Breast cancer is the most common cancer in women, with an estimated 203,500 new invasive cases diagnosed in 2002 (American Cancer Society [ACS], 2002). Although breast cancer is the second leading cause of cancer deaths in women, with an estimated 39,600 deaths in 2002, death rates have declined during the past decade, with the largest decline in younger women (ACS). Five-year relative survival rates by stage at time of diagnosis now are 96% for local stage tumors, 78% for regional stage tumors, and 21% for metastatic breast cancer (ACS). These statistics suggest that a growing number of women will survive breast cancer. About 2.5 million breast cancer survivors live in the United States (Col et al., 2001).

Lymphedema is a serious problem for many breast cancer survivors. Lymphedema results from an imbalance in capillary filtration and lymph drainage (Ramos et al.; Stanton, Levick, & Mortimer, 1997) and often is chronic and disfiguring. Prevalence of lymphedema appears to be influenced by type of breast cancer treatment (Hull, 2000) and infections (Coward, 1999). Lymphedema can occur during treatment or many years later (Ramos et al.; Stanton, Levick, & Mortimer, 1997) and often is chronic and disfiguring. Prevalence of lymphedema appears to be influenced by type of breast cancer treatment (Hull, 2000).
and is about 28% (Logan, 1995), with a range of 6.7%–62.5% reported in the literature (Passik & McDonald, 1998). Women with lymphedema view it as a constant reminder of cancer, may feel less sexually attractive, lose fine motor movement in the affected extremity, and modify their wardrobes to accommodate enlarged arms (Carter, 1997). They may be forced to change jobs because of their inability to lift, may require assistance dressing, often limit social and recreational activities, and may experience depression (Passik & McDonald). Tobin, Lacey, Meyer, and Mortimer (1993) found that women who experience lymphedema may suffer negative psychosocial effects, such as depression, anger, change in body image, disturbance in sexual relationships, social avoidance, and poor adjustment to cancer. They may see the affected limb as ugly and deformed (Farncombe, Daniels, & Cross, 1994). Woods, Tobin, and Mortimer (1995) administered the Psychological Adjustment to Illness Scales to 100 women, 50 with lymphedema and 50 without lymphedema, and reported that six months after lymphedema treatment, 81% of women with lymphedema experienced poorer psychosocial adjustment in one or more domains: healthcare orientation; vocational, social, and domestic environment; sexual relations; extended family relations; and psychological distress. Passik, Newman, Brennan, and Holland (1993), in a case presentation of a 55-year-old breast cancer survivor with lymphedema, reinforced that depression and overall limited functional ability may occur in some women who develop lymphedema after treatment for breast cancer.

Pain also may be associated with lymphedema. Newman, Brennan, and Passik (1996) reported pain in 34.8% of patients undergoing rehabilitation for lymphedema and maintained that pain was correlated highly with distress, impaired functioning, and decreased libido. Additionally, heaviness, swelling, pain, and physical disfigurement in the affected limb may limit physical activities (Newman et al.; Passik & McDonald, 1998).

Clearly, lymphedema exacts tremendous psychological and physical costs on breast cancer survivors who experience it. Nurses who work with women with breast cancer must be prepared to educate them adequately about the risk of developing lymphedema, explain precautions that may reduce that risk, and urge them to seek care promptly if they experience symptoms of lymphedema. To best accomplish these goals, oncology nurses must understand the normal physiology of the blood capillary-interstitial-lymphatic vessel interface and the pathophysiology subsequent to treatment for breast cancer that leads to the development of lymphedema. The purposes of this article are to review the normal physiology of the blood capillary-interstitial-lymphatic vessel interface, describe the pathophysiology of lymphedema secondary to treatment for breast cancer, and summarize the physiologic bases of the current National Lymphedema Network (NLN) risk reduction guidelines.

Blood Capillary-Interstitial-Lymphatic Vessel Interface

The blood capillaries and lymphatic system provide mechanisms for fluid exchange at the blood capillary-interstitial-lymphatic vessel interface (see Figure 1). Under normal circumstances, filtration pressures at the arterial side of a capillary force fluid and protein into the interstitium and reabsorption pressures pull most of the fluid back into the capillary at the venous side. The remainder of the filtered fluid and protein are removed by lymphatic vessels. Without a functioning lymphatic system, protein, cells, fat, and nonreabsorbed fluid remain in the interstitium and cause death within about 24 hours (Guyton & Hall, 2000).

Four primary pressures influence the movement of fluid into the interstitium from the arterial side of the capillary: capillary pressure, interstitial fluid pressure, plasma colloid osmotic (i.e., oncotic) pressure, and interstitial fluid colloid osmotic (i.e., oncotic) pressure (see Table 1). Capillary pressure, negative interstitial fluid pressure, and interstitial fluid colloid osmotic pressure collectively exert about 41 mmHg outward pressure, and plasma colloid osmotic pressure exerts 28 mmHg inward pressure, resulting in a net filtration pressure of 13 mmHg from the arterial side of the capillary (Guyton & Hall, 2000). Although most capillary pores are too small for protein to pass through along with fluid out of the capillary, some pores are large enough to allow small amounts of protein into the interstitium.

Reabsorption of fluid from the interstitium takes place at the venous side of the capillary. Capillary pressure, negative interstitial fluid pressure, and interstitial fluid colloid osmotic pressure exert about 21 mmHg outward pressure, and plasma colloid osmotic pressure exerts 28 mmHg inward pressure, resulting in a net venous reabsorption pressure of 7 mmHg (Guyton & Hall, 2000). Ninety percent of the fluid filtered into the interstitium from capillaries is reabsorbed into the venous side. The remaining 10% of fluid and protein is removed from the interstitium by small, terminal lymphatic vessels.

Interstitial proteins and fluid easily enter the lymphatic system through lymphatic vessel anchoring filaments that are attached to both the endothelial cells in the vessels and surrounding connective tissue. These edges overlap, creating small valves that open into capillaries, which allows entry of protein (Guyton & Hall, 2000). Once inside a lymphatic vessel, these one-way valves provide a mechanism to return protein through the lymphatic vessels to the venous system. The small vessels in the interstitium empty into a series of deeper vessels that, in turn, empty into larger lymphatic trunks that, in the case of the arm, run through nodes in the axilla and finally drain into the left thoracic duct or right lymphatic duct (see Figure 2) (Mellor et al., 2000). Fluid and proteins are pumped through lymphatic vessels by three mechanisms: contraction of segments between each valve that, when full, push lymph to the next segment; compression of vessels from external forces such as contraction of surrounding muscle tissue, extremity movement, and arterial pulsation; and contractile actomyosin filaments in the lymphatic vessel endothelial cells (Guyton & Hall). The left thoracic duct drains lymph from the entire left side of the body, the right leg, and the entire abdomen into the left subclavian vein (Seeley, Stephens, & Tate, 2001). The right lymphatic duct drains lymph from the right side of the head and neck, right side of the thorax, and right arm into the right subclavian vein, right internal jugular vein, and right brachiocephalic vein (Seeley et al.).

Many factors influence the forces that maintain fluid equilibrium at the blood capillary-interstitial-lymphatic vessel interface. Any condition that increases arterial capillary pressure, interstitial fluid proteins, or capillary permeability or decreases plasma colloid osmotic pressure alters equilibrium and increases fluid movement out of arterial capillaries into the interstitium. Occlusions or damage to either the venous side of capillaries or to the lymphatic system may decrease reabsorption or lymphatic vessel drainage of fluid and protein, causing lymphedema.
Pathophysiology of Lymphedema After Treatment for Breast Cancer

Upper extremity lymphedema that develops after treatment for breast cancer is thought to be related to the extent of axillary node involvement, type of breast surgery, and radiation treatments that include lymph nodes in the radiation field. Healthcare professionals widely believe that lymphedema is less likely to occur with less extensive axillary node involvement, more conservative breast surgery, and a lack of radiation to lymph nodes (Meek, 1998; Velanovich & Szymanski, 1999). Damage to the axillary lymphatic system by surgery, nodal resection, radiation, and infection leads to decreased lymphatic drainage and stasis of fluid in that extremity. Recent research has suggested that alterations in hemodynamic factors, if they are not primary causes of lymphedema, contribute to sustaining lymphedema after treatment for breast cancer (Mortimer, 1998). Thus, to understand potential risk factors of lymphedema after treatment for breast cancer, nurses must consider damage to the lymphatic system and hemodynamic factors.

Damage to Lymphatic Structure and Tissues

Various types of damage to the lymphatic system may occur during or after treatment for breast cancer.

- Surgical removal of nodes and breast tissue and tied off lymph vessels obstruct drainage channels and diminish the carrying capacity of the lymphatic system.
- Scarring and fibrosis of lymph structures and surrounding tissues secondary to radiation obstruct lymphatic drainage, damage lumens of lymphatic trunks, and alter cell membranes of lymphatic vessels.
- Infection may result in fibrosis of lymph structures and surrounding tissues and further diminish the transport capacity of an already-compromised lymphatic drainage system.

These sources of damage render the lymphatic system unable to transport the normal amount of fluid and protein from the affected area. Such damage is believed to be the primary reason that lymphedema occurs after treatment for breast cancer (Foldi, Foldi, & Clodus, 1989).

Pathologic changes in the lymphatic system have been documented by Koshima, Kawada, Moriguchi, and Kajiwara (1996), who studied 14 patients with lymphedema, six of whom had postmastectomy lymphedema and were undergoing microsurgical lymphaticovenous anastomoses (i.e.,

Table 1. Pressures at the Blood Capillary-Interstitial-Lymphatic Vessel Interface

<table>
<thead>
<tr>
<th>Arterial Side</th>
<th>Venous Side</th>
</tr>
</thead>
<tbody>
<tr>
<td>Filtration pressures</td>
<td></td>
</tr>
<tr>
<td>Capillary pressure</td>
<td>Plasma colloid osmotic pressure</td>
</tr>
<tr>
<td>Negative interstitial fluid pressure</td>
<td>Total outward pressure</td>
</tr>
<tr>
<td>Interstitial fluid colloid osmotic pressure</td>
<td>Total inward pressure</td>
</tr>
<tr>
<td>Total outward pressure</td>
<td>Net arterial filtration pressure</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reabsorption pressures</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma colloid osmotic pressure</td>
<td>Total outward pressure</td>
</tr>
<tr>
<td>Capillary pressure</td>
<td>Net venous reabsorption pressure</td>
</tr>
<tr>
<td>Negative interstitial fluid pressure</td>
<td></td>
</tr>
<tr>
<td>Interstitial fluid colloid osmotic pressure</td>
<td></td>
</tr>
</tbody>
</table>
insertion of a graph from lymph nodes or vessels to the nearby venous system) (Brennan & Miller, 1998). In one case of postmastectomy lymphedema, some degeneration of smooth muscle cells at the lymphatic trunk was found. In a second case, dilatation and partial loss of the truncal media was found in one trunk and small recanalized channels in another trunk in the same limb. As stated earlier, muscle contraction propels lymph forward into the venous system; damage to the musculature of the lymphatic trunks, as described by Koshima et al., may diminish the effectiveness of lymphatic drainage, resulting in accumulation of fluid and protein in interstitial spaces.

Damage to the blood capillary-interstitial-lymphatic vessel interface also has been found in breast cancer survivors who do not have lymphedema. Two years after conclusion of treatment in women who had modified radical mastectomies, removal of nodes, and radiation therapy but no lymphedema, lymphoscintigraphy (i.e., radiographic visualization of lymph structures after injection of a radiotracer) revealed decreased isotope carrying times in 60% of the subjects and decreased axillary storage in 90%, indicating lymphatic damage (Goltner, Gass, Haas, & Schneider, 1988). Additionally, 30% of the women had lymphostasis of the upper arm and 40% had absent axillary nodes. In the same study, two years after treatment, 92% of women with lymphedema had delayed lymph carrying times, 100% had altered axillary storage, 70%–85% had lymphostasis of the upper arm, and 66% had absent axillary nodes (Goltner et al.). The study suggested that damage to the lymphatic system might be present in almost all women who have breast surgery and opened to scientific debate what conditions may cause some women to develop and other women not to develop lymphedema.

Radiation therapy increases the risk of developing lymphedema after treatment for breast cancer about twofold (Bourgeois, Fruhling, & Henry, 1983; Isaksson & Feuk, 2000; Mortimer et al., 1996; Tengrup, Tennvall-Nittby, Christiansson, & Laurin, 2000). Radiation may not damage lymph vessels directly but may cause fibrosis of surrounding tissues that results in constriction of vessels and delays growth of new lymphatic vessels in damaged tissues (Fajardo, 1994). Lymph nodes, however, frequently are damaged directly by radiation. Nodal lymphocytes are depleted and replaced by fatty deposits. Fibrosis of the nodes develops over time, preventing passage and filtration of lymph fluid. Both processes inhibit the lymphatic system’s ability to transport lymph into the venous system. The inhibition of new vessel growth may account for early onset lymphedema after breast irradiation, and fibrosis may contribute to later onset lymphedema (Meek, 1998).

Infection and inflammation are believed to damage the lymphatic system and trigger lymphedema (Harwood & Mortimer, 1995; Rockson, 2001). Infection in an affected limb often is difficult to treat because of lymphostasis and may cause fibrosis, which further compromises the lymphatic drainage system (Meek, 1998). Inflammation caused by a process such as cellulitis or trauma to lymph structures also contributes to the development of fibrosis. Such fibrosis may occlude lymph vessels and decrease lymph transport capacity. Danese, Howard, and Bower (1962), in studies of dogs undergoing transection of the posterior thigh, documented that infection in surgical wounds permanently damaged the lymph transport system. Segerstrom, Bjerle, Graffman, and Nystrom (1992) found a strong association between lymphedema and recurrent infections in a study of 136 women who had undergone treatment for breast cancer. In the study, nine participants reported a history of soft tissue infection in the affected arm and eight of those nine (89%) developed lymphedema.

**Hemodynamic Factors**

**Increased arterial flow:** Studies suggest that breast cancer treatment may cause increased arterial blood flow. Using Doppler ultrasound, Svensson, Mortimer, Tohno, and Cosgrove (1994) identified the presence of increased arterial flow to affected arms after breast cancer surgery. Doppler ultrasound measured the mean arterial blood flow in 76 subjects who had undergone surgical treatment for breast cancer, 50 with and 26 without lymphedema. Affected arms were compared to nonaffected arms in both groups. Data indicated a 38% mean increase of arterial blood flow in nonswollen affected limbs when compared to contralateral limbs and a 68% increase in swollen limbs. Although the cause of increased arterial flow has yet to be identified definitively, Mortimer (1998) speculated that vasodilatation of vessels or formation of new blood vessels were potential contributing factors, citing previous research findings that local vasodilator control was impaired in postmastectomy lymphedema, without sustained vasodilation and increased blood capillary density in swollen limbs (Roberts et al., 1994). If increased arterial pressure occurs in an affected arm after treatment for breast cancer, the increased filtration rate of fluid and protein from the arterial side of capillaries could raise the interstitial fluid level. To prevent lymphedema, this fluid would require either reabsorption into the venous end of capillaries or transport by lymphatic drainage. A damaged lymphatic system could not transport additional fluid; therefore, lymphedema would occur.
Impaired venous return: Obstruction by fibrosis in the axillary collateral venous systems of women with upper extremity lymphedema was documented in a classic article by Hughes and Patel (1966). Various degrees of obstruction were found with radiation therapy and infections suspected to be contributing factors. Surgical removal of fibrosis resulted in reduction of arm lymphedema. More recently, in a study of 81 women, 70% had abnormal venous return in the affected limb (Svensson et al., 1994). Inadequate reabsorption of fluid would result in a rise in interstitial fluid, alter venous capillary-interstitial-lymphatic vessel interface pressures, and cause lymphedema. This finding has been the subject of much debate in the literature. Martin and Foldi (1996) presented data, contrary to the findings of Svensson et al., that axillary-subclavian occlusion occurred in only 14.5% of 40 women with lymphedema after treatment for breast cancer. Martin and Foldi stated that no concrete evidence was found to suggest that an impaired venous system contributes to the development of lymphedema. Although controversial, the influence of an impaired venous return warrants further investigation.

Changes in Colloid Osmotic Pressure

Historically, lymphedema occurring after breast cancer treatment has been viewed as high protein edema because of the increased amount of protein that remains in the interstitium because of impaired lymphatic transport. However, research conducted by Bates, Levick, and Mortimer (1993) on 51 women who had surgery or radiation for breast cancer and developed lymphedema found otherwise. In the study, limb size, interstitial fluid composition, serum protein content, colloid osmotic pressure, interstitial fluid pressure, and arterial and venous pressures were measured in both swollen and normal arms of each subject. Protein was found to be more concentrated in the nonswollen limbs’ interstitial fluid than in the lymphedematous limbs—an unexpected finding (Bates et al., 1993, 1994). The researchers concluded that pressures opposing filtration were less than venous pressure in normal limbs and that interstitial osmotic pressure was reduced in swollen arms. To explain the results, the authors hypothesized that a rise in arterial capillary blood pressure increased microvascular fluid filtration, actual capillary permeation of plasma protein was reduced, or degradation of interstitial protein by macrophages increased (Bates et al., 1994). In response, Foldi (1995) questioned the findings based on the methodology used in the study. Mortimer, Levick, and Stanton (1995), however, defended the study methods, maintaining that they were scientifically sound and that further investigation was indicated. In a subsequent study, increased microvascular filtration was not identified in lymphedematous limbs (Stanton, Holroyd, Mortimer, & Levick, 1999) and the researchers softened their stance that it contributed to the development of lymphedema after treatment for breast cancer.

Table 2. Grades of Lymphedema

<table>
<thead>
<tr>
<th>Grade</th>
<th>Physiologic Changes in Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Swelling and pitting with applied pressure</td>
</tr>
<tr>
<td>II</td>
<td>Firm swelling, no pitting under pressure, skin changes, and hair loss</td>
</tr>
<tr>
<td>III</td>
<td>Swelling, thick skin, and large skin folds</td>
</tr>
</tbody>
</table>

Figure 3. National Lymphedema Network Risk Reduction Guidelines

Note. Guidelines courtesy of the National Lymphedema Network. Reprinted with permission.
Once these pathologic processes produce lymphedema, if left untreated, it may progress through three grades of severity (see Table 2) (Pain & Purushotham, 2000). Initially, in grade I, a limb swells and pits with pressure. Elevation may relieve swelling. In grade II, a limb becomes firmer but does not pit and skin changes may be noted. In grade III, elephantiasis results in very thick skin and large skin folds. All grades diminish the level of functioning of an affected limb, making prevention of lymphedema a clinical priority in the care of breast cancer survivors. Early diagnosis and treatment may prevent progression of lymphedema to grades II and III.

**Physiologic Basis for Risk Reduction Guidelines**

Both the blood capillary-interstitial-lymphatic vessel interface and lymphatic structures apparently are compromised by treatment for breast cancer; additional stress to these compromised systems, such as infection or inflammation, contributes to the development of lymphedema. NLN developed 18 risk reduction guidelines (see Figure 3) to decrease the risk of lymphedema after treatment for breast cancer (NLN, 2001). Although NLN states that anecdotal reports support the guidelines, it acknowledges having been criticized for not having evidence-based support for them (NLN). Despite the lack of evidence-based research, most of the guidelines have a solid foundation in physiology and would, if followed, decrease the risk of injury to affected arms and theoretically reduce the risk of developing lymphedema (see Table 3). The guidelines aim to prevent infection, decrease trauma to the lymphatic system, and lessen damage to the venous system. As previously discussed, all of these may be factors in the development of lymphedema. For example, guidelines that caution against having blood pressure taken on affected arms, carrying a heavy shoulder-strap purse, wearing tight jewelry, or wearing a heavy prosthesis on the affected side are intended to reduce arterial pressures that may further damage an already-compromised lymphatic system (NLN). Guidelines that encourage meticulous hygiene of affected limbs, protection from sunburn or extreme temperature changes, avoidance of activities that may result in cuts to the skin, and use of electric razors decrease the risk of infection and inflammation. Avoiding venipuncture in affected limbs decreases the risk of inflammation. Encouraging consultation with a healthcare provider immediately if swelling, redness, or pain develop is in keeping with the findings of Ramos et al. (1999) that the smaller the volume of lymphedema present when treatment is started, the better the outcomes of therapy. The guidelines also caution against activities that can increase arterial blood flow, such as the use of hot tubs and excessive movement against resistance, because they may result in increased interstitial fluid.

Although these guidelines may seem restrictive, they are designed to maintain what may be a fragile balance between capillary filtration and lymphatic drainage that exists in the arms of women who have been treated for breast cancer. Research addressing the effectiveness of these guidelines would be helpful if conducted in an ethical manner that did not increase subjects’ risk of developing lymphedema. However, until evidence-based research contradicts the effectiveness of these guidelines, nurses should educate patients with breast cancer about their risk for lymphedema, NLN guidelines, and the physiologic rationale behind them.

### Table 3. Physiologic Basis for Guidelines

<table>
<thead>
<tr>
<th>Guideline</th>
<th>Infection/</th>
<th>Increased</th>
<th>Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Infammation</td>
<td>Lymphatic</td>
<td>Changes&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Report swelling of limb.</td>
<td>X</td>
<td>–</td>
<td>X</td>
</tr>
<tr>
<td>No blood draw or injection</td>
<td>X</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>No blood pressure</td>
<td>–</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Keep arm clean and dry: apply lotion after bathing</td>
<td>X</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Avoid vigorous repetitive movement against resistance</td>
<td>–</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>No lifting of heavy objects</td>
<td>–</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>No tight jewelry or elastic bands</td>
<td>–</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Avoid extreme temperature changes</td>
<td>–</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Avoid cuts or abrasions</td>
<td>X</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Do not cut cuticles</td>
<td>X</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Do not over exercise</td>
<td>–</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Rest arm if it begins to ache</td>
<td>–</td>
<td>X</td>
<td>–</td>
</tr>
<tr>
<td>Wear a compression sleeve when flying</td>
<td>–</td>
<td>–</td>
<td>X</td>
</tr>
<tr>
<td>Wear lightweight breast prostheses</td>
<td>–</td>
<td>–</td>
<td>X</td>
</tr>
<tr>
<td>Use an electric razor when shaving axilla</td>
<td>X</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Report rash, itching, pain, redness, or warmth</td>
<td>X</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Maintain ideal body weight; avoid smoking and alcohol</td>
<td>–</td>
<td>–</td>
<td>X</td>
</tr>
<tr>
<td>If you have lymphedema, wear a compression sleeve during waking hours</td>
<td>–</td>
<td>–</td>
<td>X</td>
</tr>
</tbody>
</table>

<sup>a</sup> at blood capillary-interstitial-lymphatic vessel interface

**Conclusion**

Oncology nurses should understand the normal physiology of the blood capillary-interstitial-lymphatic vessel interface and the pathophyslogic changes that contribute to the development of lymphedema in women after treatment for breast cancer. With this knowledge, they will be better able to educate their patients. They must inform patients of their lifelong risk of developing this chronic, disfiguring treatment complication and make immediate referrals for treatment should arm swelling be found. Additionally, providing such information to women at risk will enable them to make informed decisions about what, if any, lifestyle changes they need to make to reduce their risk of developing lymphedema.

**Author Contact**: Sheila H. Ridner, MSHA, MSN, RN, ACNP, can be reached at Sheila.Ridner@vanderbilt.edu, with copy to editor at rose_mary@earthlink.net.
References


For more information . . .

➤ National Lymphedema Network  
www.lymphnet.org/  
➤ Circle of Hope Lymphedema Foundation  
www.lymphedemacircleofhope.org/

These Web sites are provided for information only. The hosts are responsible for their own content and availability. Links can be found using ONS Online at www.ons.org.