

wound healing perspectives®

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VOLUME 5 NO. 3 SUMMER 2008

→ EDEMA

A PUBLICATION OF NATIONAL HEALING CORPORATION

“Doctor, why is my leg swollen?”

In this issue of Wound Healing Perspectives, we are addressing the multiple etiologies of edema—venous, metabolic, cardiac, renal, mechanical, trauma, drugs, and lipedema, which is often confused with the various forms of lymphedema.

The patient has to be evaluated for edema, the etiology of which may be multi-factorial, and each form of edema needs to be corrected if possible. Failure to correct edema in patients with chronic non-healing wounds will result in a significant delay in wound healing.

The patient will often ask, “Doctor, why is my leg swollen?” We as wound care professionals also have to ask ourselves the very same question. Here is where the art of taking a good, thorough history (the patient is telling you what is wrong with them), and asking questions appropriately, such as: When did the swelling start? How did it start? What is the location of the swelling? At what time of day is the swelling worse? How is the swelling in the morning - better or about the same? The answers to these types of questions will frequently provide clues to the diagnosis.

Elimination of edema with documentation of adequate arterial perfusion pressure, are dramatic drivers to wound healing. We hope this issue will refresh your knowledge as to the causes of edema and improve patient care and wound healing outcomes.

Sincerely,



Jack E. Lighton, DO, FACOS, CWS, FCCWS
Medical Advisory Board Member
Member of the Wound Healing Society

Understanding edema

Peripheral edema, the visible swelling of tissues especially in the feet and legs, occurs when there is an accumulation of excess fluid under the skin in the spaces within the tissues [www.medicinenet.com]. Such swelling in itself, however, should not be considered a disease but instead a sign of an underlying, and sometimes serious, disorder. Physicians, therefore, should examine the patient carefully to pinpoint the underlying cause of the edema, states Ruschhaupt [Chapter 36: The Swollen Leg].

Taking a history and physical examination of the patient is the best way to diagnose edema. These findings are important in determining what additional diagnostic tests, such as duplex imaging, Doppler ultrasound, and photoplethysmography, are necessary to confirm the diagnosis [Ruschhaupt]. The sudden onset of edema, occurring over several hours or days, can mean that an acute process is taking place such as deep vein thrombophlebitis, cellulitis, compartment syndrome, or gastrocnemius muscle rupture. If the onset is more gradual, the patient may have lymphedema, chronic venous insufficiency, or may be experiencing a reaction to certain medication(s). Many systemic influences, like renal salt and water balance, as well as physiologic concepts, such as effective blood volume



Elimination of edema with documentation of adequate arterial perfusion pressure, are dramatic drivers to wound healing.

or inadequacy of arterial vascular filling, may also cause edema [Ruschhaupt].

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Understanding edema

Causes of peripheral edema

- Increased capillary hydrostatic pressure
- Regional venous hypertension (often unilateral)
- Inferior vena cava/iliac compression
- Deep venous thrombosis
- Chronic venous insufficiency
- Compartment syndrome
- Systemic venous hypertension
- Heart failure
- Constrictive pericarditis
- Restrictive cardiomyopathy
- Tricuspid valvular disease
- Cirrhosis/liver failure
- Increased plasma volume
- Renal failure (acute, chronic)
- Drugs
- Pregnancy
- Premenstrual edema
- Decreased plasma oncotic pressure
- Protein loss
- Malabsorption

(Continued on next column)

According to Cho and Atwood in a 2002 article, total body water is divided between the intracellular and extracellular spaces of the body. About one-third of the total body water is found in the extracellular space and made up of the intravascular plasma volume (25 percent) and the extravascular interstitial space (75 percent).

Certain physiologic forces play a role in maintaining the balance of water between these two compartments. This includes the gradient between intravascular and extravascular hydrostatic pressures, differences in oncotic pressures within the interstitial space and plasma, and the hydraulic permeability of the blood vessel wall [Cho et al.]. The lymphatic system collects fluid and filtered proteins from the interstitial space and returns them to the vascular compartment.

According to Ruschhaupt, there are six potential factors that can cause edema to form in the extremities. These include four major physiologic principles including capillary blood pressure, which can occur when there is deep vein occlusion, colloid oncotic pressure, interstitial tissue pressure, and tissue pressure itself. Other factors include changes in capillary permeability and obstruction of lymphatic flow.

Causes of unilateral limb swelling

There are various causes of unilateral edema. The top three most common vascular causes for this type of edema are deep vein thrombophlebitis, chronic venous insufficiency, and lymphedema. Below is a brief description of each.

Acute deep vein thrombophlebitis

When the swelling of the limb is due to acute deep vein

thrombophlebitis, the onset of the edema can be very dramatic. The swelling also may be accompanied by bluish-red discoloration and prominent veins. Pain also may precede or accompany the swelling.

Chronic vein insufficiency

One of the results of acute deep vein thrombophlebitis can be the development of chronic vein insufficiency. This is caused by damage to the valves of the venous system, which can then lead to stasis syndrome and the potential for the development of irregular pigmentation, dermatitis, edema, cellulitis (a nonbacterial inflammatory process), and ulceration, according to Ruschhaupt. Chronic venous insufficiency also can lead to venous insufficiency ulcers, which account for 70-90 percent of all leg ulcers. It is estimated that there are approximately seven million

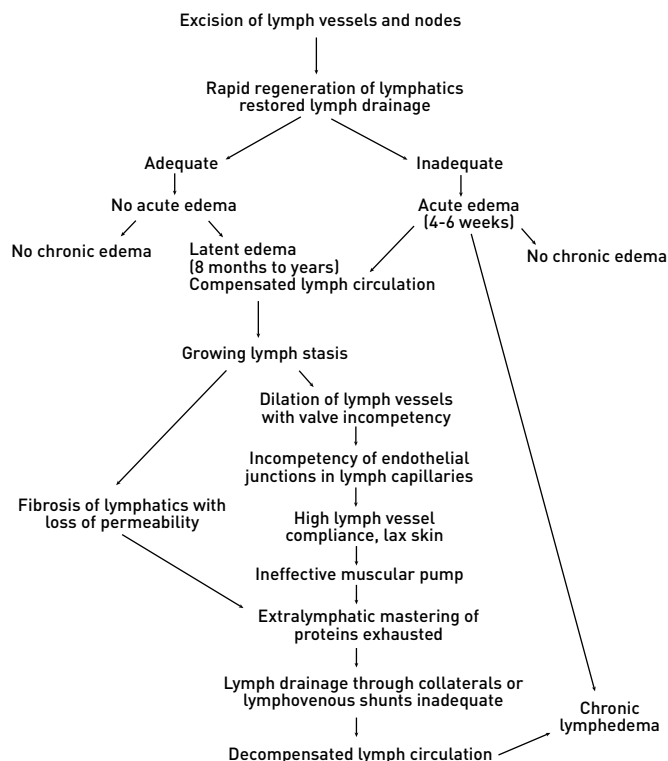
people in the U.S. with venous insufficiency, and of that number, about one million develop ulceration. See related article on venous ulcers on page 3.

Lymphedema

According to Ruschhaupt, the third most common vascular cause of limb swelling is lymphedema. Defined as an accumulation of fluid in the interstitial spaces of the body, lymphedema is caused by a defect in the lymphatic system, which fails to carry lymphatic fluid, which then causes stasis of the lymph [Schirger and Glocviczki, 1990]. This results in the abnormal collection of excess tissue proteins, edema, chronic inflammation, and fibrosis [Holcomb, 2006].

The onset of lymphedema in patients typically occurs slowly, unless it is related to an acute surgical or traumatic disruption of the lymphatic system.

Stages in development of postoperative lymphedema



[Prom Glocviczki P. and Schirger A; Lymphedema. In Spittell JA. Jr, ed; Clinical Medicine, Philadelphia, 1985, Harper & Row, Publishers.]

As a clinician, it is important to recognize that lymphedema always starts in the most distal portion of the extremity, so carefully examining the hand or foot will help to catch it early [Ruschhaupt]. Furthermore, lymphedema can be a challenge for clinicians, especially when patients present with comorbidity of peripheral vascular disease (PVD). See article on page 6 for more information on lymphedema.

Cellulitis

The most common infectious cause of limb swelling is cellulitis, which occurs when there is a portal through the normal skin's barrier for bacteria to enter. This allows toxins to be released into the subcutaneous tissues, which can occur as a single isolated event or a series of recurrent events, notes Ruschhaupt. What's more, cellulitis is often misdiagnosed as recurrent thrombophlebitis or worsening chronic venous insufficiency.



Abscess

Abscess typically follows a deep-seated infection, usually

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Guidelines for diagnosing and treating venous ulcers

The following diagnostic guidelines should be followed when assessing venous ulcers:

Determine arterial disease by using these guidelines:

- Pedal pulses and ABI greater than or equal to 0.8 (contraindicated if the person is a diabetic)
- ABI < 1.0 suggests vascular disease; if ABI less than 0.7 then compression therapy is contraindicated
- In elderly patients, diabetic patients or patients with ABI greater than 1.2, Toe: brachial index greater than 0.6 or transcutaneous oxygen pressure of greater than 30 mm Hg may help to suggest adequate arterial flow.

Color duplex ultrasound scanning is useful in confirming venous etiology.

If the clinician suspects sickle cell disease, diagnose with sickle cell prep and hemoglobin electrophoresis.

If the ulcer is older than three

months or is not responsive after six weeks of therapy, conduct a biopsy for histological diagnosis (for possible malignancy or other disease).

If worsening despite treatment or excessively painful, consider other diagnoses such as pyoderma gangrenosum, IgA monoclonal gammopathies, and Wegener's granulomatosis, among others.

Lower extremity compression

Use of Class 3, high-compression system is indicated. The degree of compression must be modified when mixed venous/arterial disease is confirmed.

Intermittent pneumatic leg compression (PIC) can be used with or without compression (another option for patients who cannot or will not use adequate compression). However, compression should not be performed until after patient has first undergone arterial and venous diagnostic testing to make

sure there is no deep vein thrombosis (DVT) as well as to identify if there is reflux and where it is located.

Treatment

If less than or equal to 10^6 CFU/g of tissue or any beta hemolytic streptococci, use topical antimicrobial. Discontinue once in bacterial balance to minimize cytotoxicity and development of resistance. Systemically administered antibiotics do not effectively decrease bacterial levels in granulating wounds. Topical antimicrobial can be effective.

Cellulitis around the ulcer should be treated with systemic gram-positive bacterial antibiotics.

Minimize the tissue level of the bacteria, preferably to less than or equal to 10^5 CFU/g of tissue, with no beta hemolytic streptococci in the venous ulcer prior to attempting survival closure by skin graft, skin equivalent, pedicled, or free flap. ■

SOURCE: CHRONIC WOUND CARE GUIDELINES, 2007

Causes of peripheral edema *cont'd*

- Preeclampsia
- Nephrotic syndrome
- Reduced protein synthesis
- Malnutrition (e.g., Kwashiorkor)
- Beriberi
- Increased capillary permeability (usually clinically obvious)
- Allergic reactions [histamine release (hives), serum sickness, Angioedema]
- Burns
- Inflammation/local infections
- Interleukin 2 therapy
- Lymphatic obstruction or increased interstitial oncotic pressure
- Lymphedema (primary or secondary nodal enlargement due to malignancy, postsurgical radiation, filariasis)
- Idiopathic
- Myxedema
- Other

SOURCE: CHO ET AL, 2002

stemming from some type of puncture wound. The swelling of the limb usually occurs with early abscess formation but can also have the appearance of a generalized edematous process [Ruschhaupt].

Osteomyelitis

Infectious limb swelling can also occur with osteomyelitis. When this is the case, the swelling usually occurs in the distal portion of the extrem-

Causes of Bilateral Limb Swelling

Heart Failure

Limb edema can be associated with congestive heart failure, notes Ruschhaupt, and the onset of swelling can be subtle. It also may not be associated with the typical findings of dyspnea (difficulty of breathing or painful breathing on exertion), orthopnea

The signs of both constrictive pericarditis and restrictive cardiomyopathy are similar to those of right heart failure, states Cho et al. Patients with distended neck veins often receive a misdiagnosis of primary hepatic cirrhosis. A possible diagnosis of constriction or restriction should be considered in a patient with unexplained edema, elevated jugular venous pressure, and relatively preserved systolic

“There are six potential factors that can cause edema to form in the extremities. These include four major physiologic principles including capillary blood pressure (which can occur when there is deep vein occlusion), colloid oncotic pressure, interstitial tissue pressure, and tissue pressure itself. Other factors include changes in capillary permeability and obstruction of lymphatic flow.”

ity. Ruschhaupt states that diabetic patients with diabetic neuropathy and neurotrophic ulcers are prime candidates for such an infection, but many patients with peripheral neuropathy are susceptible to such swelling. Patients with sickle cell anemia also may develop osteomyelitis [Ruschhaupt].

Charcot’s joints

In patients with Charcot’s joints (neuropathic arthropathy), a progressive degenerative disease of the joints, the joints are noticeably swollen and deformed and often characterized by warmth.

Other causes

Other causes of unilateral swollen limb include popliteal cysts, popliteal artery aneurysm, trauma, compartment syndrome, vascular anomalies, thermal injury, tumors, dependency, gastrocnemius rupture, revascularization, retroperitoneal fibrosis, and factitial limb swelling.

(inability to breathe easily unless sitting up or standing straight) or paroxysmal nocturnal dyspnea. In older individuals, however, edema frequently occurs concurrently with chronic obstructive pulmonary disease and congestive heart failure. In such cases, the presence of edema can be an early manifestation of the disease [Ruschhaupt].

Patients with right ventricular heart failure tend to have peripheral edema. The severity of the edema may be disproportionate to the degree of central venous pressure elevation depending on factors such as immobility, posture, and venous insufficiency. According to www.heartfailure.org, this occurs because fluid backs up in the veins, leaks out of capillaries, and accumulates in tissues. A decrease in blood flow to the kidneys can lead to an increase in fluid retention as well.

function. Although echocardiography may provide indirect evidence, more invasive studies such as right heart catheterization or tissue biopsy are often necessary to make a conclusive diagnosis [Cho et al].

Patients with left ventricular failure will have pulmonary edema, since fluid cannot be pumped to the lungs at an efficient rate. Both systemic and pulmonary congestion are present in patients with biventricular heart failure [www.aafp.org].

Cirrhosis

Cirrhosis of the liver can lead to bilateral edema, since many of the dilated venous tributaries and multiple small arterial venous fistulas help to increase total blood volume with decreased effective blood volume [Ruschhaupt]. When this is combined with alterations in serum albumin levels, there

Risk factors for peripheral edema

- Family history
- Varicose veins
- Multiple pregnancies
- Thrombophilia (protein S or C deficiency)
- DVT/phlebitis
- Trauma
- Obesity
- Lifestyle/sedentary occupation
- Advanced age

SOURCE: WOUND HEALING SOCIETY GUIDELINES, 2007

Peripheral edema and hemodynamic alterations

A 1983 study by Magrini and Niarchos examined eight hospitalized patients. Five of the eight had peripheral edema due to cirrhosis of the liver, two had edema due to nephrotic syndrome, and one had edema caused by hypoalbuminemia (where levels of albumin in blood serum are abnormally low). None of the patients had any clinical evidence of acute left ventricular failure. The study revealed that

marked peripheral edema in patients with heart failure resulted in abnormal hemodynamic findings in both the supine and head-up tilt position.

Although the patients had different causes of edema, the study revealed they, in fact, had one thing in common—they all had similar abnormal hemodynamic findings. These abnormal hemodynamic findings were reversed

back to normal when the edema was cleared as the result of diuretic therapy. The authors concluded that peripheral edema was the main factor responsible for the observed hemodynamic alterations. (These abnormalities in patients with the cirrhotic edema did not resemble the hemodynamic findings of cirrhotic patients with edema.) ■

SOURCE: MAGRINI ET AL, 1983

Patient education



Understanding edema (continued from page 4)

is further activation of the salt and water retaining mechanisms of the body. According to Ruschhaupt, peripheral edema is usually seen when both mechanisms are present and hypoalbuminemia is easily detected.

Protein deficiency states

When there is protein deficiency in a patient due to severe malnutrition of protein-losing gastroenteropathy, the decrease in plasma proteins results in a decrease in the plasma colloid pressure. These changes in pressure enhance the accumulation of interstitial fluid. For example, nephrotic syndrome results in edema through the same changes in plasma protein [Ruschhaupt].

Acute glomerulonephritis

When there is abnormal renal function, there are typically hemodynamic factors that are causing edematous states as well as intrinsic changes in the kidney that may cause edema to occur. Hematuria, proteinuria, and hypertension

also are usually present with the edema [Ruschhaupt].

Cushing's syndrome

Cushing's syndrome, the result of increased production of cortisol by the adrenal gland, can be produced by the ingestion of hydrocortisone-containing or producing drugs. At high concentration, tubular sodium reabsorption occurs, which leads to edema formation [Ruschhaupt].

Idiopathic cyclic edema

Usually not well recognized or understood, idiopathic cyclic edema usually occurs in women between the ages of 20 and 40, who also may be mildly obese. The location of the edema will usually occur in the upper and lower extremities as well as the face. It also seems to be directly related to the upright position [Ruschhaupt].

Pregnancy

Pregnancy causes peripheral vasodilation, which helps to decrease the effective blood volume. This can then lead to

sodium and fluid retention.

Obesity

Obesity is often associated with sodium and fluid retention, due to multiple mechanisms such as increased peripheral vasodilation with increased surface area of the interstitial spaces. Obesity also can lead to bilateral swelling since the fat deposits in the subcutaneous tissues, giving the appearance of enlarged limbs [Ruschhaupt].

Lipedema

Characterized by the bilateral and symmetric deposition of fat in the lower extremities, lipedema occurs almost exclusively in women and it may run in families. Patients with swelling or pain are often prescribed the use of heavy elastic support stockings [Ruschhaupt].

Other causes

Other causes include dependency, pretibial myxedema, and drug-induced edema. ■

To reduce the onset of peripheral edema, patients should:

- Understand compression therapy
- Wear stockings
- Stop smoking
- Adopt healthy nutritional practices
- Avoid trauma or injury
- Avoid prolonged sitting, standing, or crossing of legs
- Elevate legs above heart several times a day
- Practice isotonic calf muscle exercises
- Monitor the chair-bound patient
- Seek professional help with increased swelling or pain
- Monitor for signs of variceal bleeding at sites of varicosities

SOURCE: WOUND HEALING SOCIETY GUIDELINES, 2007

Lymphedema: an overview

Classifications of lymphedema



Primary (idiopathic)

- Congenital
 - Simple
 - Hereditary or familial (Milroy's disease)
- Lymphedema praecox
- Lymphedema tarda

Secondary

- Obstructive
 - Malignant occlusion by metastasis or invasion
 - Interruption of lymphatic channels or nodes by surgery or radiation therapy

Inflammatory

- Nontopical
- Topical

SOURCE: SCHIRGER AND GLOVICZKI, 1990

Lymphedema is divided into two types—primary (idiopathic) and secondary, and is based on the underlying cause. Primary lymphedema may be present at birth or the associated swelling can emerge later in life with no identifiable cause, according to Alexander Schirger and Peter Glociczki (1990). In the U.S., an estimated one to two million people are diagnosed with primary lymphedema, which is not a progressive disease, and it is twice as common in women than in men. According to Susan Simmons Holcomb in a 2006 study, lymphedema also affects the lower extremities three times more often than the upper extremities and in two-thirds of cases it is bilateral.

There are three forms of lymphedema—congenital, lymphedema praecox, and lymphedema tarda. In congenital lymphedema, the absence or abnormality of the lymphatic tissue is clinically evident at birth [Holcomb]. Congenital lymphedema can be associated with other congenital abnormalities such as Turner's syndrome, congenital absence of nails, (referred to as yellow nail syndrome). Some children with this type of lymphedema have been known to have an extra set of eyelashes [Schirger et al]. The most common type of inherited primary lymphedema is lymphedema praecox, which occurs between birth and age 35 and accounts for 65-80 percent of all cases of primary lymphedema. Lymphedema tarda, also known as Meige disease, is the rarest form of the disease, and usually does not develop until

age 35. According to Schirger et al, it usually makes its appearance around puberty.

Diagnosing Lymphedema

Unlike patients with arterial or venous disease, most patients with lymphedema don't have any symptoms other than those related to the weight and size of the limb, notes Simmons. A complete patient assessment should include a history and physical examination. Patients should be asked about past surgeries, post-operative complications, radiation treatment, and the length of time between surgery or radiation, as well as the onset of the lymphedema [Simmons].

Secondary lymphedema, which can occur acutely in some patients, is the most commonly recognized type of lymphedema, and affects two to three million in the U.S. It may be caused by obstructions of the lymphatic pathways due to trauma, surgery, irradiation, or replacement of lymph channels or lymph nodes by metastatic tumor or due to inflammatory changes [Schirger et al]. The hallmarks of inflammatory lymphedema are recurrent episodes of lymphangitis and cellulitis. In addition to intense malaise, systemic symptoms include fever, chills, headaches, nausea, vomiting, and diffuse sweating [Schirger et al].

There are four patterns of acute lymphedema. They include:

Mild acute lymphedema, which lasts just a few days, usually occurs after surgery

to remove a lymph node or is caused by injury to lymphatic vessels during surgery. Treatment includes elevating the affected extremity and contracting the muscles of the extremity to enhance blood flow, which increases lymphatic flow back to the heart [Simmons].

A second type of acute lymphedema occurs six to eight weeks after surgery or radiation to the affected area. When this occurs, the limb is usually very tender, warm to hot, and erythematous. Treatment for this type of lymphedema includes elevating the affected limb and administering anti-inflammatory agents to reduce the inflammation of the lymphatic vessels and/or veins [Simmons].

The third type of acute lymphedema affects the superficial lymphatic vessels and nodes following an insect bite or other minor injury in the area. Treatment of this includes elevating the affected limb and administering antibiotics prophylaxis, since the risk of infection is higher [Simmons].

The fourth and most common type of acute lymphedema affects the superficial lymphatic vessels and nodes. Often painful in the neck, shoulder, back, and hips, this form of acute lymphedema develops 18 to 24 months after cancer surgery, but could take years to develop. Treatment includes elevation, which may be more difficult due to the associated pain. Pain medicine and/or anti-inflammatory agents may provide some relief [Simmons].

Acute lymphedema usually resolves within six months, however some patients may develop chronic cases due to surgical drain leaking protein into the surgical site, paralysis, inflammation, paralysis of the affected limb, loss of lymphatic function in the area, and/or blockage of a vein by a blood clot or inflammation.

In general, treatment should aim at removing

as much liquid as possible from the affected extremity in order to maintain an edema-free state. Once all the possible fluid is removed, the patient should be measured for an elastic stocking (knee-length or thigh-length depending on the location of the edema). Edema can also be removed from the extremities by mechanically pumping them for two to eight hours so that they



are edema-free [Schirger et al]. Surgery also can be considered to reduce the swelling when all else has failed. ■

Treating proteinuria



Three aspects should be taken into consideration when treating the proteinuric patient. These include:

- Administering corticosteroid and immunosuppressive therapy, which is aimed at the immunologic mechanisms causing proteinuric renal disorders.
- Correcting the clinical effects of proteinuria, which addresses the problems of edema, hyperlipidemia, thromboembolism, and infection often associated with it.
- Taking measures to reduce the quantity of urinary protein by using an ACE-I or ARB, which may decrease the subsequent development of overt proteinuria and clinical diabetic nephropathy.

SOURCE: K.K. VENKAT, 2004

The International Society of Lymphology Staging System

- Stage 0:** Subclinical condition where swelling is not evident despite impaired lymph transport. May exist months or years before edema occurs.
- Stage I:** Pitting may occur and is reversible with a few hours of rest and elevation.
- Stage II:** Pitting occurs and the edema is not significantly reduced when affected limb is elevated. In late Stage II, the tissue hardens and becomes fibrotic and pitting no longer occurs.
- Stage III:** Pitting is absent; skin changes, such as acanthosis, fat deposits, and warty overgrowths, could develop. Fluid may ooze from skin. It most commonly occurs in the legs and results from longstanding, inadequately treated or untreated lymphedema. ■

SOURCE: SIMMONS, 2006

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QUESTIONS OR COMMENTS?

Contact Erica Park at 888.332.0202 or Erica.Park@nationalhealing.com

Wound Healing Perspectives

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Erica Park, Editor
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Marcia Resta, Layout & Design

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