

TISSUE REPAIR WITH SPACE-SEEKING SPIRALS OF FILAMENT

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FIELD OF INVENTION

This invention relates to a spiraling device using filament to fill, pack and repair tissue, including intervertebral disc, urethral and fecal sphincters.

BACKGROUND

Chronic back pain is an epidemic. Nerve impingement is not seen by CT or MRI in about 85% of back pain patients [Deyo RA, Weinstein JN: Low back pain, N Eng J Med, 344(5) Feb, 363-370, 2001. Boswell MV, et. al.: Interventional Techniques: Evidence-based practice guidelines in the management of chronic spinal pain, Pain Physician, 10:7-111, ISSN 1533-3159, 2007]. In fact, lumbar disc prolapse, protrusion, or extrusion account for less than 5% of all low back problems, but are the most common causes of nerve root pain and surgical interventions (Manchikanti L, Derby R, Benyamin RM, Helm S, Hirsch JA: A systematic review of mechanical lumbar disc decompression with nucleoplasty, Pain Physician; 12:561-572 ISSN 1533-3159, 2009). The cause of chronic back pain in most patients has been puzzling to both physicians and patients.

Studies indicate back pain is correlated with high lactic acid in the disc. Leakage of the acid causes acid burn and persistent back pain. In addition, as the disc degenerates and flattens, the compressive load is shifted from the flattened disc to facet joints, causing strain and pain. Both lactic acid burn and strain of the facet joints are not visible under CT or MRI.

Urinary incontinence is common among women after multiple pregnancies. Weight of the fetus partially rests on the bladder, flattening and widening the bladder neck and urethral lumen. The sphincteric action of the urethral smooth muscle cannot contract far enough to close the widened lumen for coaptation of urethral mucosa, resulting in urinary incontinence.

SUMMARY OF INVENTION

A distal portion of a filament is extended beyond a distal end of a needle with a gripper. The needle with the extended filament is inserted into a cannula with snagging points at the distal opening of the cannula. The snagging points of the cannula hook and retain the distal portion of the filament. During partial withdrawal of the needle, a section of filament is deposited in the lumen of the cannula between the distal ends of the needle and cannula. When the needle is re-advanced, the section of filament is expelled or pushed out of the cannula into tissue. The needle is then rotated; the gripper engages and spirals the expelled filament to burrow into tissue. The needle can be further advanced to push and pack the spiral of filament deep into the tissue. The process of needle

partial withdrawal, re-advancement, rotation and pushing is repeated to pack and fill the tissue with interconnecting spirals of filament. Spirals of filament repair intervertebral discs to relieve back pain, or bulk sphincters to treat urinary or fecal incontinence.

REFERENCE NUMBERS

- | | | |
|----|------|---|
| 5 | 100 | Intervertebral disc |
| | 100A | L5-S1 disc |
| | 100B | L4-5 disc |
| | 100C | L3-4 disc |
| | 101 | Needle |
| 10 | 102 | Spinal needle |
| | 103 | Guide wire or tube |
| | 104 | Strand in filament |
| | 105 | Endplate |
| | 106A | Superior diffusion zone |
| 15 | 106B | Inferior diffusion zone |
| | 107 | Capillaries |
| | 108 | Calcified layers |
| | 109 | Vascular buds in endplates |
| | 110 | Window of cannula |
| 20 | 111 | Filament gripper of filament needle |
| | 112 | Extension bar |
| | 113 | Indentation between snagging points |
| | 114 | Annular delamination |
| | 115 | Epiphysis |
| 25 | 116 | Lumen of dilator |
| | 118 | Sensory nerve |
| | 119 | Epidural space |
| | 120 | Indentation between grippers |
| | 121 | Fissure |
| 30 | 123 | Spinal cord |
| | 124 | Pores of sponge filament |
| | 126 | Filament, shunt or disc shunt |
| | 126A | U-section or distal portion of filament |
| | 126B | Second proximal portion of filament |

	126C	First proximal portion of filament
	127	Cover or wrapper of filament
	128	Nucleus pulposus
	129	Facet joint
5	130	Filament needle handle
	131	Nutrients, oxygen and pH buffering
	132	Cannula handle
	133	Transverse process
	134	Spinous process
10	135	Lamina
	140	Ilium or iliac crest
	142	Superior articular process
	143	Inferior articular process
	152	Bobbin
15	159	Vertebral body
	160	Biosynthetic product or molecule
	162	Lactic acid
	163	Contrast agent
	184	Hole or void in tissue
20	193	Muscle
	194	Spinal nerve root
	195	Posterior longitudinal ligament
	220	Dilator
	230	Cannula
25	231	Snagging point, distal edge or rim of cannula
	268	Lumen of cannula
	269	Lumen of filament needle
	276	Syringe
	277	Cell
30	278	Pedicle
	378	Annulus or annular layer
	460	Thin spinal needle
	462	Anchor or toggle
	463	Knot

	492	Funnel into lumen of cannula
	493	Marker on filament needle
	494	Flexible holder of the latch
	495	Latch
5	496	Slope or slanted surface of latch
	497	Holder of bobbin
	498	Distal protrusion of cannula handle
	499	Proximal protrusion of cannula handle
	500	Distal protrusion of filament needle handle
10	501	Proximal protrusion of filament needle handle
	502	Friction ridges of needle handle
	504	Kambin's Triangle (safe entry into disc)
	505	Skin
	516	Urethra
15	517	Urethral lumen
	518	Bladder
	519	Bladder neck
	520	Vagina
	521	Pubis
20	522	Uterus
	523	Rectum
	524	Posterior wall of urethra
	525	Forceps
	526	Filament advancer
25	527	Stem of filament advancer
	528	Barbs or thread of filament advancer
	529	Cone head of dilator
	530	Removable luer lock or dilator handle
	531	Luer lock for syringe
30	532	Urethral smooth muscle
	533	Urethral lumen mucosa

DESCRIPTION OF THE DRAWINGS

Figure 1 shows capillaries 107 in vertebral bodies 159 providing oxygen, nutrients and pH buffer for the avascular intervertebral disc 100 through diffusion.

Figure 2 shows a longitudinal view of a healthy spinal segment with nutrients **131** supplied by capillaries **107** at the vertebral bodies **159** and endplates **105** to feed the cells within the disc **100**.

Figure 3 shows a graph of distance into disc from endplate versus oxygen concentration.

Figure 4 shows anaerobic production of lactic acid **162** in the mid layer of the disc **100**, leaking
5 and burning sensory nerve **118** and spinal nerve **194**.

Figure 5 shows leakage of lactic acid **162**, burning or irritating the spinal nerve **194**.

Figure 6 shows calcified layers **108** at the endplates **105**, blocking diffusion of nutrient, oxygen and pH buffer **131** from capillaries **107**, forming and leaking lactic acid **162** to nerve **118**.

Figure 7 shows vacuoles **184** in the disc **100** from degradation of proteoglycans to release
10 sugars for maintaining survival of disc cells.

Figure 8 shows load transfer from the flattened disc **100** to facet joint **129** causing pain.

Figure 9 depicts swaying of a vertebral body **159** above a disc **100** with low-swelling pressure.

Figure 10 depicts spinal instability from the low-pressure disc **100**, straining and wearing down the facet joints **129**.

Figure 11 shows insertion of a spinal needle **102** toward the surface of the degenerated disc **100**
15 to prepare for diagnostic discography.

Figure 12 shows a fluoroscopic anterior-posterior view of the needle **102**, about half way past pedicles **278**, entering into the disc **100**.

Figure 13 shows a fluoroscopic lateral view of the needle **102** entering into the disc **100** space,
20 but not into the epidural space **119**.

Figure 14 shows a small spinal needle **460** housed within the spinal needle **102**, puncturing into the nucleus pulposus **128** of the degenerated disc **100**.

Figure 15 depicts pain diagnostic discography by flushing lactic acid **162** from disc **100** to sensory nerve **118** with contrast agent **163** to provoke and confirm the excruciating pain.

Figure 16 shows sliding of spinal needle **102** over small spinal needle **460** into nucleus **128**.
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Figure 17 shows replacing the small spinal needle **460** with a guide wire **103**.

Figure 18 shows replacing the spinal needle **102** with a cone head **529** dilator **220**.

Figure 19 shows snagging points **231** at the distal end of a cannula **230**.

Figure 20 shows insertion of the cannula **230** sliding over the dilator **220**.

Figure 21 shows a filament needle **101** with at least one filament gripper **111**.
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Figure 22 shows a U-shaped filament **126** with the U-portion or distal portion **126A** extended from the filament needle **101**.

Figure 23 shows insertion of the filament needle **101** with filament **126** into the cannula **230**.

Figure 24 shows the dilator **220** in Figure 20 is replaced by the cannula **230** and needle **101**.

Figure 25 shows the snagging point **231** of the cannula **230** snags the filament strand **126B** during partial withdrawal of the needle **101** to deposit a section of filament **126B**, **126C** in cannula **230** between distal ends of cannula **230** and needle **101**.

Figure 26 shows handle **132** of cannula, handle **130** of filament needle and a spool of filament **126** on a bobbin **152** for feeding into the lumen **269** during withdrawal of the needle **101**.

Figure 27 shows re-advancement of the filament needle **101** from Figure 25, pushing the deposited filament **126B**, **126C** out of the cannula **230**.

Figure 28 shows filament **126** in nucleus **128** by re-advancing the needle **101**.

Figure 29 shows gripping of the filament **126** by the gripper **111** of the rotating needle **101**.

Figure 30 shows the needle **101** driving the filament **126** to spiral and burrow into tissue, void **184** or fissure **121**.

Figure 31 shows pushing of the needle **101** to pack and burrow the spiraled filament **126** into tissue, void **184** or fissure **121**.

Figure 32 shows snagging of the filament **126** by the snagging point **231** during withdrawal of the filament needle **101** to deposit additional filament **126B**, **126C** between distal ends of cannula **230** and needle **101**.

Figure 33 shows another re-advancement of the filament needle **101**, pushing the additional deposited filament **126B**, **126C** out of the cannula **230** into tissue.

Figure 34 shows the needle **101** driving the filament **126** to form another spiral, burrowing into tissue with previously spiraled filament **126**.

Figure 35 shows repeated steps of withdrawal, re-advancement, rotation and pushing of filament needle **101** to form multiple spiraled filaments **126** to bulk, fill or fortify tissue.

Figure 36 shows withdrawal of the cannula **230** and needle **101** after packing the nucleus **128** of degenerated disc **100** with spirals of filaments **126**.

Figure 37 shows cutting of the filament **126** near the skin **505**.

Figure 38 shows tucking of the filament **126** beneath skin **505** with a long-thin forceps **525**.

Figure 39 shows fluid flowing through the filament **126** from low osmotic pressure in muscle **193** to high osmotic pressure in desiccated disc **100**.

Figure 40 shows a longitudinal view of a disc **100** filled with spiraled filament **126**, wicking fluid from muscle **193**, superior **106A** and inferior **106B** diffusion zones into mid layer of disc **100**.

Figure 41 shows thickening of the filament-packed disc **100** to reduce facet loading and pain by lifting the inferior articular process **143** of the facet joint **129**.

Figure 42 shows production of biosynthetic molecules **160** or disc matrix by cells **277** receiving nutrients, oxygen and pH buffer **131** transported through the filament **126**.

Figure 43 shows intradiscal injection of nutrients, oxygen and pH buffer **131** and/or cells **277** to facilitate back pain relief and/or disc **100** regeneration.

Figure 44 shows shielding of L5-S1 disc **100A** and L4-5 disc **100B** by the iliac crest **140**, blocking entry of the straight spinal needle **102** of Figures 11 and 14.

5 Figure 45 shows iliac crest blocking of the lower lumbar disc **100**, preventing entry of spinal needle **102** into the nucleus of the disc **100**.

Figure 46 shows an elastically curved cannula **230** directing a flexible filament needle **101** into the center of the disc **100**.

Figure 47 shows normal position of a bladder neck **519** of a woman with urinary control.

10 Figure 48 shows funneling or widening of a bladder neck **519** leading to urinary incontinence.

Figure 49 shows a small spinal needle **460** within a cone-headed **529** dilator **220** fastened by a removable luer lock **530**.

Figure 50 shows insertion of cannula **230** and needle **101** to implant a spiraled filament **126** in smooth muscle **532** of the bladder neck **519** under cystoscopic or ultrasound guidance.

15 Figure 51 shows a cross-sectional view of a widened urethral lumen **517** and initial spiral of the filament **126** within the smooth muscle **532** of the urethra **516**.

Figure 52 shows narrowing of the urethral lumen **517** by bulking the urethral smooth muscle **532** with spirals of filament **126** to allow coaptation of urethral mucosa **533**.

20 Figure 53 shows two bulking locations of spiraled filaments **126** to close the urethral lumen **533** and relieve urinary stress incontinence.

Figure 54 shows bulking of the urethral smooth muscle **532** with spiraled filament **126**, narrowing the urethral lumen **517** at the bladder neck **519** to regain sphincteric control.

Figure 55 shows a knot **463** to prevent retraction of the distal end of the filament **126A** in the needle **101**, and to facilitate filament **126** snagging by the snagging points **231** of cannula **230**.

25 Figure 56 shows the filament **126C** in the needle **101**, filament **126B** outside the needle **101**; and the filament **126A** is the distal portion between filament **126C** and filament **126B**.

Figure 57 shows a linear or single stranded filament **126** tied with a knot **463** extending from the needle **101**.

30 Figure 58 shows an anchor, knob or toggle **462** on a linear filament **126** extending from the needle **101** to facilitate filament **126** snagging by the cannula **230**.

Figure 59 shows a cross bar **111** as a filament gripper **111**.

Figure 60 shows the filament **126** looping over the cross bar **111** for filament **126** spiraling.

Figure 61 shows a cross plane **111** as a filament gripper **111**, dividing the cylindrical lumen **269** into semi-cylinders **269** for housing the filament strand **126C** and filament strand **126B**.

Figure 62 shows an extended cross bar 111 as a filament gripper 111 connecting to extension bars 112. The extended cross bar 111 can also be an extended cross plane within the lumen 269.

Figure 63 shows the filament 126 looping over the extended cross bar 111 or extended cross plane 111 for spiraling and pushing of the filament 126.

5 Figure 64 shows cross stubs 111 as a filament gripper 111 for spiraling and pushing of the filament 126.

Figure 65 shows longitudinal cross stubs 111 along the lumen 269 of the needle 101 for spiraling and pushing of the filament 126.

10 Figure 66 shows a filament advancer 526 with resiliently collapsible barbs 528, moving in cyclical distal-proximal motion to advance or propel the filament 126 out of the needle 101.

Figure 67 shows open and closed positions of the resiliently collapsible barb 528.

Figure 68 shows an axial or vertical view of the filament advancer 526 with resiliently collapsible barbs 528 approximately 120 degrees apart.

15 Figure 69 shows an axial or vertical view of the filament advancer 526 with resiliently collapsible barbs 528 approximately 90 degrees apart.

Figure 70 shows a rotational auger 526 as a filament advancer 526 to convey or propel the filament 126 out the needle 101.

Figure 70A shows a cross section of a needle 101 with a lumen 269 configuration to accommodate the filament advancer 526 and filament 126.

20 Figure 71 shows a cannula 230 with multiple snagging points 231.

Figure 72 shows a cannula 230 with a window 110 open to a snagging point 231.

Figure 73 shows snagging points 231 of a cannula 230 with inward bending or curvatures.

Figure 74 shows curvatures of snagging points 231 of another cannula 230.

25 Figure 75 shows inward bending or curving walls or gates 231 as snagging points 231 at the distal end of the cannula 230.

Figure 76 shows a cross-sectional view of the inward bending gates 231 of the cannula 230.

Figure 77 shows braided strands 104 forming the filament 126.

Figure 78 shows woven strands 104 forming the filament 126.

Figure 79 shows knitted strands 104 forming the filament 126.

30 Figure 80 depicts a slanted cut of the filament 126, showing the slanted orientations of strands 104 relative to length-wise of the filament 126.

Figure 81 shows a cross-section of the filament 126 made with parallel strands 104 wrapped, encircled, covered or enveloped by a sheath or cover 127.

Figure 82 shows a cross-section of the filament 126 made with parallel tubes 104 wrapped, encircled, covered or enveloped by a sheath or cover 127.

Figure 83 shows a cross-section of the filament 126 made with sponge or foam with pores 124.

Figure 84 shows a cross-section of a solid filament 126, similar to a mono-filament suture.

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DETAILED DESCRIPTION OF THE EMBODIMENTS

Intervertebral discs are avascular (no blood vessels). Nutrients, oxygen and pH buffer 131 essential for disc cells are supplied by the capillaries 107 in the vertebral bodies 159 and diffused from superior and inferior endplates 105 into the disc 100, as shown in Figures 1 and 2. Blood pH is tightly regulated between 7.35 and 7.45, mainly by the pH buffering bicarbonate dissolved in blood plasma diffused through superior and inferior endplates 105 into the disc 100.

However, depth of diffusion is shallow into thick human discs 100. Depth of oxygen diffusion from the endplates 105 is summarized in Figure 3 (Stairmand JW, Holm S, Urban JPG: Factor influencing oxygen concentration gradients in disc, Spine, Vol. 16, 4, 444-449, 1991). Similarly, depths of glucose diffusion are less than 3 mm from superior and inferior endplates (Maroudas A, Stockwell RA, Nachemson A, Urban J: Factors involved in the nutrition of the human lumbar intervertebral disc: Cellularity and diffusion of glucose in vitro, J. Anat., 120, 113-130, 1975). Nearly all animals have thin discs; depths of diffusion of oxygen and nutrients seem to be sufficient. Lumbar discs of a large sheep weighing 91 kg (200 pounds) are less than 3 mm thick. However, human lumbar discs are about 7-12 mm thick. Mid layers of our thick discs 100 suffer severe oxygen and nutritional deficiency.

Under anaerobic condition within the mid layer, lactic acid 162 is produced and leaked from the nucleus 128 through fissures 121 to burn surrounding nerves 118, 194 causing persistent back pain, as depicted in Figures 4-6. Some patients experience leg pain without visible nerve impingement under MRI or CT. Lactic acid 162 can leak from the nucleus 128 through fissures 121 to spinal nerves 194, causing leg pain as depicted in Figures 4-5. Leg pain without visible impingement is commonly called chemical radiculitis.

High lactic acid content in discs correlates with back pain. In fact, dense fibrous scars and adhesions, presumably from lactic acid 162 burn, can be found around nerve roots 194 during spinal surgery [Diamant B, Karlsson J, Nachemson A: Correlation between lactate levels and pH of patients with lumbar rizopathies, Experientia, 24, 1195-6, 1968. Nachemson A: Intradiscal measurements of pH in patients with lumbar rhizopathies. Acta Orthop Scand, 40, 23-43, 1969. Keshari KR, Lotz JC, Link TM, Hu S, Majumdar S, Kurhanewicz J: Lactic acid and proteoglycans as metabolic markers for discogenic back pain, Spine, Vol. 33(3):312-317, 2008].

As we age, calcified layers **108** form and accumulate at the endplates **105**, blocking capillaries **107** and further limiting the depth of diffusion of nutrients/oxygen/pH buffer **131** into the disc **100**, as shown in Figure 6. Mid layers of the disc **100** suffer chronic and severe starvation and anaerobic conditions. Disc cells can survive without oxygen, but die without sugars. Nucleus **128** contains glycosaminoglycans with covalently bonded sugars, essential for retaining water in the disc **100**. Degradation of glycosaminoglycans to release sugars for consumption allows disc cells to survive, but initiates compositional and structural change, creating voids **184** and loosely packed nucleus matrix in a degenerated disc **100**, as shown in Figure 7, [Urban JP, Smith S, Fairbank JCT: Nutrition of the Intervertebral Disc, Spine, 29 (23), 2700-2709, 2004. Benneker LM, Heini PF, Alini M, Anderson SE, Ito K: Vertebral endplate marrow contact channel occlusions & intervertebral disc degeneration, Spine V30, 167-173, 2005. Holm S, Maroudas A, Urban JP, Selstam G, Nachemson A: Nutrition of the intervertebral disc: solute transport and metabolism, Connect Tissue Res., 8(2): 101-119, 1981].

Composition Change of the Intervertebral Discs (approximation)

	Normal Discs	Painful Discs	% Change from Normal Discs
Glycosamino-glycans	27.4 ± 2.4%	14.1 ± 1.1%	-48.5%
Collagen	22.6 ± 1.9%	34.8 ± 1.4%	+54%
Water content	81.1 ± 0.9%	74.5 ± 1%	-8.1%
Acidity	pH 7.14 [H ⁺]: 7.20X10 ⁻⁸	pH 6.65 - 5.70 [H ⁺]: 2.23X10 ⁻⁷ to 2.00X10 ⁻⁶	[H ⁺]: +208% to +2,661%

(Reference: Kitano T, Zerwekh J, Usui Y, Edwards M, Flicker P, Mooney V: Biochemical changes associated with the symptomatic human intervertebral disk, Clinical Orthopaedics and Related Research, 293, 372-377, 1993. Scott JE, Bosworth TR, Cribb AM, Taylor JR: The chemical morphology of age-related changes in human intervertebral disc glycosaminoglycans from cervical, thoracic and lumbar nucleus pulposus and annulus fibrosus. J. Anat., 184, 73-82, 1994. Diamant B, Karlsson J, Nachemson A: Correlation between lactate levels and pH of patients with lumbar rhizopathies, Experientia, 24, 1195-1196, 1968. Nachemson A: Intradiscal measurements of pH in patients with lumbar rhizopathies, Acta Orthop Scand, 40, 23-43, 1969.)

When glycosaminoglycans diminish, water content and swelling pressure in the nucleus pulposus **128** decrease. The nucleus **128** with reduced swelling pressure can no longer distribute forces evenly against the circumference of the inner annulus **378** to keep the annulus bulging outward. As a result, the inner annulus **378** sags inward while the outer annulus **378** bulges outward, creating annular delamination **114** and weakened annular layers **378**, possibly initiating fissure **121** formation depicted in Figures 5-6. Holes or vacuoles **184** can be found during

dissection of cadaveric discs **100**, as shown in Figure 7. Nucleus pulposi **128** of degenerated discs **100** are usually desiccated, with reduced swelling pressure and decreased capability to sustain compressive loads. The compressive load is thus transferred to the facet joints **129**, pressing the inferior articular process **143** against the superior articular process **142** of the facet joint **129**, causing strain, wear and/or pain as shown in Figure 8 (Dunlop RB, Adams MA, Hutton WC: Disc space narrowing and the lumbar facet joints, Journal of Bone and Joint Surgery - British Volume, Vol. 66-B, Issue 5, 706-710, 1984).

A disc **100** with reduced swelling pressure is similar to a flat tire with flexible or flabby side walls. The vertebral body **159** above the soft or flabby disc **100** easily shifts or sways, as shown in Figure 9. This is commonly called segmental or spinal instability. As shown in Figure 10, the frequent or excessive movement of the vertebral body **159** strains the facet joints **129**, which are responsible for limiting the range of segmental mobility. Patients with spinal instability often use their muscles to guard or support their spines to ease facet pain. As a result, muscle tension and aches arise, but are successfully treated with muscle relaxants. Spinal motions, including compression, torsion, extension, flexion and lateral bending, were measured before and after saline injection into cadaveric discs. Intradiscal saline injections into slow leaking cadaveric discs reduced all spinal motions (Andersson GBJ, Schultz AB: Effects of fluid on mechanical properties of intervertebral discs, J. Biomechanics, Vol. 12, 453-458, 1979).

Discography is a common diagnostic technique for identifying or confirming a painful disc **100** before surgical intervention. A spinal needle **102** is guided by a fluoroscope toward the Kambin's Triangle **504** in Figures 8 and 11, a posterior-lateral area through which spinal needle **102** can access a lumbar disc **100** safely. The anterior-posterior view in Figure 12 guides the needle **102** between endplates **105**, but does not show the ventral-dorsal location of the needle **102** tip. Before passing the pedicle **278** midway, a lateral fluoroscopic view depicted in Figure 13 must be taken to ensure the needle **102** is not too dorsal, entering into the epidural space **119**. Figure 13 depicts the lateral fluoroscopic view, showing the needle **101** tip is ventral to the epidural space **119** and can safely enter into the mid layer of the disc **100**.

In literature, sizable disc **100** puncturing or laceration accelerates disc degeneration. In non-painful discs **100**, a small spinal needle **460** within the spinal needle **102** is used to puncture the disc **100** as shown in Figure 14. Figure 15 shows intradiscal injection of X-ray contrast **163**, flushing out lactic acid **162** in the disc **100** through fissures **121** to burn the sensory nerve **118**, instantaneously causing excruciating pain and confirming specific painful disc **100**. For normal or non-painful discs, discography is nearly painless. The spinal needle **102** is not shown in Figure 15.

The spinal needle 102 in Figures 11-16 allows joining of diagnostic discography with therapeutic intervention to relieve back pain during the same visit to save time and reduce pain.

After confirming discogenic pain with discography, the needle 102 is advanced into the painful disc 100 over the small spinal needle 460, in Figure 16. The small spinal needle 460 is replaced
5 with a blunt guide wire 103 in the spinal needle 102 into the disc 100, in Figure 17. The proximal end of the guide wire 103 is held stationary during withdrawal of the spinal needle 102 from the patient. The proximal end of the guide wire 103 is also held stationary to slide a dilator 220 with a blunt cone head 529 over the guide wire 103 into the disc 100 or tissue, as shown in Figure 18. A cannula 230 has at least one snagging point 231 and longitudinal lumen 268, as shown in Figure 19.
10 The snagging point 231 can also be a sharp edge 231 with filament gripping or catching capability. The cannula 230 can have a window 110 opened to the lumen 268 and distal opening. While the proximal end of the dilator 220 is held stationary, the cannula 230 is advanced, sliding over the dilator 220 into the disc 100, as shown in Figure 20. The guide wire 103 and dilator 220 are removed. A filament needle 101 contains at least one gripper 111 and a longitudinal lumen or
15 opening 269 as shown in Figure 21. Distal end 126A of a filament 126 is extended from the needle lumen 269, as shown in Figure 22. The distal end 126A of the filament 126 can be U-shaped. The filament needle 101 with the filament 126 is inserted into the lumen 268 of the cannula 230 into the disc 100, as shown in Figures 23-24. A funnel 492 in the proximal end of cannula handle 132 in Figure 26 is used to facilitate insertion of the filament needle 101 of Figure 22 into the proximal
20 opening of cannula 230.

During partial withdrawal of the filament needle 101 into the cannula lumen 268, the distal portion 126A or extended portion of the filament 126 is snagged, caught, hooked, retained, trapped, held or grabbed by the snagging point 231 in Figure 25. As a result, the distal portion 126A or
25 extended portion of the filament 126 remains outside in contact with surrounding tissue in front of the distal end of the cannula 230; and a section of filament 126B, 126C is deposited in the lumen 268 of cannula 230 between the distal ends of the cannula 230 and needle 101. A bobbin 152 holding a spool of filament 126 is fastened at the proximal end of a needle handle 130, as shown in Figure 26. During snagging or holding of the distal portion 126A of the filament 126 and partial withdrawal of the needle 101, additional filament 126 from the bobbin 152 feeds into the lumen
30 269 of the needle 101. The needle 101 is re-advanced from Figure 25, pushing or expelling the deposited filament 126B, 126C in front of the needle 101 out the cannula 230 into tissue, as shown in Figures 27-28. Excessive withdrawal of the filament needle 101 will deposit a long filament 126B, 126C in front of the filament gripper 111 of the needle 101 within the cannula 230. The long filament 126B, 126C within the cannula 230 can jam the re-advancing needle 101. A latch 495 on

a flexible spring or nitinol wire **494** extends from a cannula handle **132** to limit and prevent excessive length or distance of partial withdrawal of the filament needle **101** by stopping the proximal protruded end **501** of the needle handle **130**. Hence, length of filament **126B**, **126C** deposited within the cannula **230** from partial withdrawal of needle **101** is short enough to be pushed or expelled out the cannula **230** by re-advancement of the filament needle **101**, as shown in Figures 27 and 28. The deposited filament **126B**, **126C** may also exit through the window **110** of the cannula **230** in Figures 19 and 27 to avoid jamming during re-advancement of the needle **101**. During insertion of the filament needle **101** in Figure 22 through the funnel **492** leading into the lumen **268** of the cannula **230**, the needle handle **130** slides over the slope **496** of the latch **495** for filament **126** spiraling. Figure 26 shows a marker **493** on the needle **101**. Distal ends of both filament needle **101** and cannula **230** are even, as shown in Figure 23 or 27, when the marker **493** lines up at the opening of the funnel **492**. When the marker **493** enters into the funnel **492**, the grippers **111** of the needle **101** are extended outside the cannula **230** to push and pack the filament **126** into tissue.

Major blood vessels, abdominal aorta, inferior vena cava and common iliac arteries, are anterior to the lumbar discs. Distal ends of the devices must remain within the discs **100**, confirmable by fluoroscope (X-ray) or markers on the devices to minimize intermittent X-ray exposure to patients and physicians. Markers on proximal ends of the guide wire **103** and dilator **220** help physician to identify device orientation. The following table shows lengths and markers on sequential devices to guide implantation of spiraled filament **126** into disc **100** to relieve back pain. Markers in the table are measured from the distal end of the device. For safety of the patient, length of a guiding device is preferred to be longer than the subsequent device, so that the proximal end of the guiding device can be held stationary during insertion of the subsequent device into patient.

Device	OD, mm	ID, mm	Length, mm	Marker 1, mm	Marker 2, mm
Spinal needle 102	1.27	0.84	172		
Small spinal needle 460	0.51	0.25	226		
Guide wire 103	0.81		492	172	338
Dilator 220	1.83	1.00	338	185	
Cannula 230	2.41	1.97	185		
Filament needle 101	1.83	1.52	230	185	
Filament 126	0.67-0.77		900 double strands		

Distal ends of spinal needle **102** and guide wire or tube **103** are even, when Marker 1 of the guide wire **103** is at the proximal end of the spinal needle **102**, as shown in Figure 17. Distal ends of the dilator **220** and guide wire **103** are even, when Marker 2 of the guide wire **103** is at the proximal

end of the dilator **220**, as shown in Figure 18. Distal ends of cannula **230** and dilator **220** are even, when Marker 1 of the dilator **220** is at the proximal end of cannula **230**, as shown in Figure 20. Distal ends of the cannula **230** and filament needle **101** are even, when Marker 1 of the needle **101** is at the proximal end of the cannula **230**, as shown in Figure 24.

5 Annulus **378** of the disc **100** is made mainly with layers of collagen. Layers of collagen form a net-like matrix. Spinal needles **102** or **460** with single sharp tips have no problem puncturing through the net-like collagen matrix of the annulus **378**. On the other hand, the cannula **230** and filament needle **101** have multiple sharp points **231**, **111** at the distal ends, as forks. The cannula **230** contains indentations **113** between snagging points **231** in Figure 19; and filament needle **101** contains indentations **120** between grippers **111** in Figures 21-22. The indentations **113**, **120** are trapped by the net-like collagen matrix with no annular penetration capability to prevent possibility of injuring blood vessels anterior to the discs **100**. The guide wire or tube **103** and dilator **220** are blunt and have little capability of penetrating annulus **378** to ensure safety of the patient.

15 During pushing and rotation of the filament needle **101**, at least one of the grippers **111** grabs and spins the extended filament **126B**, **126C** burrowing and spiraling into nucleus **128**, fissure **121**, void **184** or soft tissue, as shown in Figure 29. Spiraling of the extended filament **126** is made possible by the twisting gripper **111** and friction between the extended filament **126** and tissue. A knot-like spiraled filament **126** is formed due to rotation of the filament needle **101**, as shown in Figure 30. The filament needle **101** can also advance or extend beyond the distal end of the
20 cannula **230** to push, pack, fill, load, cram or stuff the expelled filament **126**, burrowing into nucleus **128**, void **184**, fissure **121** or tissue, as shown in Figure 31. The filament needle **101** is partially withdrawn again; the extended filament **126** is caught again by the snagging point **231** of the cannula **230**, as shown in Figure 32. In fact, the initial spiraled filament **126** also acts as an anchor outside the distal end of the cannula **230**; partial withdrawal of the needle **101** deposits or
25 loads additional filament **126B**, **126C** within the cannula **230** between distal ends of cannula **230** and needle **101**. The filament needle **101** is re-advanced, pushing or expelling the additional filament **126** out of the cannula **230** into tissue **31**, as shown in Figure 33. Rotations of the needle **101** form another spiraled filament **126**, linking to the previously spiraled filament **126**, as shown in Figure 34. Same direction of needle **101** rotations is preferred. Rotation and pushing of the needle
30 **101** help the spiraled filament **126** to seek, burrow and fill void **184**, gap, fissure **121**, vacancy, weak spot, opening, cavity or soft spot. The steps of needle **101** withdrawal, re-advancement, rotation and pushing, are repeated to build, fill, bulk, pack, fortify, load or solidify the tissue with multiple inter-connecting spirals of filament **126**, as shown in Figures 35-36. Multiple spirals or coils of filament **126** are formed, packed or inserted individually to be shape conforming, malleable

and/or resilient in voids 184, yet inter-connected to prevent or minimize migration from tissue. Rotation and pushing of the needle 101 drive the spirals of filament 126 from the lumen 269 of the needle 101 to seek, fill and pack the distal, lateral and/or proximal space in tissue. The spirals of filament 126 are remained and probably formed in multiple axes. Tightness of the spiraled filament 126 is determined by number of rotations and intensity of pushing of the needle 101. The spiral filament 126 can be as tight as a suture knot. Fewer rotations and/or gentle pushing of the needle 101 make soft spirals of filament 126. Number of rotations and pushing intensity of the needle 101 can be alternate to allow variations in firmness or density of filament spirals 126 within repaired tissue. It is possible to sequential withdraw the cannula 230 from tissue to provide additional distal space for packing additional spirals of filament 126.

Needle handle 130 and cannula handle 132 are dumbbell shape. The needle handle 130 has a protruded proximal end 501 and a protruded distal end 500 to facilitate withdrawal and advancement of the needle 101, as shown in Figure 26. The needle handle 130 also contains gripping or friction ridges 502 to facilitate rotation of the needle 101. The cannula handle 132 has a protruded proximal end 499 and a protruded distal end 498 to facilitate withdrawal and advancement of the cannula 230. When the tissue is full with spirals of filament 126, advancement of the needle 101 becomes difficult. The cannula 230 can also be slightly and sequentially withdrawn from tissue, to pack or accumulate additional spirals of filament 126 distal to the cannula 230. A metering device can be attached to the spool of filament 126 on the bobbin 152 to monitor length of filament 126 dispensed into patient. When the tissue is packed with spirals of filament 126, the filament needle 101 and cannula 230 are withdrawn from tissue. In the event of disc 100 repair, the extended filament 126 is cut above the skin 505, as shown in Figure 37. The proximal portions of filament 126B, 126C are tucked beneath the skin 505 with a long and thin forceps 525, as shown in Figure 38. The amount of implanted filament 126 is selectable, controllable, limitable or regulateable by the physician.

In summary, the cannula 230 and filament needle 101 work together to spiral the filament 126 or shunt 126 bulking the tissue. The stationary cannula 230 with snagging points 231 prevents the extended filament 126A from retrieving or retracting into the lumen 269 of the filament needle 101. Additional filament 126 is advanced by either withdrawal/re-advancement of the needle 101 or by a filament advancer 526 within the needle 101. Rotation of the needle 101 with grippers 111 holds and spirals the filament 126 burrowing into voids 184 and fissure 121. The filament spiral 126 is individually formed by spatial allowance of the tissue, and not spiraling over a spindle, axle, axis or needle. Filament 126 spiraling driven by the rotating needle 101 is space seeking, filling, fitting or

conforming to fortify, bulk, fill, cushion or repair the tissue. Pushing of the needle **101** further packs the spiraled filament **126** to bulk the tissue.

PCT/US2011/000007, WO/2011/082390 (Internal and external disc shunts alleviate back pain, by Jeffrey E. Yeung and Teresa Yeung, filed on 3 January, 2011) contains a U-shaped shunt **126** partly within and partly outside a needle **101**, and a sleeve **220** sliding over the needle **101**. During rotation of the needle **101**, the outside draping shunt **126** spirals over the needle **101** shaft. The sleeve **220** is then advanced distally to strip the spiraled shunt **126** off the shaft of the needle **101**, pushing the spiraled shunt **126** into the nucleus of the intervertebral disc **100**. The sleeve **220** is retrieved to the proximal position. Another spiral of shunt **126** is formed by rotation of the needle **101**, the sleeve **220** is advanced again to strip the spiraled shunt **126** off the needle **101** into the nucleus. The process of needle **101** rotation, sleeve **220** advancement and sleeve **220** retrieval are repeated to form spirals of shunt **126** within the disc **100**. However, many problems occurred during usages of this device in clinical study. Due to friction between disc **100** and sleeve **220**, advancement of the sleeve **220** for stripping the spiraled shunt **126** from the needle **101** is very difficult. Significant force is required to advance the sleeve **220**, which adds significant risks of puncturing through the disc **100** and rupturing major blood vessels anterior to the disc **100**. In addition, due to direct contact of the sleeve **220** with the painful disc **100**, patient feels extreme pain during advancement of the sleeve **220** to dislodge the spiraled shunt **126** on the needle **110**. Significant pain is endured during multiple dislodgements of shunt spirals **126**. Fortunately, the patient experienced pain relief within a week due to efficacy of the disc shunt **126**.

Unlike PCT/US2011/000007, WO/2011/082390, the cannula **230** in this application is stationary during implantation of the filament **126** into disc **100** or tissue. The filament needle **101** slides freely within the cannula **230** without risk to the patient or causing pain. The spiral of filament **126** is driven by the grippers **111** and formed distal to the rotating needle **101**, capable of burrowing, drilling, packing, wiggling, building, dilating, wedging or shimming into voids **184**, fissure **121** or tissue. Unlike PCT/US2011/000007, tissue burrowing of the distal forming filament spiral **126** in this application is particularly effective, deep, and tight, as shown in Figure 30, embedding, filling, packing and bulking the tissue as shown in Figure 36. The spiraled filament **126** in PCT/US2011/000007, WO/2011/082390 is already formed on the needle **101** shaft, which may not fit into small voids **184** or fissures **121**. In addition, spiraling of the filament **126** in this application is particularly unique. Filament **126** spiraling can occur only in tissue, interference or friction surrounding the extended filament **126**, different from disc shunt **126** spiraling the over the needle **101**, as taught in PCT/US2011/000007, WO/2011/082390.

Fluid flows from low to high osmotic pressure according to the law of physics. Osmotic pressure of blood plasma in muscle **193** is approximately 250-300 mOsm/liter; intervertebral disc **100** is 300-400 mOsm/liter. Through the difference between osmotic pressures, the connecting filament **126** draws fluid from muscle **193** to hydrate the desiccated nucleus **128** without a pump, as shown in Figure 39.

In-vitro study, the filament **126** was implanted into sheep discs **100** (430 mOsm/liter) and human cadaver discs **100** (300-400 mOsm/liter) of various degenerative levels, Thompson Grade 0-4. The discs **100** with filaments **126** were submerged in saline with blue dye (300 mOsm/liter). Dissection of the discs **100** showed blue saline permeation into the nuclei **128**, confirming fluid flows from low to high osmotic pressure.

Another filament **126** was implanted through a muscle into a sheep disc **100**. The sheep muscle **193** was saturated with iopamidol (contrast agent with blue dye, 545 mOsm/l). The blue iopamidol did not permeate through the filament **126** into the sheep disc **100** (430 mOsm/liter). In fact the dissected disc **100** looked desiccated; fluid within the sheep disc **100** was probably drawn into the muscle **193** infused with 545 mOsm/liter blue iopamidol through the filament **126**. The experiment was repeated with diluted blue iopamidol solution (150 mOsm/liter). The diluted iopamidol solution saturated the muscle **193** and permeated through the filament **126** into the sheep disc **100** visible and traceable from muscle **193** to nucleus **128** under CT. Dissection confirmed permeation of the diluted blue iopamidol into the nucleus **128** of the sheep disc **100**. Again, fluid flows from low to high osmotic pressure through the filament **126**.

Sheep discs were implanted with filaments **126** and submerged in pork blood (about 320 mOsm/liter). Dissection of the discs **100** showed permeation of pork blood through the filament **126** into the nuclei of the sheep discs **100** (430 mOsm/liter).

Gradient of pH is formed in the disc **100** due to shallow diffusion of pH buffer **131** from the capillaries **107** at the endplates **105**. The superior **106A** and inferior **106B** diffusion zones are approximately 0-2 mm from the superior or inferior endplates **105**. The pH in the superior **106A** and inferior **106B** diffusion zones is neutral. Acidity increases at the mid layer of the disc **100**, where chronic deprivation of oxygen, nutrients and pH buffer occurs. Figure 40 shows a longitudinal view of a filament **126** filled disc **100** with calcified layers **108** accumulated over the endplate **105**. The hydrophilic spiraled filament **126** reaches, locates, resides or contacts at least one of the superior **106A** and inferior **106B** diffusion zones, drawing and transporting nutrients/oxygen/pH buffer **131** to neutralize lactic acid **162** and nourish cells in the mid layer of the disc **100**. As a result, lactic acid **162** burn is minimized and back pain is relieved. In essence, the spiraled filament **126** bridges, links or transports fluid between source of oxygen/nutrients/pH

buffer 131 and mid layer of the disc 100 to treat the etiology and symptom of back pain.

Furthermore, oxygen 131 from the muscle 193, superior 106A and inferior 106B diffusion zones converts anaerobic conditions into aerobic within the mid layer of the disc 100 to further reduce production of lactic acid 162 and relieve back pain.

5 The filament 126 can be further identified as internal filament 126 and external filament 126. The filament 126 in the disc 100 can be called the disc shunt 126 to form as internal shunt 126 between superior diffusion zone 106A and inferior diffusion zone 106B. The disc shunt 126 extending from disc 100 to body circulation or muscle 193 is called external shunt 126. The disc shunt 126 is a fluid-transferring or delivery device, inserted into the nucleus 128 of a degenerated
10 disc 100. Due to relaxation and compression of the disc 100 from daily activities of the patient, spiraled internal shunt 126 in the disc 100 facilitates transport of oxygen/nutrients/pH buffer 131 through out the disc 100. During relaxation, oxygen/nutrients/pH buffer 131 from diffusion zones 106A, 106B are absorbed by the internal shunt 126, and oxygen/nutrients/pH buffer 131 in muscle 193 are drawn through the external filament 126. During compression, oxygen/nutrients/pH buffer
15 131 in the shunt 126 are expelled to neutralize lactic acid 162 and fed to disc cells in the mid layer of the disc 100. Essentially, both diffusion zones 106A, 106B are expanded to cover the mid layer or acidic layer of the disc 100. Hence, fluid leaking from the fissure 121 is pH neutral or near pH neutral to alleviate back pain, as shown in Figure 40. Fluid transport or distribution is made possible by soft and pliable spirals of filament 126 as internal disc shunt 126 with hydrophilic and
20 malleable properties, absorbing and delivering nutrients/oxygen/pH buffer 131 within the avascular disc 100. Fluid flow through the shunt 126 is dynamic and continuous with potential to rebuild disc matrix for disc regeneration.

Nutrients/oxygen/pH buffers 131 are diffused from the capillaries 107 at the endplates 105 into the nutrient-poor avascular disc 100, as shown in Figure 40. Diffusion is concentration related;
25 solutes move from high to low concentration, from capillaries 107 into diffusion zones 106A, 106B. Withdrawal of nutrients/oxygen/pH buffers 131 by the internal shunt 126 leads to additional diffusion of nutrients/oxygen/pH buffers 131 from capillaries 107 and vascular buds. The net supply of nutrients/oxygen/pH buffer solutes 131 into the disc 100 will increase with implantation of the internal shunt 126 as shown in Figure 40. Distribution of nutrients/oxygen/pH buffer 131 is
30 expanded by the internal shunt 126 covering or permeating the full-thickness of the intervertebral disc 100 to neutralize lactic acid 162, nourish starving disc cells 277 and rebuild disc matrix to sustain compressive loading of the disc 100.

Depending on severity of the calcified layers 108 covering the capillaries 107 and vascular buds at the endplates 105, the superior 106A and inferior 106B diffusion zones containing

nutrients/oxygen/pH buffer 131 are between 0 and 5 mm from the cartilaginous endplates 105. For degenerated and/or painful discs 100, the superior 106A and inferior 106B diffusion zones are likely between 0 and 2 mm from the superior and inferior endplates 105. Hence, the internal disc shunt 126 should reach at least one, but preferably both superior 106A and inferior 106B diffusion zones, between 0 and 3 mm from both endplates. Repetitive formations and deployments of the coiled or spiraled shunt 126 is used to position, reside, locate, reach or contact at least one diffusion zone 106A or 106B, between 0 and 2 mm from at least one endplate 105 to form the internal disc shunt 126. Distance of the internal shunt 126 from the endplate 105 determines availability or quantity of nutrients/oxygen/pH buffer 131 for supplying the mid layer of the disc 100 to alleviate discogenic pain from lactic acid 162 burn.

Due to avascular nature, intervertebral disc 100 is immuno-isolated. In-vivo sheep study, there was no tissue response within the discs 100 to nylon filament 126 after 1, 3, 6, 12 and 30 months, using H&E histology staining. No fibrotic encapsulation over the nylon filament 126 was observed within the discs 100. Similarly, there was no noticeable inflammatory reaction to the nylon filament 126 in a human pilot clinical study in 1 week, 3, 6, 12 and 24 months. Internal transport of nutrients/oxygen/pH buffer 131 from superior 106A and/or inferior 106B diffusion zones continues without hindrance of fibrotic encapsulation over the filament 126 within the intervertebral disc 100.

The multiple coils or spirals of filament 126 or shunt 126 provide bulk, shimming, filling, cushion, mass, wedging or fortification within the disc 100 to elevate, raise, lift, increase or sustain disc 100 height as indicated by arrows in Figures 40-41. Spirals of filament 126 bulk up the nucleus 128 to elevate or support disc height for sustaining axial compression, stiffen the disc 100 for reducing spinal instability, and/or lift the inferior articular process 143 of the facet joint 129 to reduce facet pain, as shown in Figure 41.

Nutrients/oxygen/pH buffer 131 transported through the filament 126 feed cells 277 to produce biosynthetic molecules, which can be glycosaminoglycans, collagen or disc matrix, as shown in Figure 42. Cells 277 and nutrients/oxygen/pH buffer 131 can be intradiscally injected, in Figure 43, to expedite disc 100 regeneration and pain relief.

Lower lumbar L5-S1 disc 100A and L4-5 disc 100B are shielded by a pair of iliac crests 140, as shown in Figure 44. The straight spinal needle 102 enters superiorly over the iliac crest 140 at an angle, as shown in Figure 45, difficult or even impossible to deliver the shunt 126 into the nucleus 128 of the disc 100.

Figure 46 shows an elastically curved cannula 230 leading the filament needle 101 into the nucleus 128 of a degenerated disc 100. The elastically curved cannula 230 is resiliently straightened by sliding over the dilator 220 into the annulus 378 of the disc 100. Then, the dilator

220 is partially withdrawn while holding the cannula stationary. The cannula 230 resumes the curvature and advances into the loosely packed nucleus 128. After confirming distal location of the curved cannula 230 by fluoroscope, the filament needle 101 in Figure 22 replaces the dilator 220 for filament 126 spiraling, similar to Figures 25, 27-39. The cannula 230 and needle 101 can be made with nickel titanium alloy for shaped memory and super elasticity.

In summary, insertion of the spiraled filament 126 into disc 100 increases nutrients/oxygen/pH buffer 131 from muscle 193 and diffusion zones 106A and/or 106B to reduce lactic acid 162 burn and feed cells. The spiraled filament 126 also adds bulk and cushion to reduce spinal instability and facet pain.

The shunt 126 for disc 100 repair is hydrophilic with measurable characteristics under ambient temperature and pressure for transporting and retaining fluid to relieve pain and/or regenerate the degenerated disc 100. After saturation in water, the shunt 126 gains weight between 10% and 500% by absorbing water within the matrix of the shunt 126. A healthy human disc 100 contains 80% water. The preferred water absorbency after water saturation is between 30% and 120%. The shunt 126 can have pore sizes between 1 nano-meter and 500 micro-meters, serving as water retaining pockets or water transporting channels. Pores 124 of the shunt 126 also function as scaffolding or housing for cell 277 attachment and cellular proliferation, as shown in Figure 42. Water contact angle on the shunt 126 is between 0 and 60 degrees. The preferred water contact angle of the shunt 126 is between 0 and 30 degrees. Height of capillary action for drawing saline up the shunt 126 is between 0.5 and 120 cm. The preferred height of capillary action of drawing saline is between 1 and 60 cm. Height of capillary action for drawing pork blood up the shunt 126 is between 0.5 and 50 cm. The preferred height of capillary action for drawing pork blood up the shunt 126 is between 1 cm and 25 cm. Saline siphoning transport rate through the shunt 126 is between 0.1 and 10 cc per 8 hours in a humidity chamber. Human lumbar disc 100 loses between about 0.5 and 1.5 cc fluid per day due to compression. The saline siphoning transport rate through the shunt 126 is preferred between 0.5 and 5 cc per 8 hours in a humidity chamber. Pork blood siphoning transport rate through the shunt 126 is between 0.1 and 10 cc per 8 hours in a humidity chamber. The pork blood siphoning transport rate through the shunt 126 is preferred between 0.5 and 3 cc per 8 hours in a humidity chamber.

The shunt 126 used in the sheep and human clinical studies have the following physical properties under ambient temperature and pressure: (1) weight gain 80% after water saturation, (2) water contact angle zero degree, (3) height of capillary action 11 cm with pork blood, 40 cm with saline with blue dye, and (4) rate of siphoning pork blood 1.656 +/- 0.013 cc per 8 hours in a humidity chamber.

Average lactic acid concentration in painful lumbar disc **100** is about 14.5 mM, 15 cc or less in volume (Diamant B, Karlsson J, Nachemson A: Correlation between lactate levels and pH of patients with lumbar rizopathies. *Experientia*, 24, 1195-1196, 1968). An in-vitro study was conducted to show instant lactic acid neutralization by blood plasma. The spiraled shunt **126** was
5 formed within, and then extracted from a fresh portion of beef. Blood plasma absorbed in the spiraled shunt **126** instantly neutralized 42% of the 14.5 mM, 15 cc of lactic acid solution, measurable by a pH meter.

Approximately 85% back pain patients show no nerve impingement under MRI or CT. A patient without nerve impingement suffered chronic back pain with visual analog score 8-9 out of
10 (most severe), and leg pain with visual analog score 8. Five days after implantation of the shunt **126**, the visual analog score dropped to 2.5 for her back pain, but the visual analog score persisted at 8 for leg pain. In the 5.5-month follow-up, the visual analog score dropped to 2.0 for her back pain, and visual analog score dropped from 8 to zero for leg pain. Quick back pain relief may be contributed to instant lactic acid **162** neutralization by blood plasma of the patient to relieve acid
15 burning of the adjacent sensory nerves **118**. Leg pain may be caused by acid scaring of the spinal nerve **194** and chemical radiculitis, which takes time to heal and relieve the pain. In human clinical study, the outer diameters of the needle **101** and cannula **230** are only 1.83 and 2.41 mm respectively. The outer diameter of the shunt **126** is 0.55-0.77 mm.

Urinary incontinence is common among women, especially after multiple pregnancies and
20 vaginal deliveries. Major urethral sphincteric action is operated by smooth muscle **532** at the bladder neck **519**. Figure 47 shows a normal and narrow bladder neck **519** of a woman with urinary control. The smooth muscle **532** controls opening and closure of the urethral lumen **517** lined with mucosa **533**. During pregnancy, the fetus presses against the bladder **518** and the urethra **516** for months. The downward compression flattens and widens the smooth muscle **532** and
25 urethral lumen **517** of the bladder neck **519**, as shown in Figure 48. A widened lumen **517** at the bladder neck **519** is beyond the range of sphincteric closure of the smooth muscle **532** for coaptation of mucosa **533** in urethral lumen **517**. As a result, stress urinary incontinence occurs during increased abdominal pressure from coughing, sneezing, laughing or even standing. In surgical intervention for stress incontinence, the vagina **520** is pulled and fastened to ligaments
30 anteriorly, to support and push forward the posterior wall **524** of the bladder neck **519**, narrowing the urethral lumen **517** for coaptation of urethral mucosa **533** during sphincteric action.

A needle **460** within a cone-head dilator **220** in Figure 49 is inserted into urethral smooth muscle **532** of the bladder neck **519** under cystoscopic or ultrasound guidance. An echogenic gel can be injected through the needle **460** to confirm location of the needle **460** tip within the urethral

muscle 532. The cone-head dilator 220 slides over the needle 460 into the urethral muscle 532. The needle 460 is withdrawn. Addition echogenic gel can be injected through the removable luer lock 530 to ensure location of the dilator 220 tip within the urethral muscle 532. The luer lock 530 is removed to prepare for repair. Similar to disc 100 repair, a cannula 230 slides over the dilator 220 into the urethral muscle 532. The dilator 220 is replaced with a filament needle 101 loaded with filament 126 within the cannula 230, as shown in Figure 23. Spiraled filament 126 in Figure 50 is created by withdrawal, advancement, rotation and pushing of the filament needle 101, as shown in Figures 25, 27 and 29-35. Figure 51 shows an axial or cross-sectional view of the urethra 516 with a widened urethral lumen 517 and initial spiraled filament 126 formed within the smooth muscle 532 of the posterior wall 524 of the urethra 516. Figure 52 shows bulking, enlargement or filling of the urethral smooth muscle 532 with spirals of filament 126 at the posterior wall 524 to encroach or extend into the space of urethral lumen 517, resulting in size reduction of the urethral lumen 517 to facilitate coaptation of urethral mucosa 533 during sphincteric action of the urethral muscle 532. Multiple locations of spiraled filaments 126 can be implanted in urethral muscle 532 to further narrowing the urethral lumen 517, as shown in Figure 53. For bulking of urethra 516, no filament 126 should be extended into the lumen 517 to avoid infiltration of bacteria into the smooth urethral muscle 532. When closure of urethral lumen 517 with bulking of the filament 126 seems adequate through the cystoscope, the filament 126 between the proximal end 501 of needle handle 130 and bobbin 152 in Figure 26 is cut, and spiraling of the filament 126 continues with withdrawal, re-advancement and rotation of the needle 101 to completely spiral the remaining filament 126 in needle 101 into urethral muscle 532. The amount of implanted filament 126 is selectable, controllable, limitable or regulateable by the physician. The filament 126 can be a nylon, polypropylene or biodegradable mono-filament suture with some stiffness or shape memory. Coiling or spiraling of the shape memory filament 126 expands within tissue to provide elastic bulking or expansion to enhance the backboard support and sphincteric action of the urethra 516. Figure 54 shows narrowing of the bladder neck 519 by spirals of filament 126 cushioning, bulking or supporting tissue between the vagina 520 and urethral lumen 517 to relieve stress urinary incontinence and regain sphincteric control.

Gel-like bulking agent has been injected into fecal sphincteric muscle to treat fecal incontinence, but feces can be substantially firm and large to flatten and nullify bulking of the gel-like agent. On the other hand, the elastic and shape-memory spirals of filament 126 are interconnected to prevent flattening, migration or dislocation, to maintain fecal sphincteric bulking and control, similar to urethral bulking in Figures 49-54.

The filament 126 can be a suture 126. The suture 126 spiraling device can also be used to spiral and pack suture 126 under skin to fill indentations from acne scar or cosmetic defect. The spirals of suture 126 at the distal end can also be used as a suture anchor deep within tissue. The proximal end of the suture 126 can be threaded with a tissue repairing needle for tissue fastening through micro-invasive procedure, such as face lift and other suture repair.

The filament 126 or strands 104 of the filament 126 can expand or swell during hydration in body fluid. The swelled filament 126 adds size or mass within tissue to enhance bulking or efficacy of the spirals of filament 126. The filament 126 can also be coated with a hydrophilic or swelling agent, such as polyethylene glycol, collagen, hyaluronic acid or other, for expansion.

The dilator 220 in Figure 49 can be substituted with the cannula 230 connecting to a luer lock 531 at the proximal end for injection of echogenic liquid. After needle 460 puncturing into urethral muscle 532, the cannula 230 is advanced by sliding over the needle 460 into urethral muscle 532. The needle 460 is withdrawn. Echogenic gel or liquid can be injected through the cannula 230 to confirm distal location of the cannula 230 in urethral muscle 532. Since the cannula 230 has no tissue puncturing capability, injection of echogenic gel for location confirmation may not be necessary. The filament needle 101 is inserted into the cannula 230 for spiraling filament 126.

A knot 463 can be tied at the distal portion 126A of the filament 126 to prevent retrieval of the filament 126 into the lumen 269 of the needle 101, as shown in Figure 55. The knot 463 also facilitates catching or hooking by the snagging point 231 of the cannula 230, further improved from Figure 25. The U-strand of the filament 126 can be divided into distal portion 126A and proximal portions 126B and 126C. A proximal portion 126B of the filament 126 can drape outside, while another proximal portion 126C of the filament 126 can be inserted in the needle 101 as shown in Figure 56. A single stranded filament 126 can be inserted into the needle 101, as shown in Figure 57. The single stranded filament 126 can also be snagged by the snagging points 231 of the cannula 230 and rotated into spiraled filament 126. A knot 463 is tied at the distal portion 126A to prevent retrieval of the single stranded filament 126 into the lumen 269 of the needle 101, as shown in Figure 57. An anchor 462 can be attached at the distal end of the filament 126, as shown in Figure 58. The anchor 462 can be made with a biodegradable material or tablet of a pH buffer, nutrients or medication. The anchor 462 can also be a toggle 462, a protrusion 462 or latch 462.

The filament gripper 111 can be a cross bar 111 as shown in Figure 59. The filament 126 loops over the cross bar 111, as shown in Figure 60, for insertion into the cannula 230. The cross bar 111 can be a longitudinal plane, dividing the cylindrical lumen 269 of the needle 101 into two semi-cylindrical lumens 269, as shown in Figure 61. The cross bar 111 or cross plane 111 can tightly pack the spiraled filament 126 into tissue. The cross bar 111 can be extended by two extension bars

112 as shown in Figure 62. The filament 126 loops over the extended cross bar 111, as shown in Figure 63. The extended cross bar 111 can also be extended into a cross plane 111, dividing into two semi-cylindrical lumens 269. Filament grippers 111 can be cross stubs 111 as shown in Figure 64. The cross stubs 111 can also be two longitudinal stubs length-wise through the lumen 269, as shown in Figure 65.

Loading filament 126 from the bobbin 152 into proximal lumen 269 of the needle 101 is driven by holding the distal end 126A of the filament 126 and withdrawing the needle 101, as shown in Figures 25, 26 and 32. A filament advancer 526 can be manually used or motorized to advance the filament 126 without needle 101 withdrawal. The filament advancer 526 contains a stem 527
10 attaching resiliently collapsible barbs 528 pointing distally, as shown in Figure 66. The resiliently collapsible barbs 528 have a closed and open positions. In the closed position, the distal ends of the resiliently collapsible barbs 528 approximate the stem 527, solid lines of Figure 67. In the open position, the distal ends of the resiliently collapsible barbs 528 depart or move outward from the stem 527, in dashed lines of Figure 67. In operation within tissue, distal end of the filament
15 advancer 526 and the resiliently collapsible barbs 528 are concealed and operated within the lumen 269 of the needle 101. The needle lumen 269 can be non-circular shape. Cross-section of the lumen 269 can contain two connecting circular lumens 269, one for housing the filament advancer 526 and the other for housing the filament 126, similar configuration as Figure 70A. The opening between the two connecting circular lumens 269 allows the collapsible barbs 528 to extend and
20 engage with the filament 126. During distal movement of the filament advancer 526, the collapsible barbs 528 are in the open positions to grip, puncture, insert, hold, grab, attach, engage, hook or latch on the filament 126, moving the filament 126 distally. During proximal movement of the filament advancer 526, the resiliently collapsible barbs 528 release the engagement with the filament 126 and retract, retrieve or collapse into the closed positions to approximating the stem
25 527. The collapsible barbs 528 are elastic or flexible to grip and release the filament 126 during cyclical distal-proximal movement of the stem 527 or filament advancer 526. The collapsible barbs 528 are spaced out along the stem 527. The axial or vertical view of the filament advancer 526 in Figure 68 shows orientations of the resiliently collapsible barb 528 approximately 120 degrees apart. Figure 69 shows the collapsible barbs 528 approximately 90 degrees apart. The filament
30 126 can also be advanced by a manually driven or motorized rotational auger 526 as filament advancer 526 within the lumen 269 of the needle 101, as shown in Figure 70. The auger 526 contains a stem 527 and screw-like or helical thread 528 to engage, convey or propel the filament 126 out the needle 101. In operation within tissue, distal end of the filament advancer 526 and the screw-like thread 528 are concealed and operated within the lumen 269 of the needle 101. The

needle lumen 269 can also be non-circular shaped. Cross-section of the lumen 269 can contain two connecting circular lumens 269, one for housing the rotational auger 526 and the other for housing the filament 126, as shown in Figure 70A. The opening between the two connecting circular lumens 269 allows the screw-like thread 528 to extend and engage with the filament 126. The motorized speed of the resiliently collapsible barbs 528 and screw-like thread 528 can be controlled by a foot pedal. During high torque, a torque sensor initiates reduction or stopping of the motor. The motorized filament advancer 526 saves surgical time and allows physician to concentrate on needle 101 rotation and pushing to pack filament 126 for repairing tissue. The resiliently collapsible barbs 528 and the screw-like thread 528 can be called the filament engager 528 of the filament advancer 526.

Figure 71 shows a cannula 230 with multiple snagging points 231 to facilitate holding on the filament 126. Figure 72 shows a large window 110 for filament 126 to protrude during needle re-advancement to prevent jamming. The window 110 is open to a snagging point 231. Figure 73 shows elastically curved or inward bending of snagging points 231 of the cannula 230 to facilitate trapping or hooking of the extended filament 126 in Figures 22-25, 27-28, 32, 46, 50, 55-58, 60, 63 from the needle 101. The cannula 230 with elastically curved snagging points 231 can be made with nickel-titanium alloy or nitinol. During advancement of the filament needle 101, the elastically curved snagging points 231 resiliently open or straighten to allow passing of the needle 101 and the extended filament 126. The elastically curved snagging points 231 provide inward grip to trap, catch or hook the extended filament 126, similar to the result of Figures 25 and 32. Other elastically curved snagging points 231 of the cannula 230 are shown in Figure 74. The snagging points 231 can be elastically curved walls or gates 231 at the distal end of the cannula 230 in Figure 75 to hook or trap the extended filament 126 from the needle 101. Figure 76 shows a cross-sectional view of the inward bending or elastically curved gates 231, restricting the distal lumen 268 of the cannula 230. The elastic snagging point 231 can be called a spring biased arm 231.

Flexible filament 126 can be made or formed by fabric making techniques, such as braiding or twisting strands 104 as shown in Figure 77. For twisting, minimum number of strands 104 is two. For braiding, minimum number of strands 104 is three, as shown in Figure 77. Braiding or intertwining three or more strands 104 provides excellent flexibility, strength and porosity of the filament 126. The snagging point 231 of cannula 230 and gripper 111 of needle 101 can catch, snag or engage the filament 126 well. The flexible filament 126 can also be woven, as shown in Figure 78. Weaving is interlacing the strands 104 over and under each other, generally oriented at 90 degree angles. Half of the strands 104 from weaving can be oriented length-wise along the linear filament 126, to expedite capillarity or fluid flow from the muscle 193 or diffusion zones

106A, 106B into the degenerated disc 100. The flexible filament 126 can be knitted, as shown in Figure 79. Knitting is a construction made by interlocking loops of one or more strands 104. A knitted filament 126 may have the greatest elastic expansion and compression capability, delivering the most fluid transport or exchange between disc 100 and body circulation during compression and relaxation of the disc 100. In addition, the knitted filament 126 in coils, spirals or reels may have the highest porosity to enhance fluid absorbency, creating a reservoir of nutrients/oxygen/pH buffer 131 for dispersing into various parts of the avascular disc 100, as shown in Figures 39-40.

Furthermore, the coiled or spiraled filament 126 with knitted strands 104 provides an elastic cushion within the disc 100 to reduce loading and pain in the facet joints 129. The knitted filament 126 may be an excellent matrix or scaffolding for cell 277 attachment and proliferation. The knitted filament 126 may also provide highly expandable spirals for bulking sphincters to regain urinary or fecal control. The filament 126 can be made with non-woven strands 104. The term non-woven is used in fabric industry to include all other techniques, such as carded/needle-punched, spun bonded, melt blown or other. Non-woven filament 126 can provide large surface area as scaffolding for cell 277 growth and proliferation. Combinations of fabric making techniques for the filament 126 can also be used with the needle 101 and cannula 230.

Material and/or orientation of the filaments 126 can affect (1) flow rate, (2) tensile strength, (3) annular sealing, (4) porosity, (5) fluid absorbency, (6) snagging ability, (7) elasticity, (8) selectivity of solute transport, (9) scaffold attachment of cells, (10) flexibility, (11) durability, (12) sterilization technique, (13) fibrotic formation, (14) biocompatibility, and/or (15) bulking. A filament 126 is cut at a slanted angle, showing a cross-section of a filament 126; the strands 104 are slanted or diagonally oriented to the length-wise filament 126, as shown in Figure 80. Figure 81 shows cross-sections of strands 104 parallel to the filament 126, covered by a wrapper, sheath or cover 127. The parallel-oriented strands 104 and wrapper 127 can be manufactured by extrusion. The strands 104 can also be micro tubes, as shown in Figure 82, parallel to the filament 126. A wrapper 127 is used to cover, retain, enclose or house the micro strands 104 to form a filament 126. An individual micro tubular strand 104 is capable of having capillary action, drawing nutrients/oxygen/pH buffer 131 through the filament 126 into the disc 100.

The strands 104 are preferred to be made with biocompatible and hydrophilic material, absorbing, retaining or drawing fluid with nutrients/oxygen/pH buffer solutes 131 from a tissue with low osmolarity to mid layer of the desiccated disc 100 with high osmolarity. The filament 126 can be a suture, approved for human implant. Instead of fastening tissue, the suture is used as the filament 126, transporting fluid from low to high osmolarity to alleviate back pain.

The filament 126 can be made with a hydrophilic sponge or foam with pores 124, as shown in Figure 83, to transport and retain fluid in the disc 100. The pores 124 can be open, connecting to other pores 124. The pores 124 can also be closed, not connecting to other pores 124 to retain fluid and cells 277. The filament 126 can be solid, pore-less or dense, as shown in cross section in Figure 84. The solid filament 126 is similar to a mono-filament suture 126. The mono-filament 126 is relatively stiff or contains a shape-memory to resist a spiral or coiled configuration. The spiraled mono-filament 126 elastically expands within tissue, which is suitable for elastic bulking of sphincters to treat urinary or fecal incontinence.

Disc cells 277 isolated from advanced degenerated human discs 100 are still capable of producing collagen and glycosaminoglycans in tissue culture with abundant supply of nutrients in proper pH. (Gruber H.E., Leslie K., Ingram J., Hoelscher G., Norton H.J., Hanley E.N. Jr.: Colony formation and matrix production by human anulus cells: modulation in three-dimensional culture, Spine, July 1, 29(13), E267-274, 2004. Johnstone B, Bayliss MT: The large proteoglycans of the human intervertebral disc, Changes in their biosynthesis and structure with age, topography, and pathology, Spine, Mar 15;20(6):674-84, 1995.) Furthermore, stem cells have recently been found in degenerated discs. (Risbud MV, Gattapalli A, Tsai TT, Lee JY, Danielson KG, Vaccaro AG, Albert TJ, Garzit Z, Garzit D, Shapiro IM: Evidence for skeletal progenitor cells in the degenerate human intervertebral disc, Spine, Nov 1;32(23), 2537-2544, 2007.) Nutrient 131 deficiency and acidic pH may hinder disc 100 repair in-vivo.

The filament 126 or shunt 126 can be scaffolds and spigots for supplying nutrients/oxygen/pH buffering solute 131 for cell 277 attachment, as shown in Figure 42. With a continual or renewable supply of nutrients/oxygen/pH buffer solutes 131, disc cells 277 resume making biosynthetic products 160, such as the water-retaining glycosaminoglycans and collagen, the major components of the nucleus 128 and annulus 378. In sheep study, newly formed glycosaminoglycans on nylon strands 104 of the shunt 126 after 3 months can be seen using Safranin histological staining.

The rate of sulfate incorporation for biosynthesizing glycosaminoglycans is pH sensitive. The maximum rate of sulfate incorporation is with pH 7.2-6.9. The rate of sulfate incorporation drops about 32-40% in acidic pH within the disc [Ohshima H, Urban JP: The effect of lactate and pH on proteoglycan and protein synthesis rates in the intervertebral disc. Spine, Sep;17(9), 1079-82, 1992]. Hence, pH normalization with pH buffer solute 131 through the shunt 126 will likely increase production of the water-retaining glycosaminoglycans and swelling pressure of the shunted disc 100.

With a continual supply of nutrients 131, newly formed biosynthetic products 160 increase osmolarity within the shunted disc 100 and enhance inward fluid flow, as shown in Figures 40 and

42. The increased fluid flow comes through (1) the external shunt 126, (2) blood capillaries 107 through the endplates 105, and/or (3) annulus 378. The fluid is also retained by the newly formed water-retaining glycosaminoglycans 160. As a result, the swelling pressure of the shunted disc 100 increases. Segmental or spinal instability is reduced. Muscle tension and ache from guarding the spinal instability decrease. Load and pain of the facet joints 129 decrease. Lactic acid 162 is further neutralized by inflow of nutrients/oxygen/pH buffering solute 131 to reduce or alleviate acid burn. Disc 100 height is elevated, raised or increased as depicted by arrows in Figures 40-41. Implantation of the shunt 126 enables the degenerated disc 100 to be repaired.

Furthermore, adenosine triphosphate, ATP, is the high-energy compound essential for driving or energizing biochemical reactions, including the biosynthesis of the water retaining glycosaminoglycans for sustaining compressive loads on the disc 100. Under anaerobic conditions, metabolism of each glucose molecule produces only two ATP and two lactic acids 162, which irritate adjacent nerves 118. When oxygen 131 permeates through the internal and/or external shunt 126, thirty-six ATP can be produced from each glucose molecule through glycolysis, citric acid cycle and electron transport chain under aerobic conditions to energize disc regeneration and alleviate back pain.

High concentration of nutrients 131 can also be injected into the internal and/or external shunted disc 100 to instantly create high osmolarity, as shown in Figure 43. High osmolarity promotes fluid inflow into the shunted disc 100. However, glucose or sugars injection can produce additional lactic acid 162, causing more pain. Sulfate and amino acids can be injected in high concentration to boost osmolarity and production of glycosaminoglycans and collagen, as the biosynthetic product 160 in Figure 42. Magnesium sulfate, potassium sulfate, or sodium sulfate can be the injectable with high water solubility and is essential for biosynthesis of glycosaminoglycans in the nucleus pulposus 128. Proline and glycine can also be injectables with high water solubility and are essential nutrients 131 for biosynthesis of collagen in the annulus 378.

Analgesics, anti-depressant, steroid, NSAID, antibiotics, anti-inflammatory drugs, alkaline agent or other drugs can also be injected into the shunted disc 100 to instantaneously reduce pain.

Autograft disc cells 277 from a healthy disc 100 of the patient can be transplanted into the degenerated and shunted disc 100 to promote disc regeneration and production of biosynthetic product 160, as shown in Figure 43.

The avascular disc 100 is well sealed and immuno-isolated. Even small ions, such as sulfate, and small molecules, such as proline, are greatly limited from diffusing into the nucleus pulposus 128. The well sealed disc 100 may be able to encapsulate donor cells 277 from a disc 100 of another person, cadaver or even animal without triggering an immune response, and probably not

needing anti-rejection drug. For disc **100** regeneration, the donor cells **277** can also be stem cells **277**, notochord **277** or chondrocytes **277**. The filament **126** or shunt **126** is permeable to nutrients/oxygen/pH buffering solute **131** but impermeable to cells and/or cytokines responsible for triggering an immune reaction. The cells of the immune system include giant cells, macrophages, mononuclear phagocytes, T-cells, B-cells, lymphocytes, Null cells, K cells, NK cells and/or mask cells. The cytokines may also include immunoglobulins, IgM, IgD, IgG, IgE, other antibodies, interleukins, lymphokines, monokines or interferons.

The molecular weights of nutrients **131** and lactic acid **162** are much smaller than the immuno-responsive cells and cytokines. The transport selectivity can be regulated or limited by the size of the pores or channels within the semi-permeable shunt **126**. The upper molecular weight cut-off of the shunt **126** can be 100,000 or lower to allow the passage of nutrients and waste but exclude the immuno-responsive cells and cytokines. The semi-permeable shunt **126** may also contain ionic or affinity surfaces to attract nutrients **131** and waste, including lactic acid **162**. The surfaces of the semi-permeable shunt **126** can be made, coated or modified to repel, exclude or reject immuno-responsive components.

In recent years, cell transplants from cadavers or live donors have been successful in providing therapeutic benefits. For example, islet cells from a donor pancreas are injected into a type I diabetic patient's portal vein, leading into the liver. The islets begin to function as they normally do in the pancreas by producing insulin to regulate blood sugar. However, to keep the donor cells alive, the diabetic patient requires a lifetime supply of anti-rejection medication, such as cyclosporin A. In addition to the cost of anti-rejection medication, the side effects of these immuno-suppressive drugs may include cancer. The benefit of cell transplant may not outweigh the potential side effects.

The shunted intervertebral disc **100** can be used as a semi-permeable capsule to encapsulate the injected therapeutic donor cells **277** or agent, as shown in Figures 43, to evade the immune response; hence no life-long immuno-suppressive drug would be required. A variety of donor cells **277** or agent can be harvested and/or cultured from the pituitary gland (anterior, intermediate lobe or posterior), hypothalamus, adrenal gland, adrenal medulla, fat cells, thyroid, parathyroid, pancreas, testes, ovary, pineal gland, adrenal cortex, liver, renal cortex, kidney, thalamus, parathyroid gland, ovary, corpus luteum, placenta, small intestine, skin cells, stem cells, gene therapy, tissue engineering, cell culture, other gland or tissue. The donor cells **277** are immunoisolated within the shunted discs **100**, the largest avascular organs in the body, maintained by nutrients/oxygen/pH buffer **131** and waste transport through the shunt **126** or fissure **121**. The donor cells **277** can be from human, animal or cell culture. When disc pressure is low during sleep

or supine position, nutrients/oxygen/pH buffering solutes **131** are supplied through the shunt **126** to the donor cells **277**. During waking hours while the pressure within the disc **100** is high, biosynthesized products **160** by these donor cells **277** are expelled through the shunt **126** into the muscle **193** or through fissures **121** into bodily circulation and target sites.

5 The biosynthesized product **160** made by the donor cells **277** can be adrenaline, adrenocorticotrophic hormone, aldosterone, androgens, angiotensinogen (angiotensin I and II), antidiuretic hormone, atrial-natriuretic peptide, calcitonin, calciferol, cholecalciferol, calcitriol, cholecystokinin, corticotropin-releasing hormone, cortisol, dehydroepiandrosterone, dopamine, endorphin, enkephalin, ergocalciferol, erythropoietin, follicle stimulating hormone, γ -
 10 aminobutyrate, gastrin, ghrelin, glucagon, glucocorticoids, gonadotropin-releasing hormone, growth hormone-releasing hormone, human chorionic gonadotrophin, human growth hormone, insulin, insulin-like growth factor, leptin, lipotropin, luteinizing hormone, melanocyte-stimulating hormone, melatonin, mineralocorticoids, neuropeptide Y, neurotransmitter, noradrenaline, oestrogens, oxytocin, parathyroid hormone, peptide, pregnenolone, progesterone, prolactin, pro-
 15 opiomelanocortin, PYY-336, renin, secretin, somatostatin, testosterone, thrombopoietin, thyroid-stimulating hormone, thyrotropin-releasing hormone, thyroxine, triiodothyronine, trophic hormone, serotonin, vasopressin, or other therapeutic products. These biosynthetic products **160** have low molecular weights and are able to be transported through the shunt **126** and/or fissures **121**, while the donor cells **277** are trapped within the disc **100**.

20 The biosynthesized products **160** (hormones, peptides, neurotransmitter, enzymes, catalysis or substrates) generated within the shunted disc **100** may be able to regulate bodily functions including blood pressure, energy, neuro-activity, metabolism, and activation and suppression of gland activities. Some hormones and enzymes govern, influence or control eating habits and utilization of fat or carbohydrates. These hormones or enzymes may provide weight loss or gain
 25 benefits. Producing neurotransmitters, such as dopamine, adrenaline, noradrenaline, serotonin or γ -aminobutyrate, from the donor cells **277** within the shunted disc **100** can treat depression, Parkinson's disease, learning disability, memory loss, attention deficit, behavioral problems, mental or neuro-related diseases.

30 Release of the biosynthesized products **160** by the donor cells **277** within the shunted disc **100** is synchronized with body activity. During activities of daily living, the pressure within the shunted disc **100** is usually high to expel the biosynthesized products **160** by the donor cells **277** into circulation to meet the demands of the body. In the supine position, pressure within the shunted disc **100** is low; fluid inflow **161** through the shunt **126** is favorable, bringing nutrients/oxygen/pH buffer **131** into the disc **100** to nourish the cells **277**. As an example, islets of

Langerhans from a donor's pancreas can be implanted or injected into the shunted disc **100**. In supine position during sleeping, glucose enters into the shunted disc **100** to induce production of insulin from the implanted islets of Langerhans. During waking hours when disc pressure is high, insulin is expelled through the shunts **126** or fissure **121** into circulation to regulate concentration of glucose in the body. At night, the insulin released from the shunted disc **100** is minimal to prevent hypoglycemia. In essence, biosynthesized products **160** by the donor cells **277** are released concurrent with physical activity to meet the demands of the body.

Donor cells **277** can also be seeded on the shunt **126** or injected days, weeks, months or even years after implanting the disc shunts **126**, to ensure favorable biological conditions, including pH, electrolytic balance and nutrients and oxygen **131**, for cell **277** survival and proliferation in the shunted disc **100**.

In the United States, average age of patients undergoing back surgery is about 40-45 years old. The disc shunt **126** is preferred to be made with permanent material to provide long-lasting pain relief. A wide range of non-degradable materials can be used to fabricate the shunt **126**. Polymers, such as nylon, polytetrafluoroethylene, polypropylene, polyethylene, polyamide, polyester, polyurethane, silicon, poly-ether-ether-ketone, acetal resin, polysulfone, polycarbonate, silk, cotton, or linen are possible candidates. Fiberglass can also be a part of the shunt strands **104**, to provide capillarity for transporting nutrients **131** and waste.

Especially for investigative purposes, biodegradable shunts **126** may provide evidence within weeks or months. Since the disc shunt **126** degrades within months, any unforeseen adverse outcome would be dissipated. If the investigative-degradable shunt **126** shows promise, permanent shunt **126** can then be implanted to provide continuous benefits. The biodegradable shunt **126** can be made with polylactate, polyglycolic, poly-lactide-co-glycolide, polycaprolactone, trimethylene carbonate, silk, catgut, collagen, poly-p-dioxanone or combinations of these materials. Other degradable polymers, such as polydioxanone, polyanhydride, trimethylene carbonate, poly-beta-hydroxybutyrate, polyhydroxyvalerate, poly-gama-ethyl-glutamate, poly-DTH-iminocarbonate, poly-bisphenol-A-iminocarbonate, poly-ortho-ester, polycyanoacrylate or polyphosphazene can also be used.

The filament needle **101** and cannula **230** can be made with stainless steel, nickel-titanium alloy or other metal or alloy. The needle **101** and cannula **230** can be coated with lubricant, tissue sealant, analgesic, antibiotic, radiopaque, magnetic and/or echogenic agents.

The disc shunt **126** can be used as a drug delivery device, delivering oral, intravenous or injectable drugs into the avascular or nearly impenetrable disc **100** to treat infection, inflammation, pain, tumor or other disease. Drugs can be injected into the muscle **193** to be drawn into the

shunted disc 100. Discitis is a painful infection or inflammatory lesion in the intervertebral disc 100 of adults and children (Wenger DR, Bobechko WP, Gilday DL: The spectrum of intervertebral disc-space infection in children, J. Bone Joint Surg. Am., 60:100-108, 1978. Shibayama M, Nagahara M, Kawase G, Fujiwara K, Kawaguchi Y, Mizutani J: New Needle Biopsy Technique for Lumbar Pyogenic Spondylodiscitis, Spine, 1 November, Vol. 35 - Issue 23, E1347-E1349, 2010). Due to the avascular nature of the disc 100, oral or intravenous drugs cannot easily reach the bacteria or inflammation within the disc 100. Therefore, discitis is generally difficult to treat. However, the disc shunt 126 can be used as a drug-delivery device. The disc shunt 126 draws the systemic drugs from muscles 193 into the sealed, avascular disc 100. In addition, antibiotics, anti-inflammatory drugs, anesthetics or other drugs can be injected into the muscle 193 near the disc shunt 126 to increase drug concentration within the disc 100 to treat discitis or pain. Injection near the shunt 126 is called peri-shunt injection.

Staphylococcus aureus is the most common bacteria found in discitis. The shunt 126 can be loaded or coated with an antibiotic, such as nafcillin, cefazolin, dicloxacilin, clindamycin, bactrim, penicillin, mupirocin (bactroban), vancomycin, linezolid, rifampin, sulfamethoxazole-trimethoprim or other, to treat staphylococcus aureus infection. *Corynebacterium* is also found in discitis. The shunt 126 can be loaded or coated with an antibiotic, such as erythromycin, vancomycin, eifampin, penicillin or tetracycline, to treat corynebacterium infection. Other antibiotics, such as cefdinir, metronidazole, tinidazole, cephmandole, latamoxef, cefoperazone, cefmenoxime, furazolidone or other, can also be used to coat the shunt 126.

Inflammation in the disc 100 can cause excruciating pain. MRI can show inflammation at the endplates 105, and distinguish inflammatory classification as Modic I, II or III. The disc shunt 126 can be coated or loaded with nonsteroidal anti-inflammatory drugs/analgesics (NSAID), such as aspirin, diflunisal, salsalate, ibuprofen, naproxen, fenoprofen, ketoprofen, flurbiprofen, oxaprozin, indomethacin, sulindac, etodolac, ketorolac, diclofenac, nabumetone, piroxicam, meloxicam, tenoxicam, droxicam, lornoxicam, isoxicam, mefenamic acid, meclofenamic acid, flufenamic acid, tolfenamic acid, celecoxib, rofecoxib, valdecoxib, parecoxib, lumiracoxib, etoricoxib, firocoxib, nimesulide, licofelone or other NSAID, to treat inflammation in the disc 100 for pain relief.

The disc shunt 126 can also be coated or loaded with steroidal anti-inflammatory drugs/analgesics, such as betamethasone, budesonide, cortisone, dexamethasone, hydrocortisone, methylprednisolone, prednisolone, prednisone, triamcinolone or other steroid, to treat inflammation in the disc 100 for pain relief.

The shunt 126 can be loaded or coated with anesthetics, such as procaine, amethocaine, cocaine, lidocaine, prilocaine, bupivacaine, levobupivacaine, ropivacaine, mepivacaine, dibucaine,

methohexital, thiopental, diazepam, lorazepam, midazolam, etomidate, ketamine, propofol, alfentanil, fentanyl, remifentanyl, sufentanyl, buprenorphine, butorphanol, diamorphine, hydromorphone, levorphanol, meperidine, methadone, morphine, nalbuphine, oxycodone, oxymorphone, pentazocine or other anesthetic, to provide instant pain relief.

- 5 The shunt 126 can be loaded or coated with a muscle relaxant, such as succinylcholine, decamethonium, mivacurium, rapacurium, atracurium, cisatracurium, rocuronium, vecuronium, alcuronium, doxacurium, gallamine, metocurine, pancuronium, pipecurium, tubocurarine or other relaxant, to relieve muscle tension and ache.

- 10 The shunt 126 can be loaded or coated with buffering agents, such as sodium carbonate, sodium bicarbonate, potassium carbonate, potassium bicarbonate, magnesium carbonate, calcium carbonate, barium carbonate, potassium phosphate, sodium phosphate or other buffering agent, to neutralize lactic acid 162 and spontaneously alleviate pain caused by acid irritation or burn.

- 15 The shunt 126 can be loaded or coated with alkaline agents, such as magnesium oxide, magnesium hydroxide, sodium hydroxide, potassium hydroxide, barium hydroxide, cesium hydroxide, strontium hydroxide, calcium hydroxide, lithium hydroxide, rubidium hydroxide, neutral amines or other alkaline agent, to neutralize lactic acid 162 and spontaneously alleviate pain caused by acid irritation.

- 20 The shunt 126 can be loaded or coated with initial supplies of nutrients 131, such as sulfate, glucose, glucuronic acid, galactose, galactosamine, glucosamine, hydroxylysine, hydroxylproline, serine, threonine, chondroitin sulfate, keratan sulfate, hyaluronate, magnesium trisilicate, magnesium mesotrisilicate, magnesium oxide, magnosil, orthosilicic acid, magnesium trisilicate pentahydrate, sodium metasilicate, silanates, silanol group, sialic acid, silicic acid, boron, boric acid, other mineral, other amino acid or nutrients 131, to enhance or initiate production of sulfated glycosaminoglycans and collagen within the degenerative disc 100.

- 25 Oral intake of antidepressants has shown temporary pain reduction or pain tolerance in back pain patients. Anti-depressants can be coated on the shunt 126 to treat chronic back pain. The antidepressant coating may include tricyclic antidepressant, serotonin-reuptake inhibitor, norepinephrine reuptake inhibitor, serotonin-norepinephrine reuptake inhibitor, noradrenergic/serotonergic antidepressants, norepinephrine-dopamine reuptake inhibitor, serotonin reuptake enhancers, norepinephrine-dopamine disinhibitors or monoamine oxidase inhibitor. The antidepressant can be amitriptyline, amitriptylin oxide, butriptyline, clomipramine, demexiptiline, desipramine, dibenzepin, dimetacrine, dosulepin/dothiepin, doxepin, duloxetine, imipramine, imipraminoxide, lofepramine, melitracen, metapramine, nitroxazepine, nortriptyline, noxiptiline,
- 30

pipofezine, propizepine, protriptyline, quinupramine, amineptine, iprindole, opipramol, tianeptine, trimipramine, or other antidepressant.

Fibrous formation over the shunt 126 may affect the exchange of nutrients 131 and waste between the disc 100 and bodily circulation or muscle 193. Immuno inhibitor can be coated or incorporated into the shunt 126 to minimize fibrous formation or tissue response. Examples of immuno inhibitors include but are not limited to: actinomycin-D, aminopterin, azathioprine, chlorambucil, corticosteroids, crosslinked polyethylene glycol, cyclophosphamide, cyclosporin A, 6-mercaptopurine, methylprednisolone, methotrexate, niridazole, oxisuran, paclitaxel, polyethylene glycol, prednisolone, prednisone, procarbazine, prostaglandin, prostaglandin E₁, sirolimus, steroids or other immune suppressant drugs.

The shunt 126 can be loaded or coated with a calcium channel blocker for inhibiting activation of neuro-receptor to alleviate pain. The calcium channel blocker can be dihydropyridines, phenylalkylamines, benzothiazepines, magnesium ion, Amlodipine, Felodipine, Isradipine, Lacidipine, Lercanidipine, Nicardipine, Nifedipine, Nimodipine, Nisoldipine, Verapamil, Diltiazem or other calcium channel blocker.

Healthy intervertebral discs 100 are avascular. To ensure avascular conditions, the shunt 126 can be incorporated, coated or partially coated with an anti-angiogenic compound. Examples of anti-angiogenic compounds include, but are not limited to, Marimastat from British Biotech [a synthetic inhibitor of matrix metalloproteinases (MMPs)], Bay 12-9566 from Bayer (a synthetic inhibitor of tumor growth), AG3340 from Agouron (a synthetic MMP inhibitor), CGS 27023A from Novartis (a synthetic MMP inhibitor), COL-3 from Collagenex (a synthetic MMP inhibitor, Tetracycline® derivative), Neovastat from Aeterna, Sainte-Foy (a naturally occurring MMP inhibitor), BMS-275291 from Bristol-Myers Squibb (a synthetic MMP inhibitor), TNP-470 from TAP Pharmaceuticals, (a synthetic analogue of fumagillin; inhibits endothelial cell growth), Thalidomide from Celgene (targets VEGF, bFGF), Squalamine from Magainin Pharmaceuticals (Extract from dogfish shark liver; inhibits sodium-hydrogen exchanger, NHE3), Combretastatin A-4 (CA4P) from Oxigene, (induction of apoptosis in proliferating endothelial cells), Endostatin collagen XVIII fragment from EntreMed (an inhibition of endothelial cells), Anti-VEGF Antibody from Genentech, [Monoclonal antibody to vascular endothelial growth factor (VEGF)], SU5416 from Sugen (blocks VEGF receptor signaling), SU6668 from Sugen (blocks VEGF, FGF, and EGF receptor signaling), PTK787/ZK 22584 from Novartis (blocks VEGF receptor signaling), Interferon-alpha (inhibition of bFGF and VEGF production), Interferon-alpha (inhibition of bFGF and VEGF production), EMD121974 from Merck, KcgaA (small molecule blocker of integrin present on endothelial cell surface), CAI from NCI (inhibitor of calcium influx), Interleukin-12

from Genetics Institute (Up-regulation of interferon gamma and IP-10), IM862 from Cytran, Avastin, Celebrex, Erbitux, Herceptin, Iressa, Taxol, Velcade, TNP-470, CM101, Carboxyamido-triazole, Anti-neoplastic urinary protein, Isotretinoin, Interferon-alpha, Tamoxifen, Tecogalan combrestatin, Squalamine, Cyclophosphamide, Angiostatin, Platelet factor-4, Anginex,

5 Eponemycin, Epoxomicin, Epoxy- β -aminoketone, Antiangiogenic antithrombin III, Canstatin, Cartilage-derived inhibitor, CD59 complement fragment, Fibronectin fragment, Gro-beta, Heparinases, heparin hexasaccharide fragment, Human chorionic gonadotropin, Interferon (alpha, beta or gamma), Interferon inducible protein (IP-10), Interleukin-12 (IL-12), Kringle 5 (plasminogen fragment), Tissue inhibitors of metalloproteinases, 2- Methoxyestradiol (Panzem),

10 Placental ribonuclease inhibitor, Plasminogen activator inhibitor, Prolactin 16kD fragment, Retinoids, Tetrahydrocortisol-S, Thrombospondin-1, Transforming growth factor beta, Vasculostatin, and Vasostatin (calreticulin fragment).

The shunt 126 can be loaded or coated with lactic acid inhibitor or lactate dehydrogenase inhibitor. The lactic acid inhibitor or lactate dehydrogenase inhibitor includes fluoropyruvic acid,

15 fluoropyruvate, levulinic acid, levulinate, oxamic acid, N-substituted oxamic acids, oxamate, oxalic acid, oxalate, beta-bromopropionate, beta-chloropropionate, malonate, sodium formaldehyde bisulfite, chloroacetic acid, alpha-chloropropionate, alpha-bromopropionate, beta-iodopropionate, acrylate, acetoin, malic acid, glycolate, diglycolate, acetamide, acetaldehyde, acetylmercaptoacetic acid, alpha ketobutyrate, thioglycolic acid, nicotinic acid, alpha-ketoglutarate, butanedione,

20 hydroxypyruvic, chloropyruvic, bromopyruvic, 2,3-dihydroxy-6-methyl-4-(1-methylethyl)-1-naphthoic acid, diethyl pyrocarbonate, hexyl *N,N*-diethyloxamate, 3-acetylpyridine adenine dinucleotide, 7-*p*-Trifluoromethylbenzyl-8-deoxyhemigossylic acid, dihydroxynaphthoic acids, N-substituted oxamic acids, gossypol, gossylic iminolactone, derivatives of gossypol, dihydroxynaphthoic acid, 2, 3-dihydroxy-6-methyl-4-(1-methylethyl)-1-naphthoic acid, blue dye,

25 reactive blue dye #2 (Cibacron Blue 3G-A) urea, methylurea and hydantoic acid, glyoxylate, hydroxybutyrate, 4-hydroxyquinoline -2 -3 carboxylic acids, sodium bisulfite, dieldrin, L-(+) beta monofluorolactic acid, fluoro-lactic acid, tartronic acid, mesotartarate, sesquiterpene 8-deoxyhemigossylic acid (2, 3-dihydroxy-6-methyl-4-(1-methylethyl)-1-naphthoic acid), or analogues of these chemicals.

30 In summary, the disc shunt 126 alleviates back pain by (1) drawing nutrients/oxygen/pH buffer 131 into the disc 100, (2) neutralizing lactic acid 162 to alleviate acid burn, (3) converting anaerobic to aerobic conditions to reduce lactic acid 162 production, (4) increasing sulfate incorporation in neutral pH for biosynthesis of glycosaminoglycans. (5) increasing ATP production from aerobic metabolism of sugars to drive biosynthetic reactions in disc 100, (6) bulking up the

disc **100** to take load off painful facet joints **129**, (7) fortifying the disc **100** to reduce spinal instability and muscle tension, (8) rebuilding disc matrix to increase osmolarity, fluid intake and absorption, (9) re-establishing the swelling pressure to sustain disc **100** compression, (10) regenerating the disc **100** for long term pain relief, and/or (11) delivering systemic drugs in disc **100** to treat discitis.

Unlike many surgical interventions of the spine, benefits of the disc shunt **126** include (1) spinal motion preservation, (2) no tissue removal, (3) reversible by extraction, (4) micro-invasive, (5) out-patient procedure, (6) approved implant material, (7) 15-minutes per disc, (8) long-lasting and no-harm-done, (9) no incision, (10) compatible with drugs, conservative treatment or surgical intervention, if needed, and (11) drug coated shunt if needed to expedite pain relief.

The present invention of the shunt **126** or filament **126** is spirally formed distal to a needle **101** and cannula **230**, packing into a disc **100**, reaching one or both diffusion zones **106A**, **106B** between 0 and 3 mm from the endplates **105**, to draw nutrients/oxygen/pH buffer **131** diffused from capillaries **107** at the endplate **105** into the mid layer of the disc **100**. Nutrients and cells **277** can be intradiscally injected for disc regeneration and/or production of biosynthetic product **160**.

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. A pH electrode may be exposed near the tip of the needle **101** to detect the acidity within the disc **100**.

It should be clear to one skilled in the art that the current embodiments, materials, constructions, methods, tissues or incision sites are not the only uses for which the invention may be used. Different materials, constructions, methods or designs for various sections **126A**, **126B** and **126C** can be substituted and used. The disc shunt **126** can be called a filament, strand, thread, line, conduit, wick, sponge or absorbent. Spiraled shunt **126** can be called a coiled shunt or coiled filament **126**. The snagging point **231** can be called the snagger **231**. The filament gripper **111** can be called the gripper **111**. 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 deployment device for implanting a selectable amount of filament within tissue, the deployment device comprising:

- a cannula capable of being guided such that a distal tip thereof may be located within an implant location,
- a filament having a proximal portion and a distal portion,
- a filament needle comprises a proximal end and a distal end, and said distal portion of said filament extends from said distal end of said filament needle,
- said distal end of said filament needle and said distal portion of said filament entirely locatable within said cannula,
- said filament needle is capable of being rotated, thereby twisting said distal portion of said filament into a twisted distal portion located distally to said distal end of said filament needle,
- and said filament needle is capable of being advanced and retracted to pack said twisted distal portion of said filament into the implant location.

2. A deployment device for implanting a selectable amount of filament within tissue, the deployment device comprising:

- a needle capable of reaching and puncturing into an implant location,
- a guide wire comprises a distal portion insertable into said needle,
- a dilator comprises a lumen sized to slide over said guide wire,
- a cannula comprises a lumen sized to slide along said dilator, wherein said cannula comprising a distal tip,
- a filament comprises a proximal portion and a distal portion,
- a filament needle comprises a proximal end and a distal end,
- said distal portion of said filament extends from said distal end of said filament needle,
- said distal end of said filament needle and said distal portion of said filament entirely locatable within said cannula,

said filament needle is capable of being rotated, thereby twisting said distal portion of said filament into a twisted distal portion located distally to said distal end of said filament needle,

and said filament needle is capable of being advanced and retracted to pack said twisted distal portion of said filament into the implant location.

3. The deployment device of claim 1 or 2, wherein said filament needle is capable of being advanced and retracted more than once, thereby allowing said deployment device to pack multiple twisted distal portions of said filament in the implant location.
4. The deployment device of claim 1 or 2, further comprising an advancer, said advancer configured to engage at least a portion of said filament needle and when activated moves said distal portion of said filament out from said distal end of said filament needle.
5. The deployment device of claim 4, wherein said advancer is a helically threaded auger locatable within said filament needle with said filament.
6. The deployment device of claim 4, wherein said advancer is an elongated body having one or more barbs located along a length thereof.
7. The deployment device of claim 1 or 2, wherein said distal tip of said cannula has at least one filament snagger.
8. The deployment device of claim 7, wherein said at least one filament snagger takes the form of a pointed tip.
9. The deployment device of claim 7, wherein said at least one filament snagger takes the form of a spring biased arm.
10. The deployment device of claim 1 or 2, wherein said distal end of said filament needle comprises a filament gripper.

11. The deployment device of claim 10, wherein said filament gripper takes the form of a distally pointing tip.
12. The deployment device of claim 10, wherein said filament gripper takes the form of a cross bar in a lumen of said filament needle.
13. The deployment device of claim 10, wherein said filament gripper takes the form of a cross plane longitudinally dividing a lumen of said filament needle.
14. The deployment device of claim 10, wherein said filament gripper takes the form of a cross stub in a lumen of said filament needle.
15. The deployment device of claim 10, wherein said filament gripper takes the form of a cross stub longitudinal extending into a lumen of said filament needle.
16. The deployment device of claim 1 or 2, wherein said cannula comprises a proximal end, and wherein said proximal end further comprises a funnel.
17. The deployment device of claim 16, wherein said proximal end of said cannula further comprises a latch engaging said proximal end of said filament needle.
18. The deployment device of claim 1 or 2, wherein said proximal end of said filament needle comprises a bobbin, and wherein said proximal portion of said filament is spooled over said bobbin.
19. A method of implanting a selectable amount of filament within tissue, the method comprising the steps of:
 - (a) inserting a cannula into the tissue;
 - (b) inserting a filament needle comprising a length of filament distal to said filament needle into said cannula;

(c) and spiraling said length of filament into the tissue by twisting said filament needle.

20. The method of claim 19, further comprising the steps of:

(d) withdrawing said filament needle;

(e) loading a new length of filament distal to said filament needle in said cannula;

(f) advancing said filament needle;

(g) and spiraling said new length of filament into the tissue by twisting said filament needle.

21. The method of claim 19 or 20, wherein said spiraling of said length of filament is performed by using at least one gripper member located on said filament needle and friction between said length of filament and the tissue.

22. The method of claim 21, wherein the method is used to treat degenerated intervertebral disc.

23. The method of claim 21 wherein the method is used to treat incontinence.