ONLINE COURSE:
DIFFERENTIAL DIAGNOSIS
AND TREATMENT OF
LOWER EXTREMIT Y EDEMAS

Instructor:
Robyn Bjork MPT, CWS, WCC, CLT-LANA, CLWT
(3 Hours; 3.5CEUs)
Location: www.ILWTI.com
Course Description:

This continuing education course is designed for health professionals who treat patients with wounds or leg edema. Its purpose is to help the clinician identify various forms of edema so that an appropriate treatment plan can be developed, customized to the patients’ unique medical needs. By identifying and treating lower extremity edema effectively, the patient’s functional mobility and quality of life is enhanced, potential for wound healing increases, and complications, such as recurrent cellulitis, pain and recurrent wounds are prevented.

Course Objectives:

✧ 1. Understand basic anatomy, physiology and pathophysiology of the arterial, venous and lymphatic systems and how they relate to differential diagnosis and treatment of lower extremity edemas.

✧ 2. Differentiate between characteristics and treatment of Chronic Venous Insufficiency, Phlebolymphedema, Lymphedema, Malignant Lymphedema, Lipedema, Lipolymphedema, Dependent Edema, and edema due to Congestive Heart Failure, Renal Disease and Deep Vein Thrombosis.

✧ 3. Understand the importance of the Ankle Brachial Index and its relevance to compression therapy.

✧ 4. Understand the components of Complete Decongestive Physiotherapy.
OUTLINE:

I. Anatomy, Physiology & Pathophysiology of the Arterial, Venous, and Lymphatic Systems

A. Arteries

1. Anatomy & Physiology

   a) Overview

      (1) arteries carry oxygen and nutrient rich blood to tissues

      (2) delivers cells and building blocks for wound healing to the tissues/skin

2. Pathophysiology

   a) Peripheral Arterial Disease

      (1) PAD = Atherosclerosis = plaques cause narrowing of artery which impairs blood flow

      (2) Not the same as hardening of the arteries due to calcification of the artery wall. “Hardening of the Arteries” (Arteriosclerosis)

      (3) Lack of blood flow and oxygenation of the tissues can result in arterial ulcerations or tissue necrosis

      (4) Wounds are slow to heal or cannot heal if blood flow is impaired

   b) Clinical Relevance

      (1) You must perform an ABI to rule out peripheral arterial disease on patients that need compression therapy for edema management of the legs

      (2) The standard of practice for treating edema is compression therapy but compression added to undiagnosed underlying...
3. Ankle Brachial Index (ABI)
   a) Calculation
      (1) ABI is calculated by dividing the higher systolic blood pressure of either the dorsalis pedis or posterior tibial arteries in one leg by the higher of the brachial systolic blood pressures in both arms
   b) Interpretation
      (1) ABI >1.4
         (a) falsely elevated due to calcification of vessel wall
         (b) perform Toe Brachial Index (TBI)
      (2) ABI 1.0-1.4
         (a) normal
         (b) safe to initiate class I-IV compression
      (3) ABI 0.91-0.99
         (a) borderline Peripheral Arterial Disease (PAD)
         (b) safe to initiate class I-IV compression
      (4) ABI 0.80 – 0.90
         (a) mild PAD or lower extremity arterial disease (LEAD)
         (b) safe to initiate class I-III compression
         (c) manage risk factors
      (5) ABI 0.50 – 0.79
         (a) Moderate PAD or LEAD
         (b) use class I-II compression
(c) treat with multi-layered, short stretch bandaging systems only (NOT long stretch, elastic systems), up to 30-40mmHg

(d) routine vascular specialist referral is indicated

(6) ABI < 0.5

(a) indicates severe PAD or LEAD

(b) no compression

(c) urgent vascular specialist referral is indicated

(d) limb threatening ischemia

c) Compression Class Reference for Compression Garments

(1) I = 14-18mmHg (ineffective for therapeutic compression)

(2) II = 18-24mmHg

(3) III = 25-40mmHg

(4) IV = 40-50mmHg

B. Veins

1. Anatomy & Physiology

a) Overview

(1) veins carry blood back toward heart and lungs to release carbon dioxide and re-oxygenate

(2) veins have one way valves which prevent regurgitation of blood in the venous system

b) Calf Muscle Pump

(1) blood flow in veins is dependent on calf muscle contractions
2. Pathophysiology
   a) Increased pressure in veins:
      (1) forces red blood cells out of capillaries (hemosiderin staining)
      (2) increases capillary pressure and filtration and fluid retention (edema) in tissues
      (3) results in valvular failure (venous insufficiency)
      (4) creates venous stasis, inflammation and skin breakdown (stasis dermatitis, venous ulcers)
      (5) overloads the lymphatic system

C. Lymphatics
   1. Anatomy & Physiology
      a) Overview
         (1) Garbage disposal and recycling system
         (2) Recycles plasma proteins back into the bloodstream after they deliver nutrition to cells
         (3) Carries byproducts, toxins, debri, senescent cells, and bacteria from skin/tissues/wound to lymph nodes to be analyzed and phagocized
         (4) Langerhan’s cells and other white blood cells carry antigen information, or bacteria, from the skin to the lymph nodes. There they trigger an immune response.
      b) Lymphatic Capillaries
         (1) Overview
(a) Lymphatic capillaries start at the arteriovenous capillary level

(b) 70% of lymph comes from the skin

(c) Lymphatic capillaries of the skin originate in the dermis, less than 3mm from the surface of the skin!

(d) Forms a dense, spider web-like network in the skin

(2) Swinging Tips

(a) The lymphatic capillary wall is a single layer of endothelial cells, with overlapping inner and outer flaps called “swinging tips”

(b) These spaces between the cells of the lymphatic capillary are 5x larger than in the arterial/venous capillary. Allows large blood plasma proteins and cells (i.e. Langerhan cells, Macrophages) to enter the lymphatic capillaries

(c) As lymph fills the capillary, the inner flap closes to prevent reflux back into the interstitium

(3) Anchoring Filaments

(a) Swinging tips anchor to elastic fibers of the skin via anchoring filaments

(b) Anchoring filaments also attach to the capillary wall

(c) As edema increases in the tissue, the elastic fibers stretch and lift the swinging tips, allowing more drainage into the lymphatic capillaries

(d) The fibers also pull open the lumen of the capillary, increasing flow of lymph into it

(c) Collectors & Lymphangions
(a) Collectors are transport vessels that propel lymph toward heart

(b) Collectors are made up of contractile units called Lymphangions, meaning “little hearts”

(c) One way bicuspid valves prevent reflux of lymph

d) Lymph nodes

(1) 600 located primarily in neck, axillae, groin, abdomen

(2) collectors propel lymph through at least two nodes to filter the lymph before it enters a lymphatic trunk

(3) Macrophages in node digest foreign material

(4) Lymphocytes (WBC) in node detect and fight bacteria and cancer, etc. by mounting a cell-mediated (T-cells) or humoral response (B-cells)

e) Lymphatic trunks

(1) collectors converge into lymphatic trunks

(2) lymphatic trunks propel lymph to the venous angles in the neck to be dumped back into the bloodstream

(3) Thoracic Duct and R Lymphatic Duct are largest trunks in body

(4) Thoracic Duct

(a) Largest lymphatic trunk in body. 2-5mm wide and 36-45cm in length

(b) Drains both legs, lower quadrants of thorax, L head, neck and L UE (3/4 of body)

(c) 1.5 to 3 liters of lymph per day circulates through the thoracic duct alone (40% of the liquid volume of lymph entering a lymph node is re-circulated directly into the
venous system through the node's veins, before it reaches the thoracic duct)

(d) Runs along anterior spine and penetrates through the Diaphragm

(e) Deep diaphragmatic breathing milks/drains the thoracic duct

2. Pathophysiology

   a) Lymphedema is a disruption in the normal flow of lymph resulting in an accumulation of plasma proteins in the tissues

   b) Proteins act like sponges, drawing in and holding fluid/edema in the tissues

   c) Excess proteins denature, causing a pathohistological state of chronic inflammation in the tissues which attracts macrophages

   d) Macrophages stimulate fibroblasts to produce excess collagen, causing massive connective tissue proliferation and fibrosis

II. Differential Diagnosis & Treatment of Common Lower Extremity Edemas

A. Chronic Venous Insufficiency (CVI)

   1. Pathophysiology

      a) Etiology: Insufficiency of venous system due to valvular failure of deep, perforating or superficial veins

      b) results in regurgitation of blood in the veins

   2. Characteristics

      a) Distinguishing Feature: Hemosiderin Staining

      b) Protein content: low, watery form of edema

      c) Stemmer Sign: negative, no edema in the toes
d) Symmetry: symmetrical

e) Appearance: edema and skin changes primarily in ankle and gaiter area

f) Texture: brawny

g) Progression: distal, below knee

h) Response to elevation: edema reduces with elevation or overnight when sleeping horizontally

i) Onset: slow

j) Pain: achy, distention type pain, gets worse as day progresses, especially with prolonged standing or sitting

k) Wounds: weeping, blistering and shallow, painful venous ulcers are common

l) Skin changes: hemosiderin staining, atrophe blanche, lipodermatosclerosis, brawny or taut skin, varicose veins, dermatitis, distended medial ankle veins

m) Infection: rarely cellulitis; cellulitis is uncommon, dermatitis is common

3. Tests needed

a) ABI Study to rule out arterial disease

b) Venous Duplex Ultrasound with Venous Insufficiency Study determines areas of valvular failure in the superficial system, such as the greater saphenous vein

4. Treatment

a) Short stretch multi-layered compression bandaging

   (1) compression bandaging is gold standard of treatment for reducing venous edema and healing venous ulcers
(2) compression bandaging coupled with walking facilitates the calf muscle pump, compensates for incompetent valves, and increases return of blood to the heart through the veins

b) Elevation, ankle pumps, walking with bandages on, treatment for limited ankle ROM

c) Long term day use only of compression garments

(1) Ideal compression level 30-40mmHg

d) Wound care

e) Surgery

(1) corrective venous procedures obliterate insufficient veins in the superficial or perforating system

(2) can resolve or improve venous edema

(3) prevents recurrent ulcerations

f) Short term use of diuretics may be indicated

B. Deep Vein Thrombosis (DVT)

1. Pathophysiology

   a) Etiology: blood clot obstructing a deep vein

   b) can cause scarring and failure of valves in the deep system

   c) valvular failure in the deep system can cause valvular failure in the perforating veins or superficial system

2. Characteristics

   a) Protein content: low, watery edema

   b) Stemmer Sign: negative

   c) Symmetry: usually unilateral

   d) Appearance: red, hot, acutely swollen calf, ankle and foot
Differential Diagnosis and Treatment of Lower Extremity Edemas

e) Texture: firm edema in calf
f) Progression: edema below level of DVT; high risk for Pulmonary Embolism
g) Response to elevation: reduces
h) Onset: acute
i) Pain: tender or cramping calf, worse with forced dorsiflexion; 50% are asymptomatic
j) Wounds: weeping and blisters and secondary venous ulcers are common
k) Skin changes: red, hot, swollen, firm
l) Infection: not directly associated with cellulitis

3. Tests needed
   a) Venous Duplex Ultrasound

4. Treatment
   a) Acute treatment:
      (1) high risk for pulmonary embolism
      (2) rest, elevation, anticoagulants
      (3) DVT thrombectomy
      (4) wound care, if needed
   b) Subacute or chronic treatment:
      (1) elevation, exercise
      (2) short stretch multi-layered compression bandaging
      (3) long term use of compression garment during the day (ideal compression level 40-50mmHg)
      (4) wound care, if needed
C. Lymphedema

1. Pathophysiology
   a) Etiology: Disruption in the normal flow of lymph resulting in an accumulation of interstitial proteins causing edema, and thickened, fibrotic skin

2. Characteristics
   a) Distinguishing Features:
      (1) Positive Stemmer’s Sign = Lymphedema
         (a) Stemmer’s Test Procedure
            i) pinch and lift the skin on the dorsal aspect of the base of the second toe
         (b) Positive Stemmer’s sign
            i) the skin at the base of the second toe cannot be pinched and lifted
               (1) cause by toe swelling and fibrosis
               (2) never falsely positive
               (3) indicative of lymphedema
         (c) Negative Stemmer’s Sign
            i) the skin at the base of the second toe can be pinched and lifted.
               (1) negative does not exclude lymphedema
               (2) may be false negative
      (2) Distinguishing skin features are papillomatosis and hyperkeratosis in advanced lymphedema of legs or pannus
         (a) Papillomatosis (lumpy, bumpy skin, or fibrotic wart-like projections of the skin)
(b) Hyperkeratosis (scaly brown or grey patches of overproliferated keratin layers)

b) Protein content: high

c) Symmetry: asymmetrical edema of arms or legs

d) Appearance: edematous legs with square shaped toes, deep creases and folds, fibrotic tissue, loss of limb contours

e) Texture: lumpy, bumpy or hard, crust, fibrotic

f) Progression: distal to proximal progression

g) Response to elevation: in very early stages, lymphedema will reduce overnight or with leg elevation, but as it progresses elevation no longer reduces it

h) Onset: slow, develops and worsens over many years

i) Pain: typically not painful

j) Wounds: skin ulcers are uncommon, however, lymphorreah with skin denudement is common

k) Skin changes: progressive fibrosis, hyperkeratosis, papillomatosis

l) Infection: recurrent cellulitis and fungal infections are common

3. Tests needed

a) ABI Study to rule out peripheral arterial disease (PAD)

b) Venous Duplex Ultrasound to rule out DVT

4. Treatment

a) Complete Decongestive Therapy (CDT) is the Gold Standard of treatment for Lymphedema

   (1) Components of CDT

   (a) Meticulous Skin and Nail Care
(b) Manual Lymphatic Drainage
   i) Because Lymphedema is a high protein edema, compression alone is not effective in its management
   ii) Manual lymphatic drainage (MLD) is used to stimulate opening of lymphatic capillaries to drain excess proteins and edema from the tissues
      (1) stretches the anchoring filaments
      (2) opens the swinging tips of the lymphatic capillary and capillary lumen
   iii) MLD reroutes lymphatic flow through redundant and unaffected pathways
   iv) MLD facilitates lymphangion contraction

(c) Multi-layered Compression Bandaging
   i) short stretch bandages
   ii) customized foam inserts for fibrotic areas

(d) Compression Garments & Night Devices
   i) Long term compression
   ii) Garment or device for daytime use and night compression device
   iii) Ideal compression levels 40-60mmHg legs, 20-30mmHg arms

(e) Therapeutic Exercises

(f) Patient Education and Home Program: Self Bandaging, Custom Compression Garments, Night Devices, Skin Care, Self Manual Lymphatic Drainage, Home Exercise
b) Diuretics are contraindicated for treating lymphedema

D. Phlebolymphedema
1. Pathophysiology
   a) Etiology: venous edema distends the skin to the point where stretched anchoring filaments rupture the swinging tips and endothelial lining of the lymphatic capillary, causing secondary lymphedema

2. Characteristics
   a) A combination of venous (phlebo) disease and lymphedema secondary to the venous disease
   b) Has s/s of both venous disease and lymphedema

3. Treatment
   a) Treat both conditions
   b) If venous disease is managed, continued damage to lymphatics will cease
   c) Lymph capillaries can regenerate
   d) Diuretics are not indicated to reduce the edema

E. Malignant Lymphedema
1. Pathophysiology
   a) Etiology: Caused by malignant tumor obstructing lymphatics

2. Characteristics
   a) Protein content: high
   b) Stemmer Sign: positive or negative
Differential Diagnosis and Treatment of Lower Extremity Edemas

C) Symmetry: asymmetrical
D) Appearance: reddened, distended, proximal edema
E) Texture: firm edema
F) Progression: proximal to distal
G) Response to elevation: persists
H) Onset: rapid
I) Pain: pain with muscle guarding, tingling, numbness, weakness
J) Wounds: fungating tumors, radiation ulcers or burns, angiosarcoma
K) Skin changes: inflamed, distended skin; masses or enlarged nodes, tangelectasias
L) Infection: high risk of cellulitis

3. Tests needed
   A) MRI, Diagnostic testing
   B) Still need to rule out DVT or underlying PAD before applying compression therapy

4. Treatment
   A) Pressing concern is diagnosis of potential underlying malignancies and treatment for cancer
   B) Once patient is under care of oncologist and referral for CDT is made, CDT can be used to reduce edema, reduce pain and improve mobility and quality of life
   C) Still treat using all components of CDT, including manual lymphatic drainage therapy, short stretch multi-layered compression bandaging with or without foam, and compression garments or devices during day and night devices
d) In palliative care consider MLD for pain management and modify treatment based on individual patient’s energy level, tolerance, etc.

e) Ideal compression: 40-60mmHg legs and 20-30mmHg arms

f) Diuretics are contraindicated for malignant lymphedema reduction

F. Lipedema

1. Pathophysiology

a) Lipedema is NOT LYMPHEDEMA

b) Etiology:

   (1) abnormal fat metabolism and deposition (not obesity). It is an accumulation of subcutaneous fatty tissue

   (2) rare disease of unknown origin with bilateral symmetrical accumulation of fat from waist to ankle

2. Characteristics

a) Distinguishing Features: large hips & thighs with disproportionately small trunk and arms and skinny feet

b) Protein content: low

c) Stemmer Sign: there is no thickening of the skin in Lipedema and thus Stemmer sign is negative

d) Symmetry: bilateral symmetrical

e) Appearance: large hips & thighs with disproportionately small trunk and arms and skinny feet

f) Texture: loose, lobular

g) Progression: distributed evenly hips & thighs

h) Response to elevation: unaffected, edema trapped in fat cells
i) Onset: slow (primarily affects females)

j) Pain: the heavy legs are painful to palpation

k) Wounds: no ulcers or weeping

l) Skin changes: skin bruises easily because of the fragility of vascular walls

m) Infection: no cellulitis

3. Tests needed
   a) n/a unless concurrent CVI or lymphedema

4. Treatment
   a) Supportive compression garments: thigh high or pantyhose
   b) Ideal compression 20-30mmHg
   c) Education, healthy diet and exercise will prevent concurrent obesity and deconditioning but will not reduce or resolve Lipedema
   d) May progress to CVI and Lymphedema which then also require treatment
   e) Diuretics are not indicated to reduce Lipedema

G. Lipolymphedema
   1. Characteristics
      a) A combination of both Lipedema and Secondary Lymphedema
   2. Treatment
      a) Treat both conditions
      b) Diuretics are not indicated to reduce this type of edema

H. Congestive Heart Failure
   1. Pathophysiology
DIFFERENTIAL DIAGNOSIS AND TREATMENT OF LOWER EXTREMITY EDEMAS

a) Etiology: Weakened and inefficient ability of the heart to pump blood resulting in increased pressure in the heart and fluid retention in the body

2. Characteristics
   a) Protein content: low, watery form of edema
   b) Stemmer Sign: Stemmer sign is negative. Though patient may have edema of toes or fingers, skin is not thickened and skin can be pinched during test if pinch is held until edema displaces
   c) Symmetry: Symmetrical edema; levels fluctuate with heart function.
   d) Appearance: “buffalo hump” on dorsal feet
   e) Texture: Deeply pitting edema; soft and pliable like dough
   f) Progression: Distal to proximal progression. Edema may progress into torso
   g) Response to elevation: Edema reduces rapidly with leg elevation
   h) Onset: Rapid onset in acute CHF
   i) Pain: Distention discomfort
   j) Wounds: Distention and weeping, watery edema; blisters may develop which progress into shallow ulcers
   k) Skin changes: cyanosis; Coupled with s/s of CHF such as shortness of breath, fatigue, jugular distention, rales or crackles in lungs, etc.
   l) Infection: Not associated with cellulitis

3. Tests needed
   a) Rule out DVT and PAD with Venous Duplex Ultrasound and ABI study
4. Treatment
   a) Requires treatment for underlying CHF
   b) After acute CHF is treated and stabilized, reduce edema with short stretch bandaging followed by thigh-high 20-30mmHg compression garments during the day
   c) Diuretics are indicated to manage lower extremity edema resulting from CHF

I. Renal Insufficiency/Failure
   1. Pathophysiology
      a) Etiology: Inability of kidneys to adequately filter waste products and excess fluid from the blood
   2. Characteristics
      a) edema worsens until dialysis begins
      b) generalized edema throughout body
      c) predisposed to lymphedema and lower leg wounds
   3. Tests needed
      a) Rule out DVT and PAD with Venous Duplex Ultrasound and ABI study
   4. Treatment
      a) medical management/dialysis
      b) need solid containment/compression bandaging for edema in order to reduce it and heal lower extremity ulcers
      c) edema varies - need compression device that varies with it
      d) coordinate treatment with dialysis; apply compression bandaging after dialysis
      e) diuretics are indicated to reduce edema
J. Dependent Edema

1. Pathophysiology
   a) Etiology: caused by prolonged dependent positioning
   b) Examples: legs dangling off of bed all night, legs dangling in sitting all day, pannus hanging down off chair, bed, or tissue hanging down between the legrest and seat of a recliner, etc.

2. Characteristics
   a) Soft and pitting
   b) Resolves with elevation
   c) Stemmer Sign is negative
   d) Extent of edema directly related to time in dependent position

3. Tests needed
   a) Rule out DVT and PAD with Venous Duplex Ultrasound and ABI study

4. Treatment
   a) Adjust positioning so area is elevated at night and during periods of immobility. Limit dependency of the area.
   b) Improve mobility and muscle contractions through ROM and therapeutic exercise
   c) Short Stretch multilayered compression bandaging to reduce edema
   d) Compression garments or devices, as needed, to maintain it
   e) Generally use lighter compression 15-25mmHg or 20-30mmHg
   f) Diuretics are not needed to reduce dependent edema
Reference List:


