

# Wound Care & Hyperbaric Medicine magazine

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## Note from the Editor

In this issue of *WCHM*, we continue to search out practical applied topics in wound care, diving, and hyperbaric medicine.

First, Gretchen Dixon continues coverage of the changing requirements for billing and coding. As a provider, your documentation is the key source of information supporting the level of acuity with intensity of services and complexity of care for each of your patients. As we draw nearer to the ICD-10-CM go-live date of October 1, 2015, now is the time to identify and address any weaknesses that may affect future reimbursement for provided services. Start reading on page 7.

Our prolific author Dr. Strauss and his colleague Dr. La explain that while toe deformities may appear to be unimportant, they can have serious consequences. This is the situation especially in patients with diabetes who have neuropathies. They reveal a dozen paradigms about toe tenotomies. Find the full article on page 27.

This issue, we welcome Dana Graves, DDS, DMSc, who led a team of researchers investigating the FOXO1 molecule's role in promoting healing in patients with chronic wounds and diabetes. *WCHM* interviewed him about information on his recently published research. Dr. Graves is a professor in the Department of Periodontics, Vice Dean for Scholarship and Research, and Director of the Doctor of Science in Dentistry Program for Penn Dental Medicine under the umbrella of the University of Pennsylvania. Read the interview or listen to the podcast version on page 24.

Darren Mazza continues his coverage on safety issues for CHTs with his article on air breaks during hyperbaric treatment. Read more on page 21.

Also included is this issue's Clinic In Focus, which features the Swedish Edmonds Center for Wound Healing and Hyperbarics. If you are a part of an exceptional hyperbaric or wound care center, contact us today to be our next featured clinic! Find more on page 16.

We encourage our readers to take advantage and share *WCHM* with colleagues and clients alike. In addition, if you have a clinic, be sure to add your details to our Map of Wound Care and HBO Centers.

Please join us in delivering the highest quality publication in the industry, focused on advancing the knowledge and practice of wound care, diving, and hyperbaric medicine by providing your comments, articles, industry information, press releases, and updates.

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# ICD-10-CM Wound Care Review Part 1

## Bolstering Your Documentation for the Four Most Common Wound Diagnoses in the Clinical Setting

Gretchen Dixon, MBA, CCS, CPCO

### Level of Acuity with Intensity of Services and Complexity of Care

As we draw nearer to the ICD-10-CM go-live date of October 1, 2015, now is the time to identify and address any weaknesses that may affect future reimbursement for provided services. As a provider, your documentation is the key source of information supporting the level of acuity with intensity of services and complexity of care for each of your patients. As we have discussed before, if your documentation is without details, inconsistent, ambiguous, or incomplete, your patient's level of acuity and provided services may not be supported. It is anticipated that within approximately two years after the implementation of ICD-10-CM, through data mining, unspecified codes will be reimbursed at a lesser value. Why? Unspecified codes only support a low level of acuity with the intensity of services and complexity of care. The use of unspecified codes may affect your profiles, which are monitored by third party payers as well as the public. Third party payers can remove providers from their plans if they feel there is an over-reporting of services based on diagnoses reported.

### Focus on ICD-10-CM and Wound Care

Take this time to revisit your use of selected unspecified codes. In the wound care environment, a good start would be to review documentation details required for the four most common types of ulcers/wounds treated in a wound clinic. Of course, this does not exclude the fact these types of wounds are diagnosed in all healthcare settings, thus the documentation information value is for all types of providers. This article breaks out the fol-

lowing wounds/ulcers and outlines their documentation requirements for this specificity:

1. Diabetic foot ulcer
2. Pressure ulcer
3. Trauma wound (will be discussed in a future article)
4. Arterial ulcer

### REMINDER: Don't forget to sequence diagnoses!

List your first diagnosis (called the principal diagnosis or PDX), which supports the reason for the encounter and services provided. Per ICD-10-CM Coding Manual directions: code first any associated underlying condition. Instruction examples are to code first diagnosis, such as atherosclerosis of lower extremities, diabetic ulcers, or associated gangrene.

List all other diagnoses (called secondary diagnoses), which describe any co-existing conditions which require or affect patient care treatment or management.

Examples:

1. First diagnosis: type 2 diabetes mellitus with foot ulcer of right planter foot
2. Secondary diagnoses that affect the management of the patient's treatment:
  - Right foot plantar surface ulcer involves the necrotic subcutaneous tissue
  - Type 2 diabetes mellitus with diabetic peripheral angiopathy without gangrene (microvascular disease)
  - Hyperglycemia

## GENERAL ICD-10-CM DOCUMENTATION GUIDELINES FOR ULCERS OR WOUNDS

When describing ulcers and wounds, it is necessary to include additional details in specific categories:

- Anatomical location—be as specific as possible
- Laterality—right, left, bilateral
- Diagnosis-related complications (example: atherosclerosis of specified vessel involving the right lower extremity with ulcer)
- Secondary diagnoses that affect the patient's treatment and/or management of care
- Describe the severity (depth) of the non-pressure ulcer (wound descriptions provided later in this article)
- Pressure ulcers are required to have the stage reported, not the severity of the wound tissue noted above

**NOTE:** Electronic health records can help the provider include specific information, thus improving clinical documentation. However, the point-and-click process of adding information can result in the use of pre-determined verbiage for general diagnoses rather than objective observations made during the patient visit. Therefore, documentation details may need to be added as free text for clarification. **DO NOT** rely on the same identical phrases used for all patients during visits! The individuality of the patient is frequently eliminated and patient records all read the same.

## WOUNDS MOST COMMONLY TREATED IN A WOUND CLINIC

### Diabetic Foot Ulcer

Diabetic ulcers (DFUs) require detailed documentation and more than one ICD-10-CM code to accurately describe the patient's condition. The first diagnosis is always the reason for the encounter, which would be the ulcer.

**DFU Case Scenario:** The patient's diagnosis is ulcer on the plantar surface, involving subcutaneous tissue, of the right foot due to diabetes with circulatory complications and hyperglycemia. Patient's blood glucose is not well controlled and ranges from 180 to 230 mg/dL on a regular basis as reported by the patient. Currently the patient is on the following medications: metformin, glipizide, and NPH insulin injections in the morning and at bedtime.

Documentation details of DFUs are often problematic and can result in the diagnosis being incorrectly coded. This is due to documentation that is ambiguous, inconsistent, vague, or incomplete, which can cause the question of which came first: the ulcer in a diabetic patient or a diabetic patient who developed an ulcer due to complications of the disease? **Documentation needs to clearly identify the causative relationship since not all wounds/ulcers have this relationship with diabetes.**

So, what do you document? Looking at the above scenario, we can discuss the ICD-10-CM coding options that would need to be applied.

This is an established patient with an identified DFU of the plantar surface on the right foot. The ulcer involves the necrotic subcutaneous tissue and is due to diabetes microvascular disease. The patient exhibits hyperglycemia with blood sugar ranges from 180 to 230 mg/dL, with today at 190 mg/dL.

The first diagnosis would be type 2 diabetes mellitus with a skin ulcer. The following information relates to specific documented diagnoses of diabetes with complications related to wound care services. Note how the ICD-10-CM code description has been expanded as a combination code different from the broadness of ICD-9-CM diagnosis codes. Table 1 provides the specific coding description of options for type 2 diabetes mellitus with skin complications. Blue highlight represents the applicable code.

**Table 1**

ICD-10-CM Code	ICD-10-CM Code Description
E11.620	Type 2 DM with diabetic dermatitis
E11.621	Type 2 DM with diabetic foot ulcer
E11.622	Type 2 DM with diabetic other skin ulcer
E11.628	Type 2 DM with diabetic other skin ulcer complications

DM=diabetes mellitus

**Diabetes Mellitus-Related ICD-10-CM Codes**

In ICD-10-CM, several changes are noted according to ICD-10-CM Draft Official Guidelines for Coding and Reporting. During the development of ICD-10-CM, diabetes combination codes were created requiring the following three components to be reported:

1. Type of diabetes (type 2 is the most common in wound clinics)
2. Body system affected (nervous, circulatory, nephrology, skin)
3. Complication affecting a body system

**NOTE:<sup>1</sup>**

- ICD-10-CM coding guidelines: select or assign codes as necessary to describe all of the complications of the disease
- Sequence diagnoses based on reason for the encounter
- If the type of diabetes is not documented, the default diagnosis code to be selected is type 2 diabetes mellitus or ICD-10-CM code E11. XX
- Document when the patient is on insulin versus oral hyperglycemic medications
- In describing the patient’s blood sugar control, the provider needs to avoid the use of terms controlled or uncontrolled and use the terms inadequately, out of control, or poorly controlled, which all code to hyperglycemia. If the patient has periods of low blood sugars then the term hypoglycemic should be documented.

The second diagnosis would be type 2 diabetes mellitus with diabetic foot ulcer located on the plantar ulcer of the right foot. The ICD-10-CM code is a

combination code noting the anatomical location and the severity of the ulcer (DFU scenario documents involving subcutaneous tissue), so it is important to include in your documentation the depth of the non-pressure ulcer/wound. Table 2 lists the options available for selection based on the provider’s documentation. Blue highlight represents the applicable code

Keep in mind the documentation of laterality and code selection changes. There is no selection for bilateral extremity for this category. However, there is a code for unspecified laterality and unspecified severity of the ulcer, which should never be coded in the wound care setting.

Diagnosis: type 2 diabetes mellitus with diabetic foot ulcer located on the plantar surface of right foot.

Severity (depth): fat layer exposed (subcutaneous tissue)

**Table 2**

Severity (Depth) Documentation	ICD-10-CM Codes	ICD-10-CM Descriptions
Is it limited to breakdown of skin?	L97.411	Non-pressure chronic ulcer of right heel & midfoot (includes plantar surface) limited to breakdown of skin
Is the fat layer exposed?	L97.412	Non-pressure chronic ulcer of right heel & midfoot (includes plantar surface) with fat layer exposed
Is there necrosis of muscle?	L97.413	Non-pressure chronic ulcer of right heel & midfoot (includes plantar surface) with necrosis of muscle
Is there necrosis of bone?	L97.414	Non-pressure chronic ulcer of right heel & midfoot (includes plantar surface) with necrosis of bone
No severity specified (should not be used)	L97.419	Non-pressure chronic ulcer of right heel & midfoot (includes plantar surface)

The third diagnosis would be type 2 diabetes mellitus with circulatory complications supporting the microvascular disease. Table 3 provides the specific coding description of options for type 2 diabetes mellitus with diabetic circulatory complications.

**Table 3**

ICD-10-CM Code	ICD-10-CM Code Description
E11.51	Type 2 DM with diabetic peripheral angiopathy without gangrene
E11.52	Type 2 DM with diabetic peripheral angiopathy with gangrene
E11.59	Type 2 DM with diabetic with other circulatory complications

DM=diabetes mellitus

The diagnosis would be for type 2 diabetes mellitus with either hyperglycemia or hypoglycemia. Tables 4 and 5 illustrate the specific coding options for type 2 diabetes mellitus with management issues commonly occurring in a wound clinic.

**Table 4**

Hyperglycemia	
ICD-10-CM Code	ICD-10-CM Code Description
E11.65	Type 2 DM with hyperglycemia

DM=diabetes mellitus

**Table 5**

Hypoglycemia	
ICD-10-CM Code	ICD-10-CM Code Description
E11.641	Type 2 DM with hypoglycemia with coma
E11.649	Type 2 DM with hypoglycemia without coma

DM=diabetes mellitus

Possible Codes: If the patient also has peripheral neuropathy, Table 6 provides the specific documentation details and coding options for type 2 diabetes mellitus with diabetic neurological complications.

**Table 6**

ICD-10-CM Code	ICD-10-CM Code Description
E11.40	Type 2 DM with diabetic neuropathy, unspecified (needs to be specific)
E11.41	Type 2 DM with diabetic mononeuropathy
E11.42	Type 2 DM with diabetic polyneuropathy
E11.43	Type 2 DM with diabetic autonomic (poly) neuropathy
E11.49	Type 2 DM with other diabetic neurological complication

DM=diabetes mellitus

Based on the case scenario, the following ICD-10-CM codes should be selected for this patient:

- E11.621—Type 2 diabetes mellitus with diabetic foot ulcer
- L97.412—Non-pressure chronic ulcer of right heel & midfoot (includes plantar surface) with fat layer exposed (reason for the wound care department encounter)
- E11.51—Type 2 diabetes mellitus with diabetic peripheral angiopathy without gangrene
- E11.65—Type 2 diabetes mellitus with hyperglycemia

**Pressure Ulcer**

Pressure ulcers are frequently treated in the wound care setting. Therefore, ICD-10-CM includes in the description the combination of laterality with the anatomical location of the ulcer as well as the stage of the pressure ulcer. The ulcer staging can be documented by either the provider or the clinician. However, the documentation must be consistent.

**Pressure Ulcer Case Scenario: An established patient with a pressure ulcer of the right heel now presents with necrosis of soft tissue involving the tendon. The tissues of this ulcer continue to deteriorate due to non-compliance of pressure relief. This pressure ulcer is now at stage 4.**

**Table 7**

Anatomical Location	Laterality
Elbow	Right Left Unspecified—should not be a coding option selected in a wound care setting
Back	Right upper or lower Left upper or lower Unspecified part of back—should not be a coding option selected in a wound care setting
Hip	Right Left
Sacral region does not have any additional specificity other than to include staging	Unspecified—should not be a coding option selected in a wound care setting
Buttock	Right Left Unspecified—should not be a coding option selected in a wound care setting
Contiguous site of back, buttock, and hip	Document ulcer staging No laterality required with this code
Ankle	Right Left Unspecified—should not be a coding option selected in a wound care setting
Heel	Right Left Unspecified—should not be a coding option selected in a wound care setting
Other site –head	Document ulcer staging No laterality required with this code
Other site not listed above	Document ulcer staging No laterality required with this code
Unspecified site	Should not be a coding option selected in a wound care setting

**NOTE:** Pressure ulcer ICD-10-CM code descriptions do not require reporting tissue severity as with non-pressure ulcers. However, laterality and staging of the pressure ulcer are required. The broad ICD-10-CM category for pressure ulcers begins under L89.XXX and is broken down by anatomical locations and staging. Anatomical descriptions for pressure ulcers are listed in Table 7.

Staging descriptions for pressure ulcers are listed in Table 8.

**Table 8**

Stage 1 ulcers consist of but are not limited to:	Intact skin with non-blanchable redness of localized area Usually located over bony prominences
Stage 2 ulcers consist of but are not limited to:	Partial thickness loss of dermis Shallow open ulcer with a red-pink wound bed No slough in wound bed May also present as intact or open/ruptured serum-filled blister Bruising indicates suspected deep tissue injury
Stage 3 ulcers consist of but are not limited to:	Subcutaneous tissue may be visible No exposed muscle/tendon/bone Slough may be present May include undermining & tunneling Shallow ulcer on nose/ear/occiput/malleolus due to lack of subcutaneous tissue
Stage 4 ulcers consist of but are not limited to:	Full thickness loss with exposed muscle/tendon/bone Slough/eschar may be present on some parts of wound bed Often includes undermining and tunneling May extend into muscle/supporting structures/fascia/tendon/joint capsule

**NOTE:** ICD-10-CM coding requires documentation of laterality (right or left or both) along with the pressure ulcer staging to avoid unspecified codes. Without specifying laterality, the code description includes “unspecified heel.” The codes in Table 9 note the documented location of the right heel along with the applicable stages. The code that applies to the pressure ulcer described in the case scenario, a stage 4 pressure ulcer of the right heel, is highlighted.

**Table 9**

ICD-10-CM Code	ICD-10-CM Code Description
L89.610	Pressure ulcer of right heel, unstageable
L89.611	Pressure ulcer of right heel, stage 1
L89.612	Pressure ulcer of right heel, stage 2
L89.613	Pressure ulcer of right heel, stage 3
L89.614	Pressure ulcer of right heel, stage 4
L89.619	Pressure ulcer of right heel, unspecified stage—should not be a coding option selected in a wound care setting

**Arterial Ulcer**

Lower extremity arterial disease (LEAD) is most commonly caused by atherosclerosis, particularly in patients with high risk lifestyle factors such as advanced age, diabetes mellitus, hyperlipidemia, hypertension, obesity, and smoking as well as a family history of cardiovascular disease. With ICD-10-CM, a patient with an arterial ulcer will require more than one diagnosis code to accurately reflect the level of acuity with complexity of care and intensity of services. However, these new ICD-10-CM codes for arterial ulcers encompass the following details, which are required in a provider’s clinical documentation:

The disease of atherosclerosis

- Anatomical specific location
- Laterality (right or left)
- Type of arteries involved
  - Native arteries
  - Unspecified type of bypass graft(s)
  - Autologous vein bypass graft(s)
  - Non-autologous biological bypass graft(s)
  - Other type of bypass graft(s)
  - Type of bypass vein

Arterial ulcers have associated common characteristics that include:

- Wound margins well-demarcated and
  - Wounds are often full-thickness, deep, and painful
  - Wound bed may have nonviable gray-yellow tissue or eschar with a pale coloration due to compromised blood flow
  - Gangrene may be present
- Typical locations such as the following:
  - Ankles
  - Feet, including toes, tip of toes, or over the phalangeal heads
  - Lateral malleoli
  - Areas of trauma
- The severity of the ulcer requires an additional code

**Arterial Ulcer Case Scenario: A new patient presents with right lower extremity ulcers on the right great and third toes. The physician documents a diagnosis of atherosclerosis of native vessel, which had a non-autologous biological bypass graft inserted during a femoropopliteal bypass performed on the right leg to improve circulation six weeks prior. The right great toe has a gangrenous ulcer with necrotic bone exposed, and the right third toe has eschar present over half of the ulcer with subcutaneous tissue exposed. Both ulcers will require debridement and an aggressive wound care treatment regimen.**

The following ICD-10-CM code options listed in Tables 10 through 12 are based on the details contained in the provider’s documentation. The applicable codes are highlighted in blue.

**Table 10**

Diagnosis: Atherosclerosis of native arteries of right leg with ulceration of other part of foot	
ICD-10-CM Code	ICD-10-CM Code Description
I70.261	Atherosclerosis of native arteries of extremities with gangrene, right leg
I70.262	Atherosclerosis of native arteries of extremities with gangrene, left leg

**Table 11**

Diagnosis: Atherosclerosis of native arteries of right leg with ulceration of other part of foot	
ICD-10-CM Code	ICD-10-CM Code Description
I70.235	Atherosclerosis of native arteries of right leg with ulceration of other part of foot (toes)
I70.245	Atherosclerosis of native arteries of left leg with ulceration of other part of foot (toes)

**Table 12**

Diagnosis: Severity of each ulcer is to be coded separately	
ICD-10-CM Code	ICD-10-CM Code Description
L97.514	Non-pressure chronic ulcer of other part of right foot with necrosis of bone
L97.512	Non-pressure chronic ulcer of other part of right foot with fat layer exposed

Based on the case scenario, the following ICD-10-CM codes should be selected for this patient:

1. I70.261—Atherosclerosis of native arteries of extremities with gangrene, right leg
2. I70.235—Atherosclerosis of native arteries of right leg with ulceration of other part of foot (toes)
3. L97.514—Non-pressure chronic ulcer of other part of right foot with necrosis of bone (reason for the wound care department encounter)
4. L97.512—Non-pressure chronic ulcer of other part of right foot with fat layer exposed (reason for the wound care department encounter)

## Conclusion

This article has focused specifically on examples related to the most common wounds encountered in the clinical setting, along with the clinical documentation elements necessary to avoid the use of unspecified codes. Keep in mind, when treating wounds it may be necessary to report the wound with a level of severity of tissue destruction code. This L97.XXX code (level of wound severity) supports your level of acuity with intensity of services and complexity of care decisions necessary, such as additional workup evaluations, tests, procedures, and debridements, to provide your patient with the appropriate care. It is the details of your documentation that will tell the most complete and accurate story of your patient's medical care, so be sure to make the necessary documentation preparations to survive the transition to ICD-10-CM this fall.

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## Clinic In Focus

# Swedish Edmonds Center for Wound Healing and Hyperbarics

Continuing our series of interviews featuring outstanding hyperbaric and wound care centers around the world, we spoke with Susan Lowber at Swedish Edmonds Center for Wound Healing and Hyperbarics, a UHMS-accredited facility in Edmonds, Washington.

### How long has your clinic been in business?

The hospital site has been here for over 50 years and has offered wound healing services for over 10 years. We expanded to include hyperbaric medicine in the fall of 2008.

### How has seeking UHMS accreditation affected your clinic?

In order to receive accreditation, our facility underwent an intense review by a team of UHMS experts. The team examined staffing and training, equipment installation, operations and maintenance of the facility, patient safety, and standards of care. Accreditation by the UHMS recognizes a center's commitment to patient care and safety and is validation that the center meets the most rigorous industry standards.

The accreditation gives confidence to both the patient and other community providers that we are providing excellent care to the patient while upholding the highest safety standards and clinical knowledge. We are one of only two centers in the state of Washington that are accredited.

### What are the most common indications treated at your clinic?

In the wound clinic: leg ulcers (both venous and arterial), wounds resulting from peripheral edema, diabetic foot ulcers, post-surgical wounds, decubitus ulcers, and infected wounds.

For HBO treatments: severe diabetic foot ulcers, compromised surgical flaps and grafts, medical conditions resulting from radiation injury (soft tissue radionecrosis and osteoradionecrosis), and refractory osteomyelitis.

### What is the most memorable treatment success story that has come out of your clinic?

We have had many remarkable outcomes, particularly with hyperbaric medicine patients. But recently we had a patient who had complications from radiation therapy after treatment for esophageal cancer, which resulted in soft tissue radionecrosis (STRN). She developed an esophageal cutaneous fistula, an opening where fluids would leak through her throat, and she was unable to swallow or eat and had constant drainage of saliva from her neck. After 50 hyperbaric oxygen therapy treatments, her fistula healed and was no longer draining. She was ecstatic as she was now able to eat and drink again after not being able to do so for several months.

### Do you work with a management company?

No, we are independent.

## CLINIC DETAILS

Swedish Edmonds Center for Wound Healing and Hyperbarics

21600 Highway 99, Suite 150, Edmonds, WA 98026

<http://www.swedish.org/services/wound-healing-hyperbarics-edmonds>

(425) 673-3380

Date of UHMS Accreditation: January 2013

Number of chambers: 2

Chamber type: Monoplace

On staff: 2 full-time physicians / 4 full-time RNs / 1 safety supervisor CHT / 1 CHT / several part-time RNs

Dr. Peter Ro, Medical Director



*The staff of the Swedish Edmonds Center for Wound Healing and Hyperbarics in Edmonds, Washington.*

## If you had to pick one thing to attribute your clinic's success to, what would it be?

Caring. Our experienced clinical staff genuinely cares about the patients. Not only do we address the wounds, we also look at the condition of the patient as a whole for multiple factors that can affect wound healing, including nutrition, medications, insufficient blood flow to arteries, and other illnesses. When appropriate, our wound care team will refer patients for studies and collaborate with other medical specialties to ensure our patients

are receiving comprehensive, quality care. We also assist patients in obtaining social services and home healthcare if they seem unable to make arrangements for themselves. We have many satisfied patients who have given us excellent reviews upon completion of their treatments.

## What is one marketing recommendation that you can make to help clinics increase their patient load?

We believe that developing good relationships with our referring providers are key to growth for our center and would also benefit other wound healing centers. Both of our physicians regularly and proactively engage in personal communication with the referring providers, whether by phone or face-to-face contact, which is always appreciated by the medical community.

## Are there any additional questions you'd like to answer, or is there any other information about your clinic you would like to showcase?

Both of our physicians are board certified in undersea and hyperbaric medicine. One of our physicians, James Wright, MD, FACS, is a board certified plastic surgeon and has also been involved in research in hyperbaric medicine during his tenure with the US Air Force. Darren Mazza, our Hyperbaric Safety Supervisor, has been the recipient of the monthly "Most Valuable Player" (MVP Award) from our hospital, Swedish Edmonds. He has also written several articles on hyperbarics in the clinical setting.

Last May, we moved into a state-of-the-art facility near our hospital, with 10 treatment rooms located on the ground floor level for ease of access for our patients. And finally, our patient satisfaction numbers are high: 92.9% in 2014 and 96.0% to date for 2015.

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## Ask the Experts: What is the Current Perspective Regarding Supervision of Hyperbaric Dives by Nurse Practitioners?

From The Wound Care Education Partners blog, *Rx Pad*

**Question:** “I would be interested in the current perspective regarding supervision of hyperbaric dives by nurse practitioners.

1. Is it safe?
2. What preparation for supervision is appropriate?
3. How many centers across the nation are using NPs?
4. What are the pros and cons to having an NP supervise dives?

Other policy or procedure recommendations for NP supervision of dives.”

*Question from Carol, BSN, RN and student of Wound Care Education Partners.*

### Our Experts Offer the Following Answers:

**Question 1.** Is it safe?

**Answer.** It is safe with appropriate preparation. As with other disciplines within medicine that utilize nurse practitioners and physician assistants, the provision of safe care is a function of proper training and sufficient supervision. The UHMS (Undersea and Hyperbaric Medical Society) and the National Board of Diving and Hyperbaric Medical Technology (NBDHMT) consider mid-level practitioners qualified to safely supervise Hyperbaric Oxygen Therapy (HBOT) so long as those criteria are met (see position statements below).

**Question 2.** What preparation for supervision is appropriate?

**Answer.** The UHMS position statement provides the following information:

The Non-Physician provider specific recommendations:

a) The UHMS supports the on-site supervision of hyperbaric oxygen therapy by a nurse practitioner or physician assistant if each of the following conditions is met:

i. The supervising physician meets the UHMS recommendations for physician attendance as per UHMS guidelines.

ii. The supervising physician is immediately available to the Hyperbaric Medicine Department as specified by applicable government regulations.

iii. The nurse practitioner or physician assistant has obtained appropriate specialty certification through the NBDHMT as a Certified Hyperbaric Registered Nurse (CHRN) or Certified Hyperbaric Technologist (CHT), or international equivalent.

The NBDHMT position statement provides the following information:

a) HBO must be directly supervised by a physician (or nurse practitioner/physician assistant where permitted by prevailing credentialing and regulatory standards) who is formally (UHMS or other authoritative body) trained in hyperbaric medicine, involving face-to-face classroom versus online setting. Such supervision should extend to:

- a. Assessment of suitability for HBO therapy
- b. Determination of risk-benefit profile
- c. Interpretation of any related diagnostic testing
- d. Generation of a therapeutic dosing profile
- e. Evaluation of subsequent clinical course, and
- f. Management of any related side effects and complications

Further, the hyperbaric physician must be on the premises and immediately available to the chamber facility at all times that the chamber(s) is occupied. Immediately available would meet the intent of this Position Statement if the physician could arrive at the chamber facility within five minutes of being summoned and in doing so, would not place in jeopardy any other patient presently under his/her care.

It is the duty of hyperbaric nursing and technical personnel to safely implement ordered therapy and closely monitor patients during their treatments. Should a patient voice complaints or manifest signs suggesting an unanticipated change in status, considered to be hyperbaric related or otherwise, the hyperbaric physician should be immediately notified. Importantly, hyperbaric nursing and technical personnel do not assume any of the physician responsibilities noted as complaints or manifest signs suggesting an unanticipated change in status, considered to be

hyperbaric related or otherwise, the hyperbaric physician should be immediately notified. Importantly, hyperbaric nursing and technical personnel do not assume any of the physician responsibilities noted above and cannot initiate hyperbaric treatment without patient-specific hyperbaric physician signed medical orders.

**Question 3.** How many centers across the nation are using NPs?

**Answer.** At this time, there are only a few.

**Question 4.** What are the pros and cons to having an NP supervise dives?

**Answer.** As with many pro/con discussions, the determination of which elements fit into a given category hinges largely on one's perspective. Any substantive answer must acknowledge the parties represented in this discussion, which include: patients, mid-level providers, physicians, and administration. Each group will see this topic through a unique lens and there are opinions that won't be represented here. The points made below are not listed as pro or con for those reasons.

**Patient:** Some may prefer physician-level supervision, but there shouldn't be any difference in the actual treatment provided. There are no cost savings to the patient subsequent to having mid-levels supervise HBOT.

**Mid-level:** Enjoyment of engaging in hyperbaric medicine, expanded scope of practice, potential for additional income.

**Physician:** Potential for reduced work hours and revenue loss due to less time working in HBO, increased requirement for direct supervision of mid-levels and immediate availability. Responsibility for mid-levels requires additional work and liability which are frequently not associated with added compensation.

**Administration:** Potential for greater scheduling flexibility as more people can supervise treatments. Mid-levels are paid less but physician supervision and immediate access may offset these overhead reductions.

**Question 5.** Other policy or procedure recommendations for NP supervision of dives.

**Answer.** One recommendation is to include a nurse practitioner job description per NP scope of practice guidelines. In addition to broadening the scope of practice, hyperbaric specific training, experience, and competency standards are necessary for credentialing purposes within a healthcare facility. While stand-

alone wound care centers may not require that level of documentation, proof of training and experience is part of the UHMS and NBDHMT position statements.

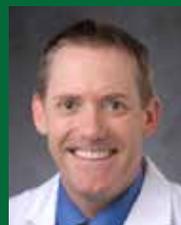
**References:** Expert answers provided by Laura Josefsen, RN and Nick Bird, MD, MMM

This article was previously published in *Wound Care & Hyperbaric Medicine* magazine Volume 5 Issue 1.

## About the Experts



**Laura Josefsen, RN, ACHRN,** is on the UHMS (Undersea and Hyperbaric Medical Society) Board of Directors as the current Nurse Representative on the Associates Council, and has been a member of the UHMS Accreditation Team as a nurse surveyor since its inception. She is a founding member of the (BNA) Baromedical Nurses Association, served as president from 1996-1998, and has been active on the executive board since 1985. She served for many years as an Executive Board Member of the NBDHMT (National Board of Diving and Hyperbaric Medical Technology), and is a previous member of the BNA Certification Board. She is currently on the Board of Directors of the TMAA (Texas Medical Auditors Association). She is a member of the Undersea and Hyperbaric Medical Society Associates, former member of DAN (Diver's Alert Network), and HTNA (Hyperbaric Technologists and Nurses Association) of Australia. She has numerous publications and is an internationally recognized speaker in the field of hyperbaric medicine.



**UHM Nick Bird, MD, MMM,** is a fellowship-trained, board-certified hyperbaric physician. He is the past CEO and Chief Medical Officer for Diver's Alert Network (DAN) in Durham, North Carolina. Prior to his position with DAN, he served as the Medical Director of Hyperbaric Medicine at Dixie Regional Medical Center in St. George, Utah. Additionally, Dr. Bird served in the United States Air Force as a flight surgeon and received his initial training and experience in hyperbaric medicine at Travis AFB while working as the Deputy Flight Commander of the hyperbaric/wound center. He was honorably discharged with the rank of Major, but not before serving as the final Commander of the Base Hospital in Jordan during Operation Iraqi Freedom.

Earning his Bachelor of Arts degree from the University of California at Santa Cruz in 1992, Dr. Bird went on to earn a medical degree from the Royal College of Surgeons in Ireland in 1999. He completed a residency in family medicine under the University of Washington and a fellowship in hyperbaric medicine at the University of California at San Diego. In addition to his fellowship training, he attended the US Air Force hyperbaric course, the International ATMO program, and the NOAA Diving Medical Officer course.

Dr. Bird has remained a clinical instructor in hyperbaric medicine and wound care. He is an active member of the UHMS, works as part of the UHMS accreditation team, has authored articles and book chapters in diving medicine, is a member of the UHMS Diving Medicine Committee, and he has both attended and presented at multiple conferences on diving and hyperbaric medicine. Additionally, he was the course director for the DAN DMT course and author of the revised series of DAN educational programs.

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## Give Me a Break

### Air Breaks During Hyperbaric Treatment

Darren Mazza, EMT, CHT

When treating patients with hyperbaric therapy in the monoplace chamber using 100% oxygen, the supervising hyperbaric physician may order air breaks to be provided to the patient at certain intervals during the treatment.

What is an air break? During a typical air break, the patient will be instructed to breathe medical air from a mask at certain intervals during the treatment for around five minutes at a time, depending on the provider's written order. In my past working as an EMT, any time we had a patient with an inhalation injury such as from smoke inhalation or exposure to chemical fumes, the primary treatment guideline was to immediately get the patient on high flow oxygen and into a clean air environment. A saying we used to use was "the solution to pollution is dilution." When providing air breaks to a patient, you dilute the oxygen concentration the patient breathes in the chamber from 100% down to around 21% via demand valve mask. This switch to air will in turn reduce the risk of CNS oxygen toxicity by giving the patient a break from the 100% oxygen concentration in the chamber.

The use of air breaks during treatment is dependent upon two key CNS oxygen toxicity potentiating factors: treatment depth/time and predisposing factors noted in the patient's medical history.

1. Treatment depth/time: Studies have shown that CNS oxygen toxicity occurrences are 1.3 in 10,000 patients at treatment depths >2.0 ATA.
2. Prior medical history: Patients with hyperthyroidism or who are CO<sub>2</sub> retainers are at greater risk for CNS oxygen toxicity due to vasodilation in the brain. Seizure disorders and certain medications, such as epinephrine and steroids, may also increase susceptibility.

It is important to be aware of the signs and symptoms of CNS oxygen toxicity, which are represented in the below acronym.

**CON – Convulsions (seizure)**

**V – Vision changes**

**E – Ears (tinnitus)**

**N – Nausea**

**T – Twitching, facial**

**I – Irritability**

**D – Dizziness**

Keep in mind that any of these symptoms can occur at any time, and the patient may only present with one of the symptoms such as nausea, dizziness, or even irritability. This is why it is essential to observe the patient during the entire hyperbaric treatment. I have found that the more I observe each patient during the course of the treatment, the easier it is for me to notice any changes in his or her demeanor, such as increased irritability. At the first sign of any symptoms of CNS oxygen toxicity during a hyperbaric treatment, the patient will be instructed to breathe from the mask while being brought to surface, and treatment will be aborted. If the patient begins to have a seizure, the treatment depth would not change until the provider determines the patient to be in a postictal state.

When providing patients with instruction on the use of the air break equipment, it is extremely important that they demonstrate competency in using the equipment so they can adequately provide themselves with an air break during treatment. Before every treatment, I instruct the patient to take two breaths from the air break line and mask and confirm two things: that the mask provides an

adequate seal and the regulator provides proper air flow, allowing the patient to breathe freely with no resistance. Occasionally, when a patient is instructed to begin an air break, the patient places the mask carelessly without providing a proper seal to their mask. Risk of oxygen toxicity to the patient will be increased if they are not properly utilizing the equipment by sealing the mask and breathing appropriately from the regulator. Again, it is important to observe the patient during treatment to ensure the correct use of the equipment and to note any changes in demeanor that may point to possible CNS oxygen toxicity.

### Final Note

Keeping patients safe during their hyperbaric treatment is my absolute focus; in the hyperbaric environment there is no room for complacency. Providing proper education to a patient about possible risks that may occur during treatment and corresponding safety guidelines will encourage the patient to become proactive in his or her care and understand the importance of correctly using equipment, such as air break equipment, during treatment.

### About the Author

*Darren Mazza is the CHT and Safety Director at the Center for Wound Healing and Hyperbarics at Swedish/Edmonds, located in the greater Seattle area. He has 20 years of experience in healthcare, which includes 8 years as an EMT in the greater Sacramento region. Darren also worked as a preceptor trauma tech in a Sacramento hospital for several years. After leaving California and moving to Idaho in 2005, his hyperbaric career began after becoming the department head of an outpatient wound care and hyperbaric center. His hobbies include fly fishing and fly tying.*

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## FOXO1CATCHER

### An Interview with Dr. Dana Graves on the Role of FOXO1 in Diabetic Patients

**W**CHM had the good fortune to land an interview with Dr. Dana Graves, who led a team of researchers investigating the FOXO1 molecule's role in promoting healing in patients with chronic wounds and diabetes. We interviewed him about information on his recently published research. Dr. Graves is a professor in the Department of Periodontics, Vice Dean for Scholarship and Research, and Director of the Doctor of Science in Dentistry Program for Penn Dental Medicine under the umbrella of the University of Pennsylvania. Penn Dental Medicine is an ivy-league institution with a deep history in forging precedents in dental education, research, and patient care.

**WCHM:** Dr. Graves, can you please tell us about your background and what led to your interest in mucosal wound healing and diabetes?

**DG:** Well, I am a clinician. I am periodontist and I treat patients. One of the striking things about periodontal disease is that it is enhanced or increased by diabetes. Diabetes has effects on different tissues. As a periodontal surgeon, I place implants and perform surgeries where the wound healing response is impaired by diabetes. I became interested in the molecular mechanisms by which diabetes affects different tissues. I have studied bone as well as soft tissue healing, and I've been interested in how diabetes slows the healing process.

**WCHM:** Would you please explain your research of diabetes and non-healing wounds and your findings?

**DG:** In diabetes, a non-healing wound results from a number of events. In the early aspects, the wound heals slowly. A slower healing wound then can become colonized by bacteria, which sets up a non-healing situation. The aspect that I'm interested in is the initial aspect, which is the diabetic wound heals more slowly. I'm interested in why the wound heals more slowly, and I have not focused on the second aspect, which is why a slowly healing wound converts to a non-healing wound.

So think of it as two processes, and they're linked together. The more slowly healing wound allows a situation to develop where the wound becomes colonized and doesn't heal.

**WCHM:** What do your findings suggest is the link between FOXO1 and diabetes?

**DG:** We were originally examining FOXO1 in normal wounds. And we deleted the FOXO1 gene keratinocytes, which are the cells that form the outer layer of skin and are involved in closing the wound. The goal of wound healing is to close the wound as rapidly as possible. And the cells that are really essential for that are the keratinocytes, which are the outer layer. So we deleted FOXO1 in this particular layer of cells, and in the normal wound we found that when this particular gene was deleted, heal-

ing was much slower. This shows that the gene is needed because when it was removed, the healing got worse.

Then we found out what FOXO1 was regulating and we figured out the mechanism. I was then interested in finding out what might happen in a diabetic wound. So we created a wound on a diabetic animal and it healed more slowly, which is what you would expect.

Then we deleted the FOXO1 in the diabetic animal and the odd thing was that it speeded up healing. This was a real surprise. So you delete FOXO1 in the normal wound and it behaves like a diabetic wound. You delete FOXO1 in the diabetic wound and it behaves like a normal wound.

So it seems like this particular factor is a good factor in a normal wound but becomes a bad factor in a diabetic wound. The reason we say this is that when you delete this particular gene in the outer layer of cells, healing in the diabetic wound speeds up.

So our interpretation of this observation is that the FOXO1 molecule is interfering with healing because when you remove it, healing is faster and occurs at a more normal pace. What we then establish from this work is that FOXO1 plays an important role in promoting healing under normal circumstances, but in diabetic situations (or diabetic healing), this same molecule becomes a problem. So it becomes a “bad apple.”

We then went to figure out how this occurs.

**WCHM:** Can you talk about what these findings mean for the future of diabetes care?

**DG:** When you take a molecule that has gone from being a good factor to a bad factor, what you normally do is inhibit it under conditions where it's harmful. This would suggest that if you can in-

hibit it in the diabetic wound, healing would be improved.

The next step is to try and develop a therapeutic factor. We have so far reduced the activity of the gene through genetic manipulation, so now we need to test what the impact of inhibiting it with a small molecule would be. These are experiments we have on the drawing board and will carry out very soon.

**WCHM:** I think you may have answered our next question. What are your future research plans for FOXO1?

**DG:** The progression is to now perform experiments in a large animal model. We have established the concepts in mice and in order to do pre-clinical trials, we need to move to a larger animal. We plan to do these experiments in a larger animal to see if we applied this therapeutic factor, which is to inhibit FOXO1 with a small molecule, can we improve the healing environment for the cells.

**WCHM:** A number of our readers practice both wound care *and* hyperbaric medicine, so can you tell us a little about the impact your research findings

might have on hyperbaric medicine in the future and how this could affect hyperbaric practitioners?

**DG:** Well, the link between FOXO1 and wound healing is pretty clear, and it is possible that there are links to the hyperbaric question you asked. However, at this point they're not known, and I wouldn't be able to provide any real insight to that.

**WCHM:** Is there anything additional about your research and findings that you would like to address?

**DG:** Yes, well there is something that I think is quite interesting. It's a bit technical. And that is when we looked to see what caused FOXO1 to change from being a good factor that promotes healing to a bad factor that inhibits it, we found that glucose itself

“We deleted the FOXO1 in the diabetic animal and the odd thing was that it speeded up healing . . . So you delete FOXO1 in the normal wound and it behaves like a diabetic wound. You delete FOXO1 in the diabetic wound and it behaves like a normal wound.”

caused that change. There is something about the high glucose environment that modifies the activity of FOXO1 which determines its particular consequences.

**WCHM:** Dr. Graves, if any of our readers would like to find more details or follow the research you're doing, where can they get more information?

**DG:** They could look up my name, Dana Graves, in *PubMed*, which is a website run by the government that lists all the published articles. I have a follow-up study coming out in April in the *Journal of Cell Biology* that describes the impact on skin. So the first set of studies were on mucosal wound healing and the second set of studies, which we'll publish in April, deals with skin healing. And I think that it's interesting that this particular molecule FOXO1 is important to both types of wounds.

**WCHM:** Do you have any plans for presentations at upcoming conferences that you'd like to tell our readers about for anyone who might be interested in connecting with you personally and hearing more about your research?

**DG:** Yes, I'll be presenting on this in the June 2015 Diabetes Conference in Boston.

**WCHM:** Dr. Graves, thank you for joining us today and sharing your research and findings with our readers. We look forward to connecting with you again and finding out more about your research and your future studies.

**DG:** Thank you very much for having me and having this discussion.

Dr. Grave's article is published in *Diabetes*.

Xu F, Othman B, Lim J, Batres A, Ponugoti B, Zhang C, Yi L, Liu J, Tian C, Hameedaldeen A, Alsadun S, Tarapore R, Graves DT. Foxo1 inhibits diabetic mucosal wound healing but enhances healing of normoglycemic wounds. *Diabetes*. 2015 Jan; 64(1): 243-56. doi: 10.2337/db14-0589. Epub 2014 Sep 3.

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**CLICK HERE** to listen to the podcast of the interview with Dr. Graves.

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# Keeping Things Simple and . . . Speedy A Dozen Paradigms About Toe Tenotomies

**Michael B. Strauss, MD, FACS, AAOS**  
Medical Director Hyperbaric Medicine  
Long Beach Memorial Medical Center

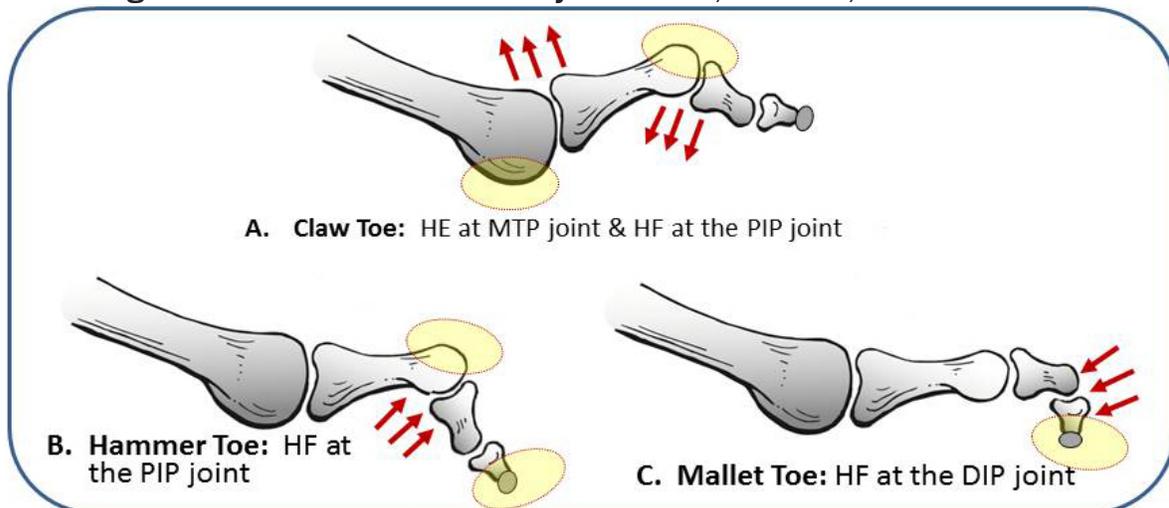
**Steven La, DPM**  
Resident Physician, Long Beach Memorial Medical Center

## INTRODUCTION

While toe deformities may appear to be unimportant, they can have serious consequences. This is the situation especially in patients with diabetes who have neuropathies. A small ulcer can evolve to osteomyelitis of the phalanges, septic joints of the toes, ascending tenosynovitis and progressive necrotizing soft tissue infections. Mal perforans ulcers are invariably the consequence of underlying bone and joint deformities. The pathology in

the toes is no different than when they occur in the foot and ankle.<sup>[1]</sup> Consequently, early attention to correction of gnarled toes is an essential proactive wound prevention measure for the insensate foot that has more than cosmetic ramifications. This article describes the anatomy of claw, hammer, and mallet toes, explains the pathophysiology that leads to forefoot and toe wounds, and provides a dozen paradigms about tenotomies to manage the axial (toes in-line with the metatarsals) deformities of the toes.

**Figure 1: Bone and Joint Anatomy of Clawed, Hammer, and Mallet Toes**



**Legend:** Axial deformity classification of toes based on levels of joint involvement. Table 1 explains the pathophysiology causing the deformities. Red arrows indicate levels of joint contractures. Yellow oblongs indicate sites for ulcerations. The clawed toe is the cause of the malperforans ulcer under the metatarsal head—note the dorsal subluxation of the proximal phalanx which “drives” the MT head downward.

**Key:** DIP = Distal interphalangeal, HE = Hyperextension, HF = Hyperflexion, MTP = Metatarsal phalangeal, PIP = Proximal interphalangeal joint

**CLASSIFICATION OF AXIAL TOE DEFORMITIES**

The classification of axial toe deformities is straightforward (Figure 1). A hammer toe occurs with hyperextension at the metatarsophalangeal joint and flexion at the proximal interphalangeal joint with or without extension of the distal interphalangeal joint. A claw toe is present when the metatarsophalangeal joint is hyperextended and the proximal interphalangeal joint is flexed, similar to a hammer toe. However, the distal interphalangeal joint may also be flexed. In a mallet toe, the metatarsophalangeal joint and proximal interphalangeal joints are unaffected, but the distal interphalangeal joint is flexed.

tarsophalangeal joints and extend the toes at the interphalangeal joints, hyperextension of the metatarsophalangeal joints occurs due to over-pull of the toe extensor muscles. Meanwhile, over-pull of the flexor muscles cause toe interphalangeal joint contractures (Table 1).

Consequences of the muscle imbalances include:

- 1) Retraction of the toes proximally onto the dorsum of the foot,
- 2) Dorsal subluxation of the proximal phalanges over the metatarsal heads,
- 3) Depression of the metatarsal heads into the forefoot fat pad with muscle activity, and
- 4) A malperforans ulcer develops as the plantar surface of the metatarsal head erodes from inside-to-outside with weight

**Table 1: Pathology and Management of Axial Toe Deformities**

Diagnosis	Pathology / Deformity	Management Minimally Invasive
Claw Toe	1) Hyperextensions at the metatarsophalangeal joint, 2) Hyperflexion at the proximal interphalangeal joint and 3) possibly at the distal interphalangeal joint	Releases of the flexor & extensor tendons; Joint manipulations
Hammer Toe	Hyperflexion at the proximal interphalangeal joint and possibly at the distal interphalangeal joint	Releases of the flexor tendons & joint manipulation
Mallet Toe	Hyperflexion at the distal interphalangeal joint	Releases of the flexor tendons & possibly joint capsule
Hyperextended Toe	Hyperextension at the metatarsophalangeal joint; intrinsic muscles maintain interphalangeal joint extension	Releases of the flexor tendons & possibly joint capsule

**PATHOPHYSIOLOGY OF AXIAL TOE DEFORMITIES**

The pathophysiology of the toe deformities that leads to forefoot and toe wounds results from neuropathy. The essential problem is a motor neuropathy where fine muscle balance between flexor and extensor muscles is altered or lost. With loss of the intrinsic muscles which flex the toes at the meta-

bearing because of the deformity, if not corrected (Figure 2). If the intrinsic muscles of the foot continue to function, but there is over-pull of the extensor muscles, the toes may remain straight, but be hyperextended at the metatarsophalangeal joints.

The hammer and mallet toe deformities occur

**Figure 2: Pathophysiology of Clawed, Hammer, and Mallet Toes**



**Legend:** Of note are the clawed toes with hyperextension at the metatarsal phalangeal joints and hyperflexion at the proximal interphalangeal joints.

Because of the muscle imbalances, the toes have been retracted proximally onto the dorsum of the foot resulting in dorsal subluxation of the metatarsal phalangeal joints and pulp pads of the toe tips not touching the floor when standing.

because of over-pull of the flexor tendons and loss of the intrinsic muscle abilities to extend the interphalangeal joints. If the problem lies primarily with the short intrinsic flexors of the toes, the hammer toe deformity occurs and causes the toe tip to “drive” into the sole of the shoe. The consequence is a pressure sore at the tip of the toe. This typically progresses to a penetrating ulcer to the distal tuft and osteomyelitis of this structure (Figures 2, 3, & 4).

Over-pull of the long flexor muscles of the toes results in mallet deformity. When the combination of the above problems occur, the hyperflexed proximal interphalangeal joint (PIP) in association with the hyperextended metatarsophalangeal joint causes a pressure sore over the apex of the PIP joint (Figure 5). With progression, the ulceration erodes into the joint causing a septic joint and osteomyelitis. If unchecked the infection can track proximally along the tendon sheaths resulting in ascending tenosynovitis and progressive necrotizing soft tissue in-

fection.

**Figure 3: Toe Tip Wound from Hallux Malleolus**



**Legend:** Because of the clawed hallux, the toe tip was “driven” into the patient’s footwear. This resulted in a diabetic foot ulcer that penetrated to the distal tuft of the hallux and caused osteomyelitis with erosion of the bone (red arrows). A partial toe amputation was required to manage this problem.

There as also a mallet deformity of the second toe as well as fungus infection of the hallux toenail.

Usually, a sensory neuropathy is associated with the motor neuropathy, especially in the patient with diabetes mellitus. However, other motor neuropathies have hereditary causes such as Charcot Marie Tooth disease. Acquired causes (such as the results of trauma and/or demyelinating causes, such as amyotrophic lateral sclerosis), may not have sensory neuropathy components to them. While a

**Figure 4: Toe Tip Ulceration from Clawed Toe**



**Legend:** The reason for the toe tip ulceration is obvious from the examination. The clawed toe increased the contact pressure when standing and walking between the toe tip and the underlining supporting surface enough to generate a pressure sore over toe tip.

Note the flexor creases of the clawed toe are obscured because of the flexion contractures. We label this clinical finding the “hidden crease” sign.

sensory neuropathy does not cause a deformity, it may delay the diagnosis of the deformity. The absence of pain may sidetrack the patient from seeking care until a complication arises such as obvious infection at the deformity site or systemic sepsis occurs. Consequently, any patient with sensory neuropathy in the feet and associated toe deformities should be informed of the need for proactive interventions anytime an impending pressure sore is observed in the forefoot and/or toes. Also, it is essential that all foot-care providers are also aware of this information and counsel their patients accordingly.

#### **PARADIGMS FOR EVALUATION AND MANAGEMENT OF AXIAL TOE DEFORMITIES**

With the above considerations, and experiences with over 400 tenotomies of toe tendons (done by the first author) in the past dozen years, we want to reiterate some of the above information as well as share our “pearls” in the evaluation and

management of axial toe deformities with the readers of *Wound Care & Hyperbaric Medicine*. A large review experience of toe tenotomy surgeries can be found in Tamir, et al’s article in *Foot & Ankle International*.<sup>[2]</sup> Whereas some of the procedures need to be done in the operating room, we do the majority of tenotomies in our outpatient wound care programs. The following are 12 paradigms that relate to axial toe deformities and their management with tenotomies:

**1. Often toes are retracted proximally onto the dorsum of the foot due to over-activity of the long extensor tendons.** This leads to downward pressure on the metatarsal heads and is a precursor to mal perforans ulcerations (Figure 2). The retracted toe sign is an indication for toe extensor releases (Figure 4).

**2. Manipulation of interphalangeal joint contractures should always be a component of the tendon release procedures (Figure 6).** Occasionally, the manipulation results in avulsion of skin at flexor creases of the interphalangeal joints. All of these superficial wounds have healed without incidence.

**3. In some instances, extensor tendon releases are difficult to perform in the presence of edema, scar, or hidebound skin.** In these cases, we perform these procedures in the operating room, as described in the following text box.

Two centimeter incisions are made and carried through the subcutaneous tissue level in the intermetatarsal spaces just proximal to the metatarsal necks. The extensor tendons are then captured with a curved hemostat brought to the skin surface and incised under direct visualization with a scalpel, scissors, or electric cautery.

Much variation in the extensor tendon anatomy has been observed, often requiring release of more than one tendon for each toe. The short incisions are usually closed with small nylon sutures or staples (if already on the field from closing a wound at a different site).

**4. Releases of extensor hallucis longus tendons**

**for managing clawing of the great toes have generally not been effective in managing mal perforans ulcerations of these toes.** In such cases, the underlying bony deformities necessitate sesamoid planning or excision.

**Figure 5: Toe Tip Ulceration from Clawed Toe**



**Legend:** Serous crust over the apex of the clawed second toe proximal interphalangeal (PIP) joint.

When the crust was lifted, a tract to the septic PIP was identified.

Unfortunately a partial second ray resection was required. Had a proactive extensor tenotomy been done before the ulceration developed, this complication could have been prevented.

operating room for a foot surgery unrelated to toe deformities, then tenotomies should be recommended to the patient at the time of the pre-operative evaluation. We perform our toe flexor tenotomies at the level of the metatarsal head rather than at the proximal interphalangeal joint levels. The additional soft tissue padding (metatarsal head fat pad) affords more reliable healing rates

compared to hidebound flexion joint creases.

**6. With ankylosed interphalangeal joints, tenotomies may need to be supplemented with realignment interphalangeal joint resections.** We also do this minimally invasively, as described in the following text box.

An ovoid incision is made about 8 mm wide centered over the apex of the deformity with the ends of the ovoid at about the midpoint of the medial and lateral sides of the toe. The ovoid skin incision is carried down to the bone level and the skin, extensor tendon and extensor joint hood excised. Next precise parallel osteotomies perpendicular to the long axes of the phalanges on both sides of the joint are made. The ovoid skin is closed with small nylon sutures. The approximation of the skin edges brings the osteotomized phalangeal ends in contact with each other, straightens the toe and acts as a splint to maintain the toe alignment.

Our approach negates the need for maintaining alignment of the interphalangeal joint fusion with temporary placement of a Kirshner wire through the medullary canal and the potential problems it imposes, such as keeping the patient non-weight bearing, infection of the pin tract and/or breakage or bending of the pin.

**7. Infrequently, toes straightened with tenotomies develop recurrent deformities.** In the pre-operative orientation for the patient, this should be mentioned. If necessary, second stage tenotomies and/or joint resections (see previous paradigm) are done.

**8. Active toe flexion and extension for respective extensor and flexor tenotomies by the awake patient facilitates the releases.** This makes the tendons taut like a bowstring (and sweeping the #11 scalpel blade transversely across the tendon in a pendulum-like fashion with the two-to three-mm skin incision as the pivot point) easy to release (Figure 7). Usually, audible and palpable

**Figure 6: Toe Deformity Management with Joint Manipulation**



**Legend:** Post-traumatic 90° flexion contracture (i.e. mallet toe) of distal interphalangeal joint of a 3<sup>rd</sup> toe. Toe could be straightened with manipulation, but because of fibroanklylosis of the joint, reduction could not be maintained.

Definitive manage requires a joint resection (see Paradigm #7)

sensations confirm the tenotomy is complete and verified by the patient being unable to actively flex or extend the toe. If the patient is insensate as is frequently the situation in patients with diabetes mellitus, no anesthesia is needed. If sensation is present, a field block with 1% lidocaine (without epinephrine) proximal to the incision provides adequate anesthesia for the procedure.

**9. Anticoagulation is not contraindication to doing these minimally invasive surgeries.** In the anti-coagulated patients, we typically double the time we apply direct pressure to the operative

site(s), i.e. ten minutes instead of five.

**10. Advanced peripheral arterial disease even in those patients with barely perceptible Doppler detected pulses, has not been a contraindication for doing these minimally invasive tenotomies.** After the tenotomies, we allow our patients to walk out of the office. Bandaging is minimal and done to maintain the toe in the corrected position, usually with weaving between the toes or horse shoe wrap under or over the toe depending on the correction desired.

**11. After having dealt with many toe deformities with “textbook” management (i.e. correction utilizing flexor tendon transfers and arthrodesis of the interphalangeal joints) of clawed, hammer, and mallet toe deformities, the minimal invasive toe tenotomy procedures without question**

**Figure 7: Percutaneous Toe Extensor Tendon Tenotomy**



**Legend:** Pressure placed on the end of the clawed toe (as the patient is asked to forcefully hyper extend his/her toes and ankle) causes the extensor tendon to bowstring over the dorsum of the foot.

This makes the tendon an easy target for transection. With a 2 to 3 mm incision and the skin as a pivot point, the #11 scalpel blade is swept transversely in a pendulum-like fashion over the taut tendon. Audible and palpable sensations signal the release of the tendon and the release is confirmed by the patient being unable to actively extend the toe.

**Figure 8: Percutaneous Toe Extensor Tendon Tenotomy**



**Legend:** In the left-side photo the patient had the more traditional (textbook) approach for dealing with her claw toe deformities including rerouting the flexor tendons to the dorsums of the proximal phalanges, interphalangeal joint fusions and temporary percutaneous joint pinning. Note the shortened 2<sup>nd</sup> and 3<sup>rd</sup> toes and their recurrent deformities. These toes were almost totally immobile. Thirty to 45 minutes surgical times, at best, are required to perform these procedures for each toe.

The right-hand photo shows the photos of previously clawed toes managed with tenotomies. Limited open for the extensor tendons and percutaneous for the flexor tendons. The patient elected to have the bilateral procedures done in one “sitting” in the operating room versus serial releases in the office. Although the toes are immobile, they remain straight, at normal length and not prone to develop ulcers. Note the mild recurrent hammer deformity of the little toe. If the patient so elects, this can be managed with a 2<sup>nd</sup> stage in-office percutaneous flexor tenotomy.

are our preferred recommendations (Figure 8)<sup>[3]</sup>.

**12. When patients are presented with the options of living with non-functional deformed toes at risk for developing ulcerations versus straight, cosmetically pleasing, not actively mobile toes not prone to ulceration, the answer is invariably the straight toe choice.** This option is especially recommended for the patient with comorbidities such as diabetes mellitus, peripheral artery disease, and peripheral neuropathy. Percutaneous and limited open (may be required for extensor tendons) tenotomies is the minimal invasive, quick-and-easy solution to achieve this goal.

### CONCLUSIONS

Our approach to managing clawed, hammer, and mallet toes has been uniformly successful with almost 100% satisfaction in the patients. One non-anticoagulated patient did develop a hematoma at a single, open extensor tenotomy site, and a couple of patients had minor superficial skin dehiscence, which subsequently healed with minor care. No complications were associated with the solely percutaneous technique. Several patients required delayed secondary procedures, such as flexor tenotomies after their extensor tenotomies. If the procedures are performed in the office, clinic, or at bedside, no more than two tenotomies are done at any one time. At these venues, the patients are given prophylactic oral antibiotics for a day or two.

What surgical skills are needed to perform the percutaneous tenotomies? Surgeons familiar with foot anatomy and an understanding of the pathophysiology of the axial toe deformities are the logical choices. Although bowstringing tendons are easy to transect, more difficulty may be experienced with tendons “hidden” by fatty subcutaneous tissues, edema, scar tissue, or hidebound skin. For such situations, the tenotomies should be performed by a foot and ankle surgeon, especially when these conditions are present on the dorsum of the foot. For easily accessible tendons, any wound care giver with suturing and debriding experience should be able to perform the minimally invasive percutaneous procedures after being suitably mentored and found to be competent in doing the tenotomies.

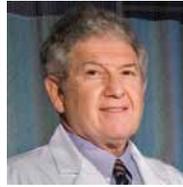
Occasionally, a two-to three mm incision becomes extended because of patient movement or inexperience. If this occurs and the accidental incision is large enough, it can be easily approximated with a couple of small nylon sutures.

Another advantage of our minimally invasively, keep-it-simple, and speedy approach to axial toe deformities is its cost effectiveness. The charges are a fraction of what they would be when performed in the clinic or office setting rather than the operating room. Use of the operating room may cost \$5,000-\$10,000 versus the in-clinic, in-office procedure costing 1/10<sup>th</sup> to 1/20<sup>th</sup> of this. The paradigms described above demonstrate the considerations we address when providing this effective, cosmetically, pleasing and cost-effective option to our patients with clawed, hammer and/or mallet toes.

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