral disc 100 is usually in a compressed state from gravity and muscle tension. Traction pulls vertebrae apart to expand the disc 100 to increase disc volume and to decrease disc pressure. As a result, oxygen and nutritional supplement 317 in bodily circulation flow into the low pressure disc.
through convective endplate flows 326 and annular flows 325. Diffusion and convective flows can be further intensified with additional oxygen pressure in the hyperbaric chamber 318.

Since uptake of nutrients and oxygen is challenging, ultrasound can be used to enhance uptake of nutrients 317 to regenerate the degenerated disc 100. Figure 33 shows a patient on a traction bed 320 within a hyperbaric chamber 318, partially submerged in solution 330 and strapped 332 with ultrasound transducers 331 posterior-laterally. The solution 330 can be heated to relax the muscles to minimize tensile forces required to expand the disc 100. For maximum effectiveness using traction on the disc 100, muscle relaxants and/or local application of heat can also be used with traction. The solution 330 can be saline or fortified with nutrients 317 in the solution. In addition, a gel between the ultrasound transducer 331 and the skin can be used.

Vascular restriction or dilation can significantly affect the availability of nutrients diffusing into the avascular disc 100. A US Army study shows that cigarette smoking increases the risk of low back pain (O'Connor FG, Marlowe SS: Low back pain in military basic trainees. A pilot study, Spine, Aug;18(10):1351-1354, 1993). A subsequent study from Finland confirms that cigarette smoking leads to back pain through disc degeneration and spinal instability (Fogelholm RR, Alho AV: Smoking and intervertebral disc degeneration. Med Hypotheses, Apr; 56(4) 537-539, 2001). In animal studies, nicotine injection or exposure of cigarette smoke leads to acidic pH within the disc 100, reduced biosynthesis of glycosaminoglycans within the nucleus pulposus and/or annular delamination in rabbits or pigs (Hambly MF, Mooney V: Effect of smoking and pulsed electromagnetic fields on intradiscal pH in rabbits, Spine, June: 17(6 suppl) S83-5, 1992). Authors of the studies suggest that nicotine causes vasoconstriction, which limits the supply of nutrients into the intervertebral disc 100. (Holm S, Nachemson A: Nutrition of the intervertebral disc: Acute effects of cigarette smoking, An experimental animal study, Ups J. Med Sci, 93(1) 91-9, 1988). Similarly, disc degeneration can also be initiated by poor blood circulation. Patients with abdominal atherosclerosis (obstruction in the arteries) have a significantly higher risk of experiencing low back pain (Kurunlahti M, Tervonen O, Vanharanta H, Ikko E, Suramo I: Association of atherosclerosis with low back pain and the degree of disc degeneration, Spine,
Oct 15; 24(20) 2080-2084, 1999). In essence, inadequate blood supply to the cartilaginous endplates 105 or periphery of the disc 100 leads to disc degeneration.

Contrary to nicotine or atherosclerosis, vasodilators dilate or open blood vessels and capillaries to enhance blood flow to the calcified endplates 105 and periphery of the degenerated disc 100. As a result, the supply or availability of nutrients and oxygen 317 from blood vessels increases to nourish and regenerate tissue within the deprived intervertebral disc 100. The vasodilator can be adenosine, adrenomedullin, alkyl nitrite, amlodipine besylate, amyl nitrate, bradykinin, butyl nitrite, diazoxide, fenoldopam, flunarizine, hydralazine, isobutyl nitrite, isosorbide dinitrate, kinin, mannitol, minoxidil, niacin, nicardipine, nimodipine, nitric oxide, nitroglycerin, papaverine, pentaerythritol tetranitrate, pentoxifylline, piperazine, prazosin, prostacyclin, protaglandin E-1 (alprostadil), sildenafil citrate, sodium nitrite, sodium nitroprusside, tetrahydrocannabinol, theobromine, tolazoline or verapamil. The vasodilator can be ingested, injected, intravenous administered or inhaled. The vasodilator can be delivered in a pill, syringe, intravenous bag or canister. In addition, the vasodilator can also be used with nutritional supplements, traction devices, oxygen, hyperbaric chamber and/or ultrasound to further enhance diffusion and convective uptake for repairing the degenerated disc 100.

Similar to the effect of micro-gravity experienced by astronauts, traction of the spine promotes water uptake into the disc 100, leading to increased disc height. In essence, the intervertebral disc 100 is expandable or inflatable. However, the water is not retained when compressive loads return, unless more sulfated glycosaminoglycans or proteoglycans are available to retain the water within the disc 100. Under hyperbaric oxygen pressure within the chamber 318 with traction, the rate of water, nutrients and oxygen 317 uptake into the disc 100 is intensified through convective endplate flow 236 and annular flow 325. Unlike the astronauts in space, oxygen uptake into the disc 100 is concentrated by the increased oxygen partial pressure in the hyperbaric chamber 318. Oxygen and nutrients 317 are used to biosynthesize chondroitin and keratan sulfate to retain water and restore swelling pressure for sustaining compressive loads. In essence, the combination of traction bed 320 and hyperbaric chamber 318 with high partial pressure of oxygen is used to treat disc degeneration and back pain.
To further show the importance of sulfated glycosaminoglycans, a disc degenerative model was made using rat tails (Nishimura K., Mochida J: Percutaneous reinsertion of the nucleus pulposus, Spine, 23(14), 1531-9, 1998). A tail section involving three discs was twisted or rotated 45° and held for 2 weeks. The section was then compressed by coil springs and held for a period of time. All discs within the section degenerated quickly. The experiment was repeated. The discs were injected with additional nucleus pulposus from donor discs. Disc degeneration of the injected discs was significantly delayed. This experiment demonstrated the significance of sustainable swelling pressure from the water retained by the sulfated glycosaminoglycans. In summary, disc height is more likely to be maintained to protect the disc 100 when water is retained, even under extreme compressive loads.

Severity of endplate occlusion varies with age, genetic factor and life style. Uptake of fluid, nutrients and oxygen 317 depends on diffusion, convection and absorbency of the sulfated glycosaminoglycans. In mildly occluded endplates 105, daily nutritional supplements as mentioned before may be adequate to enhance diffusion of nutrients 317 into the intervertebral disc 100 to biosynthesize sulfate glycosaminoglycans. In moderately occluded endplates 105, oxygen inhalation can boost the uptake of both nutrients and oxygen 317 into the disc 100 in conjunction with the nutritional supplements, especially in the supine position. Vasodilator opens blood vessels and capillaries at or near endplates 105 and the periphery of the degenerated disc 100 to shorten the distance and improve the exchange of nutrients and waste between the degenerated disc 100 and bodily circulation. In addition, traction is useful to enhance convective flow to restore the water retaining sulfated glycosaminoglycans and proteoglycans within the disc 100. In cases of severe endplate occlusion, combinations of hyperbaric chamber 318, traction, nutritional supplements, vasodilator and ultrasound can be used to re-supply and repair the degenerated disc 100 with nutrients and oxygen 317.

Nutrients and oxygen 317 carried by the enhanced endplate flow 326 or annular flow 325 reach deep into the inner disc 100 and produce more sulfated glycosaminoglycans within the nucleus pulposus 128. The newly produced glycosaminoglycans or proteoglycans provide additional swelling pressure within the inner disc 100, as if re-inflating a flat tire. Restoration of swelling pressure within the nucleus pulposus 128 recreates the tensile stresses within the collagen fibers of the annulus 316, reducing the inner bulging and shear stresses between the
lamellae of the annulus 316. Disc bulging reduces and nerve impingement 329 is minimized. The load on facet joints 129 is significantly lifted. The motion segment is stabilized. Disc space narrowing ceases or is reversed.

In addition, oxygen supplied by increased partial pressure in the hyperbaric chamber 318 may drastically reduce the anaerobic production of lactic acid, thus decreasing chemical irritation and pain. With pH normalized within the disc 100, uptake of sulfate increases production of chondroitin sulfate and keratan sulfate. In the presence of oxygen, production of chondroitin sulfate may be favored over the anaerobic production of keratan sulfate, thus increasing the water-retaining capability in the disc 100.

The operative therapeutic pressure for treating back pain ranges between 1 and 5 ATA. For most patients, the most appropriate therapeutic pressure should be 1 to 2.5 ATA. The pressurized gas can contain between 15% and 100% oxygen. For high oxygen concentrations, especially under pressure, frequent air breaks are required to avoid oxygen toxicity. In addition, observation of patients by a trained operator is required. Nitrogen, helium, neon and/or other gases can be used to blend with oxygen to avoid oxygen toxicity. Some common compressed air for diving, such as Nitrox, Trimix, Heliox, Heliair, Neox, can also be used to pressurize the hyperbaric chamber 318. Different types of gas or mixture of gases can be used in series to (1) minimize time within the hyperbaric chamber 318, (2) avoid oxygen toxicity, (3) maximize treatment effectiveness, and (4) prevent nitrogen narcosis. In addition, the patient can also breathe through a mask with gases other than the one used to pressurize the chamber 318.

The hyperbaric chamber 318 can be made with steel, aluminum, acrylic and/or other material. The chamber 318 is equipped with air inlet 279, inlet valve 280, air outlet 281, outlet valve 282, entry hatches, airlock(s), two-way intercom, closed-circuit television, carbon dioxide scrubber and other equipment. The carbon dioxide scrubber within the pressure chamber 318 can be made with soda lime (75% calcium hydroxide, 20% water, 3% sodium hydroxide, 1% potassium hydroxide). The airlock allows medicines, instruments, food, drink, bedpan or other items to enter without excessively changing the pressure within the chamber 318. The chamber 318 may also be large enough to accommodate more than one patient.

The traction bed 320, jack 323, tilt bed 320, tensile machine 322 and other equipment within the hyperbaric chamber 318 are preferred to operate without using electricity to avoid
sparks or fire in the presence of high oxygen partial pressure. The equipment can be operative using hydraulic, air, gears or other means. The traction can also be created by weight or spring. The gas is frequently removed from the chamber to prevent build up of oxygen, which could provoke fire or oxygen toxicity.

The upper holder 285 can be a harness 285 around the chest, connected to an upper strap 287. The upper holder 285 can also be two axillary supports with soft shape-conforming wrappers for pulling from under the arms of the patient. In addition, the upper holder 285 can be one or two handles with soft wrappers for the hands. The handles also contain a switch. When the hands grab the handles, traction or tensile force begins. When the patient lets go of the handles, traction stops; then the handles reverse direction for about a foot, 0.3 meter, to allow the patient to re-grab the handles to resume traction treatment. Tensile forces or traction can be voice activated by the patient and/or operator. The tensile force can also be programmed with upper limits, intermittent, continuous, cycles, step-wise intensities, set time and/or combination. The tensile force can be on hold, creating a static stretch on the spine. Traction can be operated by movement of the upper holder 285, lower holder 286, or both. The lower holder 286 can be a harness 286 around the waist connected to a lower strap 288.

For the head-down tilt traction bed 320, the tensile force is related to the tilt angle, body weight of the patient and friction between the patient and the bed 320. The head-down tilt traction bed 320 does not require upper body support 285, although upper support 285 can intensify the traction. The compartment of the hyperbaric chamber 318 can accommodate the traction bed 320 with tilt angles ranging from 0° to 90°, where 0° is horizontal and 90° is vertical with head down. The tilt of the traction bed 320 can be achieved with a jack 323 or lifting element. Traction bed 320, head-down tilt bed 320, upper holder 285, lower holder 286, foot holder 324, head harness 321 and tensile machine 322 are traction devices design to provide tensile force to the disc 100. The term traction is used as a device or method. The purpose of traction, tensile force, tension, stretching and/or bending in this invention is related to methods and devices to promote uptake of nutrients and oxygen 317 into the deprived disc 100.

It is to be understood that the present invention is by no means limited to the particular constructions disclosed herein and/or shown in the drawings, but also includes any other modification, changes or equivalents within the scope of the claims. Many features have been
listed with particular configurations, curvatures, options, and embodiments. Any one or more of the features described may be added to or combined with any of the other embodiments or other standard devices to create alternate combinations and embodiments. Sequences of the suggested chemical bonding can be re-arranged or deleted. Molecules, such as protein, collagen, serine, threonine, silicon, boron compound, carbohydrate chain, can be substituted, re-arranged or deleted.

It should be clear to one skilled in the art that the current embodiments, materials, constructions, methods or tissues are not the only uses for which the invention may be used. Different materials, constructions, nutrients and methods can be substituted and used. Nothing in the preceding description should be taken to limit the scope of the present invention. The full scope of the invention is to be determined by the appended claims.
What is claimed is:

1. A treatment device for treating a patient having back pain, the treatment device comprising:
   - a hyperbaric chamber having dimensions to allow the patient to be located within the chamber,
   - and a traction device capable of providing traction to at least a portion of a spine of the patient.

2. The treatment device of claim 1, further comprising an oxygen supply capable of providing oxygen concentration in a range of 15% and 100% oxygen to the patient.

3. The treatment device of claim 2, wherein said oxygen supply provides 80% to 100% oxygen.

4. The treatment device of claim 2, wherein said oxygen supply provides approximately 100% oxygen.

5. The treatment device of claim 1, further comprising a breathing mask capable of supplying oxygen directly to the patient.

6. The treatment device of claim 1, further comprising a nutritional supplement located in the patient's body.

7. The treatment device of claim 6, wherein said nutritional supplement comprises:
   - at least one glycosaminoglycan building block chosen from the group of glycosaminoglycan building blocks consisting of: sodium sulfate, potassium sulfate, glucuronic acid, glucose, galactosamine, galactose and glucosamine,
   - and an at least one connector compound chosen from the group of connector compounds consisting of: xylose, serine, threonine, hydroxylysine, hydroxylproline, orthosilicic acid, silanol compound, boron compound, boric acid and calcium borogluconate.
8. The treatment device of claim 7, wherein said nutritional supplement further comprises at least one protein building block chosen from the group of protein building blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan.

9. The treatment device of claim 7, wherein said nutritional supplement further comprises at least one catalytic compound chosen from the group of catalytic compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid.

10. The treatment device of claim 7, wherein said nutritional supplement further comprises at least one compound chosen from the group of compounds consisting of: adenine, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

11. The treatment device of claim 7, wherein said nutritional supplement further comprises:
   at least one protein building block chosen from the group of protein building blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan,
   at least one catalytic compound chosen from the group of catalytic compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid,
   and at least one compound chosen from the group of compounds consisting of: adenine, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

12. The treatment device of claim 1, further comprising an ultrasound transducer locatable on a portion of skin of the patient.

13. The treatment device of claim 1, wherein said traction device is a head-down tilt bed.
14. The treatment device of claim 1, wherein said traction device includes an upper holder for holding a portion of the patient's upper body.

15. The treatment device of claim 1, wherein said traction device includes a lower holder for holding a portion of the patient's lower body.

16. The treatment device of claim 1, wherein said traction device is configured to provide a tensile force of 10% to 80% of a body weight of the patient.

17. The treatment device of claim 1, wherein said hyperbaric chamber provides pressure from 1 to 5 atmospheres.

18. The treatment device of claim 1, wherein said hyperbaric chamber provides pressure from 1 to 2.5 atmospheres.

19. The treatment device of claim 1, further comprising a vasodilator ingested, injected, intravenous administered or inhaled into the patient, said vasodilator selected from the group of vasodilators consisting of: adenosine, adrenomedullin, alkyl nitrite, amlodipine besylate, amyl nitrate, bradykinin, butyl nitrite, diazoxide, fenoldopam, flunarizine, hydralazine, isobutyl nitrite, isosorbide dinitrate, kinin, mannitol, minoxidil, niacin, nicardipine, nimodipine, nitric oxide, nitroglycerin, papaverine, pentaerythritol tetranitrate, pentoxifylline, piperazine, prazosin, prostacyclin, protaglandin E-1 (alprostadil), sildenafil citrate, sodium nitrite, sodium nitroprusside, tetrahydrocannabinol, theobromine, tolazoline and verapamil.

20. A nutritional supplement to enhance uptake of nutrients into an intervertebral disc of a patient for treatment of back pain, the nutritional supplement comprising:
   at least one glycosaminoglycan building block chosen from the group of glycosaminoglycan building blocks consisting of: sodium sulfate, potassium sulfate, glucuronic acid, glucose, galactosamine, galactose and glucosamine,
and at least one connector compound chosen from the group of connector compounds consisting of: xylose, serine, threonine, hydroxylysine, hydroxyproline, orthosilicic acid, silanol compound, boron compound, boric acid and calcium borogluconate.

21. The nutritional supplement of claim 20, further comprising at least one protein building block chosen from the group of protein building blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan.

22. The nutritional supplement of claim 20, further comprising at least one catalytic compound chosen from the group of catalytic compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid.

23. The nutritional supplement of claim 20, further comprising at least one compound chosen from the group of compounds consisting of: adenine, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

24. A vasodilator device to enhance the supply or availability of nutrients and oxygen from blood vessels into an intervertebral disc of a patient for treatment of back pain, the device comprising a vasodilator ingested, injected, intravenous administered or inhaled into the patient, said vasodilator selected from the group of vasodilators consisting of: adenosine, adrenomedullin, alkyl nitrite, amlodipine besylate, amyl nitrate, bradykinin, butyl nitrite, diazoxide, fenoldopam, flunarizine, hydralazine, isobutyl nitrite, isosorbide dinitrate, kinin, mannitol, minoxidil, niacin, nicardipine, nimodipine, nitric oxide, nitroglycerin, papaverine, pentacyrthritol tetranitrate, pentoxifylline, piperezine, prazosin, prostacyclin, protaglandin E-1 (alprostadil), sildenafil citrate, sodium nitrite, sodium nitroprusside, tetrahydrocannabinol, theobromine, tolazoline and verapamil.

25. A method of treating a patient having back pain, the method comprising the steps of:
(a) placing a patient having back pain in a hyperbaric chamber;
(b) increasing pressure within said hyperbaric chamber;
(c) and increasing a partial pressure of oxygen to the patient.

26. A method of treating a patient having back pain, the method comprising the steps of:
   (a) placing a patient in a hyperbaric chamber;
   (b) increasing pressure within said hyperbaric chamber;
   (c) and providing traction to a spine of the patient.

27. The method of claim 26, wherein said pressure in said hyperbaric chamber is between 1 and 5 atmospheres.

28. The method of claim 26, wherein said pressure in said hyperbaric chamber is between 1 and 2.5 atmospheres.

29. The method of claim 26, wherein said traction provides a tensile force of 10% to 80% of a body weight of the patient.

30. The method of claim 26, wherein said traction is provided by a head-down tilt bed.

31. The method of claim 26, wherein said traction is provided by a foot holder and a head harness.

32. The method of claim 26, wherein said traction is provided by an upper holder engaging a portion of the patient’s upper body.

33. The method of claim 26, wherein said traction is provided by a lower holder engaging a portion of the patient’s lower body.

34. The method of claim 26, wherein said pressure contains between 15 and 100% oxygen.
35. The method of claim 26, wherein said pressure contains between 80 and 100% oxygen.

36. The method of claim 26, further comprising a step (d) administering a nutritional supplement to the patient.

37. The method of claim 36, wherein the nutritional supplement administered includes:
   at least one glycosaminoglycan building block chosen from the group of glycosaminoglycan building blocks consisting of: sodium sulfate, potassium sulfate, glucuronic acid, glucose, galactosamine, galactose and glucosamine, and an at least one connector compound chosen from the group of connector compounds consisting of: xylose, serine, threonine, hydroxylysine, hydroxylproline, orthosilic acid, silanol compound, boron compound, boric acid and calcium borogluconate.

38. The method of claim 37, wherein said nutritional supplement administered further includes at least one protein building block chosen from the group of protein building blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan.

39. The method of claim 37, wherein said nutritional supplement administered further includes at least one catalytic compound chosen from the group of catalytic compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid.

40. The method of claim 37, wherein said nutritional supplement administered further includes at least one compound chosen from the group of compounds consisting of: adenine, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

41. The method of claim 37, wherein said nutritional supplement administered further includes:
at least one protein building block chosen from the group of protein building blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan,
at least one catalytic compound chosen from the group of catalytic compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid,
and at least one compound chosen from the group of compounds consisting of: adenine, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

42. The method of claim 36, wherein administering the nutritional supplement is done by a method chosen from the group of methods consisting of ingestion, injection and intravenous procedure.

43. The method of claim 26, further comprising a step (d) activating an ultrasound transducer located on a portion of the patient.

44. A method of treating a patient having back pain, the method comprising the steps of:
   (a) administering a nutritional supplement to the patient;
   (b) and providing an elevated concentration of oxygen to the patient.

45. The method of claim 44, wherein said elevated concentration of oxygen is approximately 100% oxygen.

46. The method of claim 44, further comprising a step (c) providing traction to the spine of the patient.

47. The method of claim 44, wherein the nutritional supplement administered in step (a) includes:
at least one glycosaminoglycan building block chosen from the group of
glycosaminoglycan building blocks consisting of: sodium sulfate, potassium
sulfate, glucuronic acid, glucose, galactosamine, galactose and glucosamine,
and at least one connector compound chosen from the group of connector compounds
consisting of: xylose, serine, threonine, hydroxylysine, hydroxyproline,
orthosilicic acid, silanol compound, boron compound, boric acid and calcium
borogluconate.

48. The method of claim 47, wherein said nutritional supplement administered in step (a)
further includes at least one protein building block chosen from the group of protein building
blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine,
aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and
tryptophan.

49. The method of claim 47, wherein said nutritional supplement administered in step (a)
further includes at least one catalytic compound chosen from the group of catalytic
compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine
hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid.

50. The method of claim 47, wherein said nutritional supplement administered in step (a)
further includes at least one compound chosen from the group of compounds consisting of:
adrenaline, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

51. The method of claim 47, wherein said nutritional supplement administered in step (a)
further includes:

at least one protein building block chosen from the group of protein building blocks
consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine,
histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine
phenylalanine, tyrosine and tryptophan,
at least one catalytic compound chosen from the group of catalytic compounds
consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine
hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid,
and at least one compound chosen from the group of compounds consisting of: adenine,
guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

52. A method of treating a patient having back pain, the method comprising the steps of:
   (a) administering a vasodilator to the patient
   (b) administering a nutritional supplement to the patient;
   (d) and providing traction to a spine of the patient.

53. The method of claim 52, wherein the vasodilator is selected from the group of
vasodilators consisting of: adenosine, adrenomedullin, alkyl nitrite, amlodipine besylate,
amyl nitrate, bradykinin, butyl nitrite, diazoxide, fenoldopam, flunarizine, hydralazine,
isobutyl nitrite, isosorbide dinitrate, kinin, mannitol, minoxidil, niacin, nicardipine,
nimodipine, nitric oxide, nitroglycerin, papaverine, pentahydrothritol tetrannitrate, pentoxifylline,
piperazine, prazosin, prostacyclin, prostaflavin E-1 (alprostadil), sildenafil citrate, sodium
nitrite, sodium nitroprusside, tetrahydrocannabinol, theobromine, tolazoline and verapamil.

54. The method of claim 52, wherein the nutritional supplement administered in step (b)
includes:
   at least one glycosaminoglycan building block chosen from the group of
   glycosaminoglycan building blocks consisting of: sodium sulfate, potassium
   sulfate, glucuronic acid, glucose, galactosamine, galactose and glucosamine,
and at least one connector compound chosen from the group of connector compounds
consisting of: xylose, serine, threonine, hydroxylysine, hydroxyproline,
orthosilicic acid, silanol compound, boron compound, boric acid and calcium
borogluconate.

55. The method of claim 54, wherein said nutritional supplement administered in step (b)
further includes at least one protein building block chosen from the group of protein building
blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan.

56. The method of claim 54, wherein said nutritional supplement administered in step (b) further includes at least one catalytic compound chosen from the group of catalytic compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid.

57. The method of claim 54, wherein said nutritional supplement administered in step (b) further includes at least one compound chosen from the group of compounds consisting of: adenine, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

58. The method of claim 54, wherein said nutritional supplement administered in step (b) further includes:

- at least one protein building block chosen from the group of protein building blocks consisting of: glycine, alanine, valine, leucine, isoleucine, lysine, arginine, histidine, aspartate, glutamate, asparagine, glutamine, cysteine, methionine phenylalanine, tyrosine and tryptophan,
- at least one catalytic compound chosen from the group of catalytic compounds consisting of: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine hydrochloride, cyanocobalamin, biotin, folic acid and ascorbic acid,
- and at least one compound chosen from the group of compounds consisting of: adenine, guanine, thymine and cytosine, uracil, ribose and sodium phosphate.

59. The method of claim 52, wherein said traction provides a tensile force of 10% to 80% of a body weight of the patient.

60. The method of claim 52, wherein said traction is provided by a head-down tilt bed.
61. The method of claim 52, wherein said traction is provided by a foot holder and a head harness.

62. The method of claim 52, wherein said traction is provided by an upper holder engaging a portion of the patient's upper body.

63. The method of claim 52, wherein said traction is provided by a lower holder engaging a portion of the patient's lower body.
Figure 28

Figure 29

Chondroitin 6-sulfate

Keratan sulfate
Proteoglycans

(Chondroitin sulfate)$_n$ -(Terminal unit)-(Serine)-(Core protein)-Hyaluronic acid backbone

(Chondroitin sulfate)$_n$ -(Terminal unit)-(Threonine)-(Core protein)-Hyaluronic acid backbone

(Chondroitin sulfate)$_n$ -(Terminal unit)-(Hydroxylysine)-Collagen backbone

(Keratan sulfate)$_n$ -(Terminal unit)-(Serine)-(Core protein)-Hyaluronic acid backbone

(Keratan sulfate)$_n$ -(Terminal unit)-(Threonine)-(Core protein)-Hyaluronic acid backbone

(Keratan sulfate)$_n$ -(Terminal unit)-(Hydroxylysine)-Collagen backbone