

## VENOUS DYNAMICS IN LEG LYMPHEDEMA

D.I. Kim, S. Huh, J.H. Hwang, Y.I. Kim, B.B. Lee

Division of Vascular Surgery (DIK,SH,YIK,BBL) and Department of Rehabilitation (JHH), Samsung Medical Center, College of Medicine, Sung kyun kwan University, Seoul, Korea

### ABSTRACT

*To determine whether there is anatomical and/or functional impairment to venous return in patients with lymphedema, we examined venous dynamics in 41 patients with unilateral leg lymphedema. A Volometer® was used for computer analysis of leg volume, a color Duplex Doppler scanner was used to determine deep vein patency and skin thickness, and Air-plethysmography was used to assess ambulatory venous pressure, venous volume, venous filling index and the ejection fraction.*

*In the lymphedematous leg, volume and skin thickness were uniformly increased ( $126.4 \pm 21.3\%$  and  $156.9 \pm 44.5\%$ ) (mean  $\pm$  S.D.), respectively. The ambulatory venous pressure was also increased ( $134 \pm 60.7\%$ ) as was the venous volume ( $124.5 \pm 37.5\%$ ), and the venous filling index ( $134.5 \pm 50.5\%$ ). The ejection fraction was decreased ( $94.9 \pm 26.1\%$ ). Greater leg volume correlated with increased venous volume and venous filling index (values = 0.327, 0.241, respectively) and decreased ejection fraction ( $r = -0.133$ ). Increased subcutaneous thickness correlated with increased venous filling index and venous volume ( $r = 0.307, 0.126$ , respectively) and decreased ejection fraction ( $r = -0.202$ ).*

*These findings suggest that soft tissue edema from lymphatic stasis gradually impedes venous return which in turn aggravates the underlying lymphedema.*

Altered arterial and venous blood flow have been described in patients with

lymphedema. We wondered, however, whether functional venous insufficiency superimposed on chronic lymphedema contributes to worsening of soft tissue swelling. Accordingly, we studied peripheral venous dynamics using Doppler duplex scanning, Air-plethysmography, and a Volometer® for limb volume in 41 patients with leg lymphedema.

### CLINICAL MATERIAL

Three hundred fifty-nine patients were registered in our lymphedema clinic from February 1996 to July 1997. They included 8 men and 33 women with a mean age of  $46.5 \pm 15.8$  years. Eleven patients had primary and 30 had secondary lymphedema. Fifteen had right and 26 had left leg lymphedema. Mean duration of lymphedema was  $7.1 \pm 5.9$  years. Leg volumes (ml) were measured by Volometer® (Medizin Technik. BÖSL) which takes into account the cylindrical shape of the limb and via computer analysis calculates its volume. Subcutaneous thickness and venous flow patency were assessed by B-mode ultrasonography (Ultramark-9®, ACI) using a 5-10 MHz transducer. Ambulatory venous pressure (AVP), venous volume (VV), venous filling index (VFI), and ejection fraction (EF) were determined by Air-plethysmography (APG, ACI Medical, Inc., Sun Valley, CA). In brief, a special air chamber is fitted with the patient in a supine position and the leg is elevated on a heel support. The leg is kept in this position for 5

**TABLE 1**  
**Venous Dynamics With and Without Leg Lymphedema (Mean\_S.D.)**

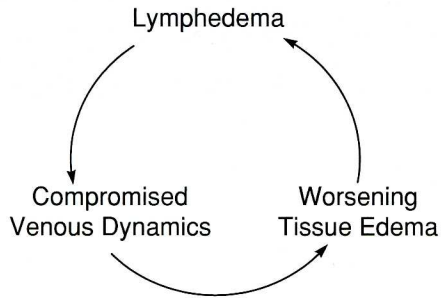
	Vol(ml) <sup>a</sup>	SC(cm) <sup>b</sup>	AVP(mmHg) <sup>c</sup>	VV(ml) <sup>d</sup>	VFI(ml/sec) <sup>e</sup>	EF(%) <sup>f</sup>
NL	3054±429	2.1±0.6	23.4±12.5	77.6±22.7	1.3±1.1	68.6±13.8
AL	3854±827	3.1±0.8	31.4±15.9	94.1±30.8	2.1±1.6	63.0±13.6
%	126.4±21.3	156.9±44.5	134±60.7	124.5±37.5	134.5±50.5	94.9±26.1
p	NL vs AL <sub>p</sub> <0.0003	<0.0001	<0.002	<0.0003	<0.0006	0.028
r values	a vs* b~f	0.553	0.022	0.327	0.241	-0.133
	b vs* c~f		-0.048	0.126	0.307	-0.202
NL=normal leg; AL=affected limb; %=AL/NL x 100; Vol=volume; SC=Subcutaneous thickness; AVP=ambulatory venous pressure; VV=venous volume; VFI=venous filling index; EF=ejection fraction r value=Pearson correlation coefficient						

minutes to allow for a stable leg and air chamber environment and to ensure resting arterial inflow. The patient then stands on a stool with his weight on the opposite leg. An increase in leg volume is the result of venous filling. When the chart recorder reaches a plateau, the leg veins are full and therefore represents the functional venous volume (VV). The venous filling index (VFI) is a measure of the average filling rate of the leg veins while standing and is expressed in milliliters per second. The patient then stands on both legs, performs one tiptoe movement, and returns to the initial position. The recorded decrease in VV as a result of calf muscle contraction represents the ejection volume (EV). The ejection fraction (EF) = EV/VV. After a new plateau is reached, the patient performs 10 tiptoe movements at a rate of one per second and then returns to rest. A volume decrease to a new steady state is the residual volume or the amount of blood remaining in the calf at the end of ten tiptoe exercises. The residual volume fraction (RVF) is the percentage of functional VV remaining

in the calf, and correlates directly with the ambulatory venous pressure (AVP) (1). To compare venous hemodynamic differences, the data were expressed as a percentage of the contralateral non-edematous leg. The data were represented as mean ± S.D. Statistical significance was determined by Student's t-test. P values less than 0.05 were considered significant.

## RESULTS

Each patient was shown not to have overt venous outflow occlusion or obstruction by B-mode ultrasonography. The leg volume increase in the lymphedematous leg was 126.4 ± 21.3% (p = <0.0003) and the % increase in subcutaneous thickness was 156.9 ± 44.5% (p = <0.0001). The % ambulatory venous pressure (AVP) was increased by 134 ± 60.7% (p = <0.002), the % venous volume (VV) by 124.5 ± 37.5% (p = <0.0003), the venous filling index (VFI) by 134.5 ± 50.5% (p = 0.0006). The ejection fraction (EF) was decreased (94.9 ± 26.1%) (p = 0.028). *Table 1*



*Fig. 1. Proposed vicious circle in lymphedema whereby soft tissue edema initiates progressive venous insufficiency thereby aggravating the underlying lymphedema.*

summarizes these differences. Increased leg volume was directly correlated with VV, VFI and EF ( $r = 0.327, 0.241, -0.133$ , respectively). Increased subcutaneous thickness was directly correlated with VFI, VV and EF ( $r = 0.307, 0.126, -0.202$ , respectively).

## DISCUSSION

Altered arm circulatory dynamics in patients with upper extremity lymphedema after treatment of breast cancer has been reported previously. Martin and Földi (2) studied 48 women and reported increased arterial inflow in the swollen arm compared with the non-edematous control arm. Svensson et al (3) studied 50 patients with similar findings. These workers concluded that increased arterial blood flow contributed to worsening of arm edema. Jacobsson (4), using venous occlusion plethysmography, arteriovenous  $pO_2$  differences and isotope clearance rates, also demonstrated a 42% increased arterial flow in 25 such lymphedematous arms. The physiologic basis for increased arterial flow in lymphedema is unclear although greater skin flow has been postulated. Blockx and Driessens (5) observed a 150% increase in arterial flow in a limb affected by stage I sympathetic dystrophy. Cooke and Ward (6) and Brunning et al (7) also described a correlation between increased

regional blood flow and edema in patients with sympathetic dystrophy. Together these reports suggest that increased arterial flow may be a consequence of decreased vasoconstrictor autoregulation and contribute to arm lymphedema after axillary dissection and irradiation for breast cancer. In 81 patients with arm swelling after breast cancer treatment, Svensson et al (8) observed a 57% restriction to venous outflow and 14% had overt venous congestion; only 30% had normal arm venous outflow. Hughes and Patel (9) and McIvor et al (10) reported a 57% incidence of venous blockage in postmastectomy lymphedema.

Although previous authors refer to structural impedance to venous return, we showed in our patients by Duplex Doppler scan that there was no anatomical obstruction to venous return in our patients with leg lymphedema. On the other hand, venous functional dynamics as determined by Air-plethysmography (APG) showed notable impairment. Our findings suggest that leg swelling from chronic lymphedema contribute to the changes of ambulatory venous pressure, venous volume, and ejection fraction and thereby tend to aggravate the underlying soft tissue edema (Fig. 1). In further support of this hypothesis, we have begun to examine venous dynamics in the early stages of lymphedema soon after radical hysterectomy with or without irradiation for treatment of cancer of the uterus.

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**Dong Ik Kim, M.D., Ph.D.**  
**Division of Vascular Surgery**  
**Samsung Medical Center**  
**50 IL Won Dong, Kangnam-Ku**  
**Seoul, 135-710, KOREA**  
**Tel.: 02-3410-3467**  
**Fax: 02-3410-0040**