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# SPINE: An Initiative to Reduce Pressure Sore Recurrence

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**Summary**The recurrence rate after pressure sore reconstruction remains high. Primary inciting factors can be organized into efforts aimed at wound prevention: spasticity relief, pressure off-loading, infection and contamination prevention, nutrition optimization, and maximizing extremity function. This article presents our detailed protocol, SPINE, to address each inciting factor with a summary of cases at our facility and review best practices from evidence-based medicine in the literature. *(Plast Reconstr Surg Glob Open 2022;10:e4625; doi: 10.1097/GOX.0000000000004625; Published online 15 November 2022.)*

## INTRODUCTION

The cascade of events leading to pressure necrosis in the spinal cord injury (SCI) patient is precipitated by many factors. Ultimately, lack of attention to position changes and the surface characteristics that the patient is on initiate the wound.[1] Inattentiveness to wound prevention stems from other factors that limit patient care: spasticity, stool and urine contamination, infection, poor nutritional status, older caretakers, heavy patients, and poor upper extremity function. Traditional approaches aimed at treating pressure sores simply as an open wound may not adequately address each factor responsible for the original development, which may be responsible for the high recurrence rate in patients with SCI, ranging from 31% to 79%.[1–4]

In this article, we summarize our protocol to minimize pressure ulcer recurrence using the mnemonic SPINE: spasticity management, pressure off-loading, infection control, nutrition enhancement, and extremity function improvement. The protocol aims to reduce wound recurrence by increasing patient motivation (eg, nutritional optimization with anabolic steroids), maximizing extremity function with tendon transfers and contracture releases, and minimizing dependence on caretakers (eg, colostomy).

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## MATERIALS AND METHODS

An institutional database was reviewed for all immobilized patients undergoing pressure sore reconstruction. Patients were categorized by whether they participated in the SPINE protocol. Patients were assessed for factors contributing to the development of their pressure wounds based on the SPINE protocol. Each factor is considered before surgical reconstruction and a plan for each implemented.

### Spasticity

Spasticity is a velocity-dependent increase in muscle reactivity due to hyperexcitability of the muscle stretch reflex.[5] Even light touch can be enough to incite an involuntary contraction. The longer joints remain in fixed positions, the greater the risk of producing an irreducible contracture.[6] Eventually, the affected extremities become set in a fixed position that alters pressure off-loading capacity. The pressure that develops because of spasm-induced positioning can exceed capillary pressure, leading to necrosis. These pressure-point-position changes also create difficulties in relief positioning for patients and caregivers, ultimately contributing to the creation of a pressure wound or prevention of healing. The modified Ashworth scale grades spasticity on a scale of 0 (none) to 4 (rigid flexion/extension) and allows for a quantifiable measure of progression (Table 1).[7] Information regarding the pharmaceutical treatment of spasticity can be found in Table 2.

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### Surgical Treatments of Spasticity

Patients with multifactorial etiologies for their wounds and long-standing contractures are often malnourished, leading to osteoporosis and a deconditioned state. Staged reconstructive procedures create greater metabolic demands for healing and a higher risk for complications; however, patients with spasticity should have their contractures controlled before pressure ulcer reconstruction for optimal prevention of pressure ulcer recurrence.[8,9] The SPINE protocol prioritizes spasticity relief when it contributes to the patient's pressure-induced wound.

Recalcitrant spasticity or fixed joint contractures require tenotomies and contracture releases. These procedures relieve spasticity and enhancing the ease of care, including turning and moving. The risk profile for complications, including unintended fractures, is derived from the pediatric orthopedic literature reviews on treating patients with cerebral palsy.[10,11] Surgical techniques to minimize the risk profile are discussed next.

### Surgical Technique: Lower-extremity External Fixator Placement

While under anesthesia, the patient's lower extremities are passively stretched to determine the degree of contracture that is fixed. External fixators may not be required if the extremity can be completely passively straightened; if not, then tenotomies, capsulotomies, and external fixators are generally required to achieve the most favorable result (Fig. 1).

To place the external fixator, two 2-cm incisions are made on the anterior border of the tibia. Anteriorly

**Takeaways**

**Question:** How can pressure ulcer recurrence be reduced in the spinal cord injured population?

**Findings:** Factors contributing to ulcer recurrence can be organized by the acronym, SPINE: spasticity control, pressure reduction, infection control, nutritional optimization, and extremity function improvement. Addressing these elements reduces the redevelopment of pressure wounds.

**Meaning:** SPINE offers a systematic approach for addressing elements leading to pressure sores in those with limited mobility. Considering SPINE factors reduces recurrence rate.

**Table 1. Modified Ashworth Scale for Grading Spasticity**

| Grade | Description |
|---|---|
| 0 | No increase in muscle tone |
| 1 | Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the ROM when the affect part(s) is moved in flexion or extension |
| 1+ | Slight increase in muscle tone, manifested by a catch and release or by minimal resistance throughout the remainder (less than half) of the ROM |
| 2 | More marked increase in muscle tone through most of the ROM, but affected part(s) easily flexed |
| 3 | Considerable increase in tone, passive movement difficult |
| 4 | Affected part(s) rigid in flexion or extension |

ROM, range of motion.

**Table 2. Pediatric and Adult Dosing of Frequently Used Antispasmodic Agents**

| Drug | Dosage | | |
|---|---|---|---|
| Oral baclofen | Pediatric | 2–7 years: 10–15 mg/d divided every 8 h; titrate every 3 d in increments of 5–15 mg/d. Maximum of 40 mg/d. | |
| | | ≥8 y: Titrate dose as above to a maximum of 60 mg/d. | |
| | Adult | 5 mg tid. May increase by 5 mg/dose every 3 d. Maximum of 60 mg/d | |
| Oral tizanidine | Pediatric | Pediatric dosing unavailable. | |
| | Adult | 4 mg recommended, may be increased by 2–4 mg/dose. Dose may be repeated every 6–8 h to a maximum of 36 mg/d. | |
| Oral diazepam | Pediatric | 0.12–0.8 mg/kg/d every 6–8 h | |
| | Adult | 2–10 mg given 2–4 times/d. Maximum of 40 mg/d. | |
| ITB | Pediatric | Screening | **Test bolus:** 50 mcg over <1 min. If inadequate response in 8 h, 75 mcg 24 h later; if still inadequate, 100 mcg 24 h later (max). May start with 25 mcg for small patients. |
| | | Titration (after 24 h) | **Initial implant dose:** 2× screening dose that gave positive effect administered over 24 h. After 24 h, daily dose should be increased by 5–15% only once q24h. |
| | | Maintenance | Increase by 5–20% or decrease by 10–20% during periodic pump refills; usual range 90–700 mcg/d. Max 1000 mcg/d. |
| | Adult | Screening | Same as pediatric screening. |
| | | Titration (after 24 h) | **Initial implant dose:** 2× screening dose that gave positive effect administered over 24 h. **SCI origin:** Increase by 10–30% q24h. Cerebral origin: Increase by 5–15% q24h. |
| | | Maintenance | **SCI origin:** Increase by 10–40% or decrease by 10–20% during periodic pump refills. Usual range 300–800 mcg/d. Max 1000 mcg/d. **Cerebral origin:** Same as pediatric maintenance. |

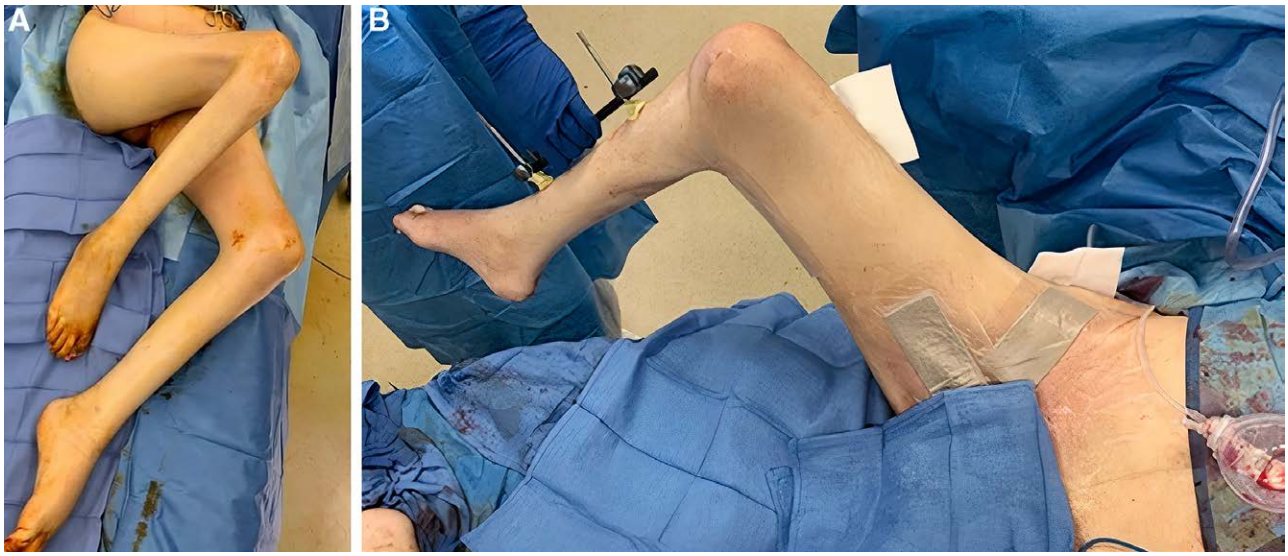


**Fig. 1.** A 29-year-old tetraplegic man with severe fixed contractures and spasticity from a traumatic brain injury. A, Preoperative fixed position of the lower extremities. B, Postoperative placement of external fixator. Thirty pounds of progressive traction was placed to straighten the leg.

placed tibial external fixator neutralizes anteroposterior and transverse bending forces.[12] The dissection is carried down to the bone level and the elevated periosteum. Next, a Shanz screw is placed at both sites and is confirmed fluoroscopically. The diameter of the screw should be less than one-third of the bone diameter to minimize fracture risk at the pin site.[13] We favor 3–4 mm Shanz screws in most patients. The placement of the screws is at least 15 mm (two fingers breadth) below the joint line to avoid placement into the synovium or capsule of the knee joint.[14]

Connectors and a carbon rod are used to connect the screws. The patient is placed on a traction bed with progressively increasing weight until the extremity is straightened 2–3 weeks postoperatively. The external fixator pulls tension on the leg during the procedure. We have used progressive traction followed by application of medium intensity, up to 30% of patient total body weight (usually up to 25 or 30 kg). Extension by stages reduces complications associated with excessive traction.[15]

**Surgical Technique: Hip and Knee Flexor Tenotomies and Capsulotomies**

The hip adductors and flexors are addressed from an inguinal and medial thigh approach at the same time as external fixator placement. The most powerful muscle-tendon unit is the adductor longus, which can easily be palpated through the skin. This is released proximally near the pubis. Deep to the adductor longus, the adductor brevis is divided through the muscle. The anterior branch of the obturator nerve lies on the anterior surface of the brevis and, if nonfunctional, may be divided. More medially and posterior, the gracilis muscle is divided proximal to its vascular pedicle to be rotated and fill dead space in the inguinal area. Preservation of neurovascular supply of the gracilis should be considered for that reason. These muscles are also divided distally to reduce forces on knee flexion. Division of the adductor magnus, with or without division of the posterior branch of the obturator nerve, is considered at this point. Attention is directed proximally, and through the same incision, the pectineus is divided. The femoral neurovascular bundle lies just lateral to that muscle and is preserved (Fig. 2).

After all tendons are released, the joint is passively stretched, performing a controlled capsulotomy. We have sometimes found that the hip capsule itself is excessively tight and must be released to achieve the desired hip extension. The femoral vessels are between the iliopsoas and pectineus; thus, the intervals between the sartorius muscle laterally and TFL and the TFL and iliopsoas are safe intervals to approach the hip capsule.

The knee flexors, including the semimembranosus, semitendinosus, biceps femoris, gracilis, and sartorius, are released after the hip is addressed or can be addressed in the prone position if the pressure wounds are debrided first. Two incisions are made: laterally over the biceps femoris tendon and medially over the semimembranosus and semitendinosus tendons. The popliteal nerve and vessels can feel taught but should not be mistaken for a tendon. Gentle traction can be applied to these joints; however, fractures can occur if the operator is too aggressive.

**Prevention of Pressure Necrosis**

The treatment of pressure wounds begins with preventing new ones from occurring. The pressure sore begins with an isolated event (eg, new illness) or poor habits aggravated by limited mobility, moisture from incontinence, decreased or absent sensation, and substance use.[16] The wounds commonly occur from poor pressure relief

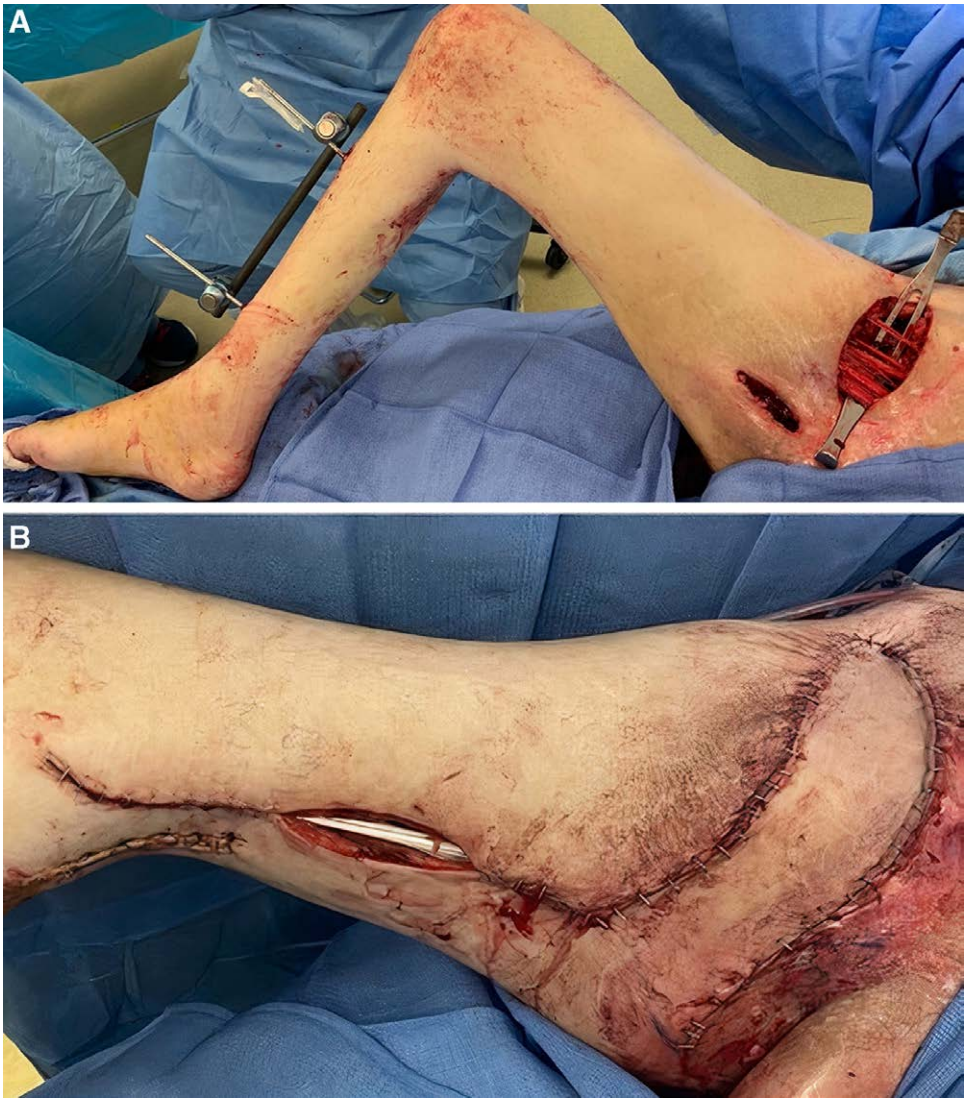


**Fig. 2.** Incision site for release of hip flexors and hip adductors is placed at their origins. The femoral vessels are bordered medially by the adductor longus and laterally by the sartorius muscle. The retractor is placed under the vessels (A) after the tenotomies have been performed and the hip has been released from its contracted position. The gracilis muscle with skin paddle is used to fill in the defect after tenotomies and hip straightening (B).

habits; thus, relearning good habits is essential before final reconstruction.

A physical and occupational therapy regimen to retrain shifting positions and off-loading is initiated and continued until it becomes second nature. To start, we ask patients to set an alarm every 15 minutes while awake as a reminder to shift from side to side and every 2 hours to change positions completely. Establishing this change of habits early in care provides the best chance of operative success. Once the patient has demonstrated commitment to preventing pressure necrosis, the final reconstructive procedure is scheduled.

After reconstructive surgery, the patients are maintained on air-fluidized mattresses (eg, Clinitron) for 6 weeks, as it takes approximately 42 days for skin to achieve 85% preoperative tensile strength.[17] The specialty mattress provides a proper flotation environment; thus, changing the patient's position is not required while they recover.[18] The occupational therapist performs pressure mapping with a textile sensor mat (Fig. 3), which determines high-pressure points and tailors the alternating air cells to the unique patient. Alternating mattresses slowly inflate and deflate to redistribute pressure at an adjustable time, allowing blood flow to nourish the skin. In addition to relieving pressure, the low air loss mattresses keep the skin dry, further preventing maceration and pressure necrosis.[20,21]

An adequate wheelchair cushion and bed mattress are vital to preventing new ulcerations. Five randomized controlled trials compared pressure ulcer frequency in various wheelchair foam cushion configurations and found that there is no overall superior cushion; however, the gel cushion plus foam pad appears better than foam alone.[22] There is still insufficient evidence to

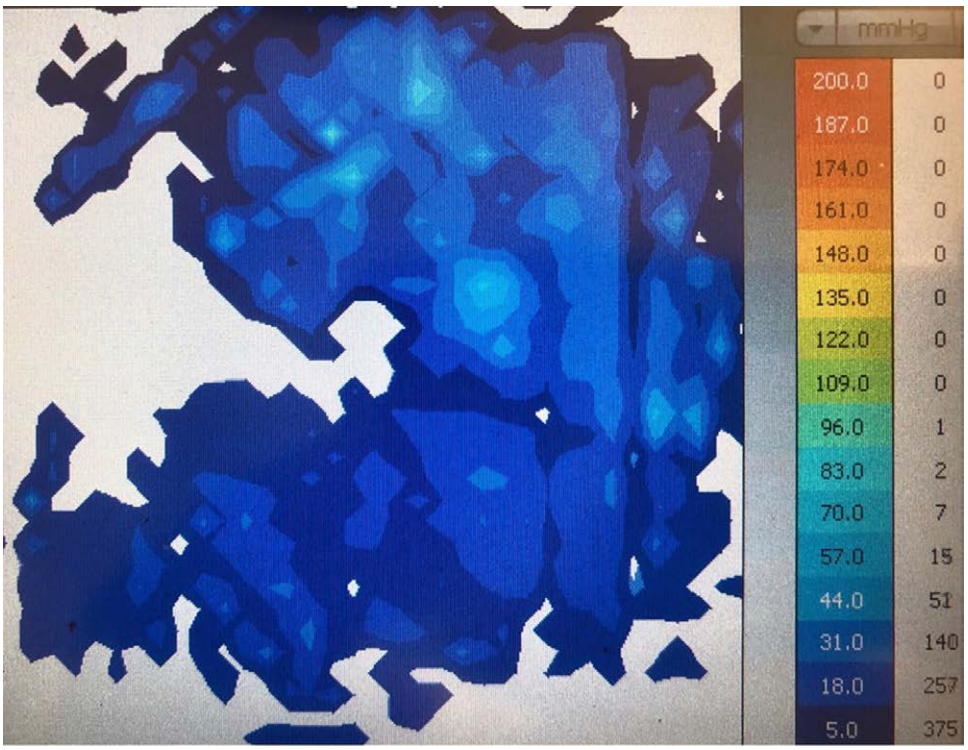


**Fig. 3.** This patient was sitting on a fitted gel cushion in a power wheelchair during her mapping. Pressure load mapping is important for assessing the risk an individual has for developing an ulcer. A color-coded pressure map is generated by a thin sensor mat placed on the patient's wheelchair and connected to the tablet that displays the map. Evidence shows that capillary blood flow with normal blood pressure is approximately 32 mm Hg.[19] Thus, any load greater than 32 mm Hg should not be sustained to decrease the risk of pressure ulcer development. This image demonstrates areas at risk for pressure ulcer development, most noticeably at the right ischial and trochanteric sites (identified with the lighter blue colors). This is a satisfactory pressure distribution, as the weight from sitting alone will increase pressure on the vessels. Even with the satisfactory pressure distribution, this patient will still require position changes to best prevent ulcer development. An unsatisfactory pressure distribution would contain green/yellow/red colors, indicating higher pressure.

determine "the best" cushion due to the lack of replication in most comparisons and the poor quality of many of the trials.[22] We prefer the 4 inch air-cell cushion (eg, ROHO) because it provides excellent alternating pressure off-loading.[23]

**Infection Control**

Permanent wound closure is only achievable after drainage of infected areas, debridement of devitalized tissue, and attentive wound care to encourage healing. Recurrent bacterial burden prevents the body from advancing past the inflammatory phase of wound healing, ultimately leading to a nonhealing chronic wound.[24] The sacral, ischial, and trochanteric bony prominences can develop osteomyelitis due to repeated stool or urine contamination and necessitate more extensive surgery, staged reconstruction, and prolonged antibiotic courses. Osteomyelitis occurs in approximately one-third of these ulcers.[25]

Incontinence is a leading cause of skin breakdown and infection of pressure ulcers. The wound and surrounding skin must be kept clean and free of urine and feces. Recommendations for colostomy creation are related to a patient's or caregiver's motivation and continence. The colostomy is generally reversible; however, most patients find them convenient and keep it permanently. We have found they improve patient independence and caregiver attentiveness to the patient. Pressure sore reconstruction can be performed on the same day as colostomy creation.

The need for surgical debridement of pressure sores depends on the stage. Stage I pressure ulcers, a localized area of nonblanchable redness, can heal in approximately 3 days with off-loading alone if caught early.[26] Left unnoticed, the area will progress to a shallow ulcer with partial thickness skin loss, classified as stage II. These take an average of 23 days to heal without surgery.[27] Once the ulcer reaches stage III (full skin thickness loss) or IV (full tissue thickness loss), it may take 6 months to heal if not treated surgically; however, some never heal.[28] Surgical reconstruction of grossly infected stage III or IV ulcers is typically conducted via a two-stage strategy, debridement and flap coverage, followed by antimicrobial therapy.

**Surgical Treatment of Pressure Sores**

1. *Debridement:* The key to successful reconstruction is initial excisional debridement of all devitalized, infected, and chronically fibrosed tissue. Predebridement wound cultures of soft tissue and bone guide antimicrobial therapy. The wound is painted with methylene blue, and all painted tissue is excised. The wound is irrigated and re-inspected for additional rounds of selective debridement. Finally, postdebridement quantitative cultures are obtained. If this culture has less than 50,000 colonies per gram, it is safe to perform wound closure.[29] Single-stage debridement and reconstruction can safely be performed in exceptional cases.
2. *Reconstruction.* Flap selection is designed to avoid incisions over prominent pressure points and allow flap readvancement if breakdown or recurrence occurs. We mark the greatest prominence area and attempt to avoid incising directly over the marked area. Defect size, additional wounds, prior reconstructions, and pressure points all influence the ultimate flap design (Fig. 4). Sacral wound reconstruction can be accomplished with fasciocutaneous gluteal rotational flaps, V-Y advancement flaps, or superior gluteal artery perforator islandized transposition flaps. For ischial wounds, medial or lateral thigh rotational flaps, inferior gluteal rotational flaps, inferior gluteal artery perforator, or gracilis flaps can be used. Tensor fascia lata flaps can also be used when reconstructing trochanteric defects simultaneously. Trochanteric wounds are generally reconstructed with tensor fascia lata or vastus lateralis muscles flaps.

After closure, an incisional negative pressure wound therapy dressing vacuum-assisted closure (VAC) is applied and kept on the wound and changed weekly over a silver nonstick dressing (eg, Acticoat) for the duration of the initial healing phase (eg, 6 weeks).

The VAC keeps the wound clean and reduces tension. Suh and colleagues evaluated the effect of negative pressure wound therapy on incisional wounds with dead space and found a significant drainage reduction and increased skin perfusion. Additionally, increased collagen synthesis was observed, indicating enhanced healing compared to treatment with gauze dressing alone.[30] Drawbacks include the cost, nursing expertise, and patient compliance during VAC changes in challenging areas. Patient discomfort or bleeding with VAC bandage changes can also occur, although the benefits generally far outweigh the risks.[30,31]

**Nutrition Optimization**

Hypoalbuminemia is traditionally associated with poor nutritional status and subsequent high incidence of postoperative complications, including wound dehiscence and ulcer recurrence.[32] Recent evidence has shown that this nutritional marker alone is inadequate as an independent predictor of complications in pressure sore patients.[33] Serum albumin levels may be affected only in those experiencing "extreme" starvation (defined as a body mass index <12 $kg/m^2$) or greater than 6 weeks' starvation.[34]

A variety of factors lead to hypoalbuminemia, and the underlying etiology for creating these wounds is pressure, not malnutrition.[35] A multifactorial, regressional analysis demonstrated that preoperative albumin level does not predict postsurgical complications in pressure ulcer patients.[36] The patient's nutrition is vital to wound healing, and this is better assessed through the patient's history, dietary habits, and body mass index.

The overall level of the patient's motivation, self-care, and resources are essential in wound healing after

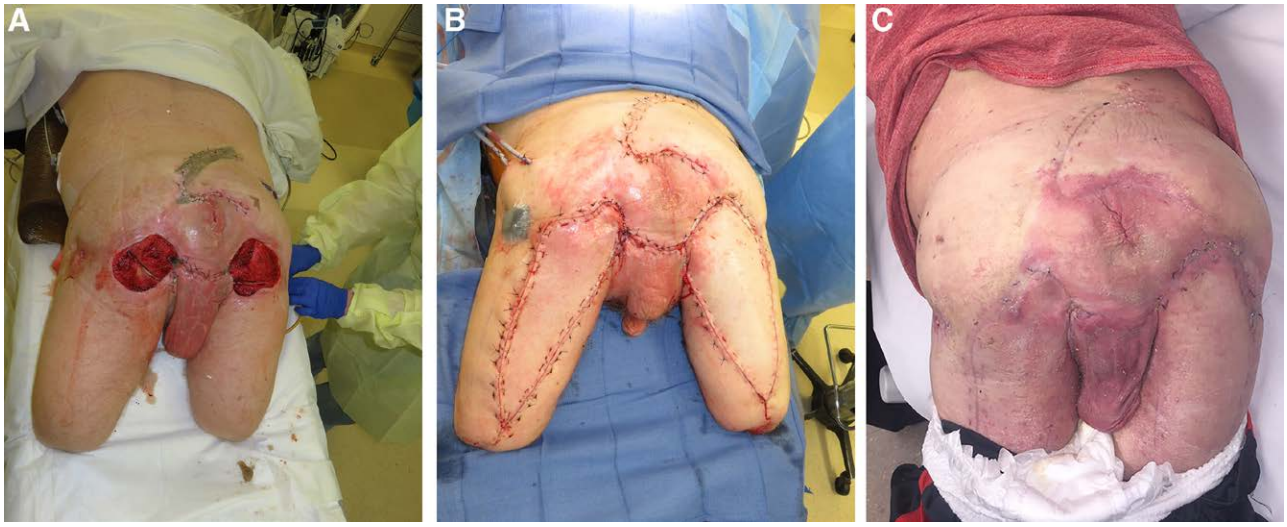


**Fig. 4.** A 50-year-old man with sacral, perineal, and bilateral ischial pressure wounds. Perineal wounds occur after multiple debridements of the ischial prominence or after Girdlestone procedures. This patient underwent reconstruction in multiple stages (A) until all wounds were closed (B). This patient completed the SPINE process and healed completely 1 month after surgery (C).

reconstruction. If patients are unwilling to comply with a preoperative nutrition plan, they are unlikely to comply with postoperative pressure relief programs. Participating in a 6-week nutritional program can build a sense of interest in their outcome while providing the clinician with a sense of their willingness to comply with future physician-instructed programs.

Appetite stimulants with anabolic steroids give patients more energy, strength, and motivation to participate in their care, thus reducing wound recurrence. We prefer oxandrolone supplementation for creating an anabolic state and engaging patient motivation when needed. Oxandrolone is metabolized hepatically and has a favorable side-effect profile suitable for most patients.[37] Added benefits of oxandrolone include stimulation of lean muscle mass, including the upper extremities, which is imperative because this may provide some patients with a greater ability to assist with transfers.[38]

Although not required by all patients, we use oxandrolone in most patients undergoing reconstruction for pressure wounds, given its overall safety and efficacy. Our treatment algorithm includes supplementing the diet with three protein shakes per day and 2.5–20 mg/day oxandrolone for at least 6-weeks after surgery and continuing up to a year.

**Extremity Function Improvement**

Tetraplegic patients are limited in assisting with position changes, transfers, hygiene, and self-care. This lack of independence affects nutrition, motivation, independence, and ultimately increases the development of pressure wounds and their recurrence.[39] Tendon and muscle transfers are designed to provide more patient independence for self-care.

Upper extremity tendon transfer surgeries are performed in fewer than 10% of tetraplegic patients,[40] even though it is estimated that 65%–75% of this population could benefit from this procedure. Surgical goals are to restore elbow extension, pinch, grasp, and release. Motivated patients who are active in therapy and understand their expected outcomes appear to have the highest satisfaction after tendon transfer procedures.[41]

Improvement of upper extremity function begins with identifying redundant functional muscles to transfer for restoration of lost movements.[42] The International Classification of Surgery of the Hand in Tetraplegia identifies candidates by grouping them into categories: 0 (no functioning muscles) to 9 (all function except intrinsics) (Table 3). Tetraplegic patients most amenable to meaningful tendon transfers are classified as O/Cu group 4–5. For this group, redundant wrist extensors can be used to restore finger flexion and pinch function, which aids in operating an electric hand-controlled wheelchair (Fig. 5). Additionally, patients with functioning elbow flexors can transfer the biceps or deltoid to the triceps to restore elbow extension that can be used for changing position, push-offs, and transfers (Fig. 6).

The pressure sore is the immediate priority; however, the wound will recur if contributing factors are left unaddressed. We perform tendon transfers while a patient is in their postoperative recovery period from a pressure sore reconstruction, generally on a Clinitron mattress. Data and discussions of tendon transfers reducing pressure ulcers in the tetraplegic population are still forthcoming; however, available data have supported increased quality of life and reduced ulcer recurrence after improving hand and arm function.[43] (**See Video [online],** which displays the improved range of motion of the left upper extremity following tendon transfer. This is the same patient from Figure 6. The increased range of motion can be compared with that of the right upper extremity, which has not undergone tendon transfer.)

**Table 3. International Classification for Surgery of the Hand in Tetraplegia Was Developed to Identify Candidates for Upper Extremity Restoration via Tendon Transfer**

| Group | Motor Characteristics with Lowest Muscle Research Council Grade ≥ 4 |
|---|---|
| 0 | No functioning muscles |
| 1 | Br |
| 2 | Br + ECRL |
| 3 | Br + ECRL + ECRB |
| 4 | Br + ECRL + ECRB + PT |
| 5 | Br + ECRL + ECRB + PT + FCR |
| 6 | Br + ECRL + ECRB + PT + FCR + EDC |
| 7 | Br + ECRL + ECRB + PT + FCR + EDC + EPL |
| 8 | Br + ECRL + ECRB + PT + FCR + EDC + EPL + Partial FDP |
| 9 | Lacks only intrinsics |
| Sensibility | O or Cu |

The grading indicates the number of functioning muscles available for transfer. O or Cu = ocular or cutaneous sensation of the thumb (Cu); Br = brachioradialis; ECRL = extensor carpi radialis longus; ECRB = extensor carpi radialis brevis; PT = pronator teres; FCR = flexor carpi radialis longus; EDC = extensor digitorum communis; EPL = extensor pollicis longus; FDP = flexor digitorum profundus.

## RESULTS

Over a 5-year period between 2015 and 2020, a total of 69 pressure sores were reconstructed on immobilized patients, with an overall recurrence rate of 31.9%. Of the 13 pressure-induced wounds treated with the SPINE protocol, the recurrence rate was 7.6% (N = 1). In tetraplegic patients who underwent upper extremity tendon transfers to improve independence, zero of eight wounds recurred versus 37.5% (21 of 56) in patients who did not complete the SPINE protocol. A comprehensive review of the patients included in the study can be found in Supplemental Digital Content 1. **(See table, Supplemental Digital Content 1,** which displays the summary of 39 cases. **http://links.lww.com/PRSGO/C227**.)

## DISCUSSION

The lifetime incidence of pressure ulcers in the SCI patient is 31%–79%,[1–4] due to a combination of immobility and decreased sensation. Recurrence rates after pressure sore reconstruction are even higher, with some studies indicating up to almost 100%.[4,44] In the United States, 2.5 million people are affected by pressure ulcers, and the cost is estimated to exceed $26.8 billion annually ($107,000 per patient per year).[45] Prevention of pressure ulcers will make a significant economic impact; thus,

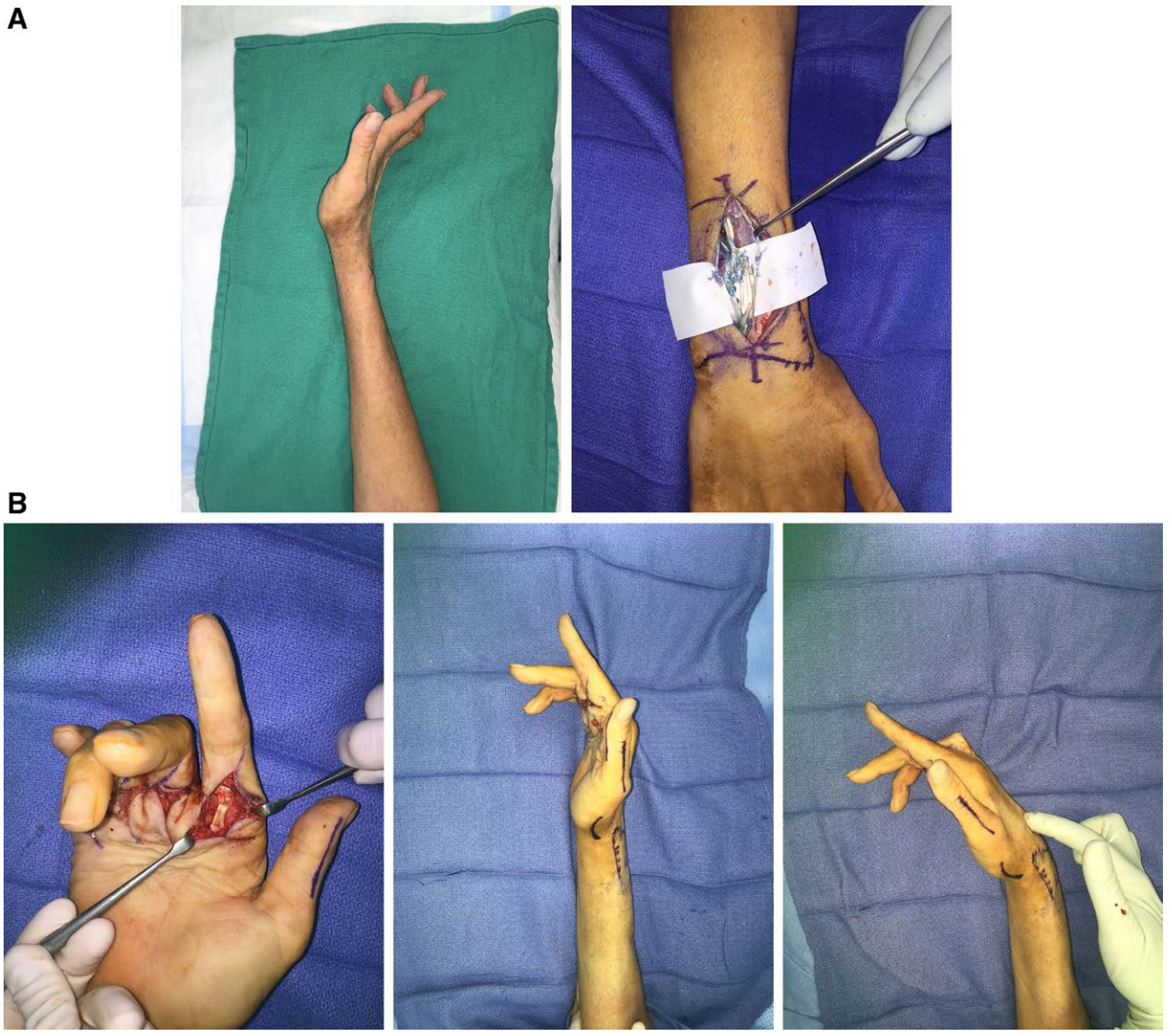


**Fig. 5.** A 59-year-old man in O/Cu group 5. A, Preoperative static position of the hand with myostatic contracture of the extensor digitorum communis and extensor pollicis longus muscle bellies preventing wrist or digit flexion. This patient underwent fractional lengthening of the finger extensor tendons and zancolli lasso FDS tendon transfers to improve static and dynamic hand function. B, Postoperative static position of the hand. The patient graded his result as having a major improvement in his quality of life.

pressure ulcer reconstruction should greatly emphasize prevention postoperatively.[16]

Patients with a C5-8 SCI in their 20s live up to 40 years post-injury[41]; therefore, risk factors for pressure ulcers should be minimized early. When surgery is required, it becomes crucial for the physician to address contributing factors while educating the patient about their role in preventing recurrence to prevent readmits and provide the best possible quality of life.

Addressing each secondary provoking factor is necessary to maintain closure, promote healing, and prevent a recurrence. Organized by the acronym SPINE, these include:

1. Treatment of ***S***pasticity with medications and, if necessary, tenotomies and joint contracture releases;
2. Eliminating ***P***ressure from rigid surfaces by using high-profile air–cell cushions and low air loss mattresses;
3. Treatment of ***I***nfection, including control of stool and urine contamination;
4. Creating a ***N***utritional anabolic state; and finally,
5. Improving ***E***xtremity function with tendon transfers.

Reducing pressure sore recurrence requires changing the patient habits leading to its occurrence. This article presents our multifaceted approach to reducing pressure-induced wounds and introduces our SPINE algorithm. This protocol guides the resolution of pressure ulcer factors and includes a multi-disciplinary approach, incorporating plastic surgery, orthopedic surgery, neurology, occupational therapy, urology, colorectal surgery, infectious disease, nutrition specialists, and social work. Each


**Fig. 6.** A 34-year-old incomplete tetraplegia man underwent (A) biceps-to-triceps tendon transfer to restore (B) elbow extension. Addition of this function allows the patient to assist with transfers and perform independent pressure off-loading.

topic of SPINE deserves a separate discussion with an analysis of data, which we hope to present in future articles.

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## PATIENT CONSENT

*Patients provided written consent for the use of their images.*

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