Does severe venous insufficiency have a different etiology in the morbidly obese? Is it venous?

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Objective: **Chronic venous insufficiency (CVI) is the most common cause of leg ulcers. Patients with morbid obesity are remarkable for particularly recalcitrant ulcers. Because obesity is not specifically incorporated in CEAP or other venous scoring systems, we sought to characterize this group of patients more completely.**
Methods: **Patients with severe CVI (CEAP clinical class, 4, 5, and 6), and class III obesity (body mass index [BMI], >40) were reviewed. Findings from clinical and duplex ultrasound scan (DU) examinations were compared with the CEAP classification, its adjunctive venous clinical severity score, and sensory thresholds.**
Results: **A review of clinic records identified 20 ambulatory patients with a mean age of 62 years, a mean BMI of 52, and a mean weight of 164 kg (361 lbs); all but one had bilateral symptoms. No evidence of venous insufficiency was detected with DU in 24 of the 39 limbs. Although some valvular incompetence was detected with DU in 15 of 39 limbs, these abnormalities were widely dispersed between 28 sites; eight limbs had findings at only one site. Ulceration (mean area, 29 cm^2) was present in 25 limbs and necessitated 7 months for healing; 13 (52%) recurred at least once during a mean observation period of 36 months. The mean sensory threshold of 5.21 exceeded current risk thresholds used in diabetic screening programs. The distribution of CEAP clinical class was C4 (n = 14), C5 (n = 14), and C6 (n = 11). Increasing CEAP class correlated with an increased mean BMI of 47, 52, and 56, respectively ($P < .01$). CEAP also correlated with a rising mean venous clinical severity score of 10, 11, and 15, respectively ($P < .05$).**
Conclusion: **Patients with class III obesity had severe limb symptoms, typical of CVI, but approximately two thirds of the limbs had no anatomic evidence of venous disease. The association of increasing limb symptoms with increasing obesity suggested that the obesity itself contributes to the morbidity. (J Vasc Surg 2003;37:79-85.)**

Because chronic venous insufficiency (CVI) is considered the most common cause of leg ulcers, our clinical examination was supplemented with the noninvasive examination to determine cause, anatomic location, and pathophysiology (CEAP) of the disorder. Reported surveys of individuals with severe CVI have generally described a postthrombotic cause in 38% to 44% of limbs with these clinical findings.[1,2] In similar populations, superficial valvular reflux was reported in 56% to 88% and deep valvular reflux in 48% to 72% of limbs.[2-4] Thus, when screening our clinic population for a clinical protocol investigating the role of physical therapy for CVI, we were surprised and disappointed to find that many obese candidates with the typical clinical findings of severe venous insufficiency had little or no anatomic evidence of venous reflux or obstruction.

Skin changes (diffuse lower leg pigmentation, lipodermatosclerosis, healed ulceration, and active ulceration) were the distinguishing characteristics of CEAP clinical classifications 4, 5, and 6 and indicated greater severity of CVI. The limbs of obese individuals were remarkable for particularly recalcitrant ulcers, prolonged healing, and frequent recurrence of leg ulceration. Although commonly associated with venous insufficiency, obesity was not incorporated in the CEAP classification system.[5]

To characterize this group of limbs more completely, we evaluated morbidly obese individuals with clinical, duplex ultrasound scan (DU), and sensory examinations. The limbs then were classified with the CEAP system and its adjunctive venous clinical severity score (VCSS).[5-7]

METHODS

Obese patients with limb symptoms typical of CVI underwent evaluation in our Veterans Affairs clinics from 1998 to the present with a complete clinical and noninvasive examination. Individuals with CEAP clinical class 4, 5, and 6 were included; those with class 3, edema of venous origin without other findings, were excluded to reduce the difficult distinction between CVI and other diseases affecting fluid management. However, clinical factors influencing chronic fluid balance were assessed (ie, congestive heart failure, hepatic failure, and renal failure). Height, weight, and specific information regarding diabetes mellitus, coronary heart disease, and sleep apnea were recorded as factors known to increase the risk of mortality and morbidity related to obesity; limbs with lower extremity arterial occlusive disease were excluded if the ankle brachial index was less than 0.75. Age was also compared because the frequency of ulceration increases with age.

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Table I. Clinical factors (20 patients)

	Mean	*Median*	*Range*
Age (y)	62	59	51-78
Height (cm)	178.1	180.3	167-193
Height (in)	70	71	66-76
Weight (kg)	163.8	158	114-218
Weight (lbs)	361	348	252-481
BMI	52	52	40-63
Comorbid disease			
DM	10 (50%)		
CAD	8 (40%)		
CHF	8 (40%)		
Sleep apnea	5 (25%)		
Renal insufficiency	1 (5%)		

Percentages given are for number of patients out of 20.
DM, Diabetes mellitus; *CAD,* coronary artery disease; CHF, congestive heart failure.

Table II. Ulcer characteristics (25 of 39 limbs)

Mean area	29 cm^2
Mean time to heal	7 mo (227 d)
Never healed	2 limbs
Recurrence	13 of 25 (52%)
Usual location	Anterior/lateral calf

Obesity was determined on the basis of the body mass index (BMI), which was calculated as the weight in kg divided by the height in cm^2. Obesity is defined as BMI of more than 30; class II morbid obesity is defined as BMI 35 to 40, and class III as BMI 40 or more.[8,9] The risks of mortality and complications are increased for individuals with a higher classification, which is used to guide therapeutic recommendations and to individualize management. Inclusion in this report was restricted to subjects with BMI of 40 or more (class III).

Duplex ultrasound scan examinations were conducted on a Siemens Sonoline Elegra or a Siemens Quantum 2000 (Iselin, NJ), with 2.5-multihertz to 7.5-multihertz probes. An imaging survey evaluated both lower extremities for patency, compressibility, wall thickness, scarring, and thrombosis of any age. Spectral Doppler survey evaluated each vein for spontaneous phasic flow and response to augmentation. Sites examined included the common femoral, superficial femoral (proximal and distal), popliteal (proximal and distal), posterior tibial, peroneal, gastrocnemius, greater saphenous (above-knee and below-knee), lesser saphenous, and perforating veins of the thigh and lower leg. Limbs were evaluated in the standing position and reflux noted when retrograde flow persisted for greater than 0.5 seconds after distal compression and release.[10,11]

New methods of patient assessment incorporating the adjunctive VCSS and pressure aesthesiometry were introduced in 1999 to 2000.[6,7] Because this retrospective review includes patients who underwent evaluation from 1998 to the present, these scores were only applied to 27 of the 39 limbs. The VCSS has 10 items with severity rated from 0 to 3 by the examiner; it was reported as a numeric score, which ranged from 0 to 30 and increased linearly with the clinical classification. A mean score of 14.9 ± 2.88 was reported with C6 CVI.[12] Three items specific to ulcer severity were number, duration, and size; recurrence, which was not specifically scored, was also noted. The last VCSS item scored compliance with stocking use as 3 points and noncompliant individuals with 0 points; to facilitate compliance, gradient compression hosiery were provided at no cost.

Pressure aesthesiometry determined the sensory threshold for lower leg skin and has been related to the susceptibility to local injury. Scores are expressed as a logarithmic value that represents the pressure applied by a graded series of filaments. An increased risk of ulceration and limb loss has been documented with values of more than 5.07 in patients with diabetes mellitus.[13] In previous work, we characterized the sensory threshold in the supramalleolar skin to be <4.65 (range, 4.08 to 6.65) in limbs with C5 CVI.[7] Thus, higher sensory thresholds suggest that limbs with CVI may also be more susceptible to skin injury (ie, ulceration). Scores were generally assigned by one individual (FP) or by surgical resident staff under supervision.

Age, BMI, VCSS, and sensory threshold scores were compared between CEAP clinical classes. Patients and limbs with objective ultrasound scan findings (DU+) were compared with those who had no findings (DU−). Comparisons were made with Student *t* test, assigning significance when the *P* value was less than .05. Data were expressed as mean ± standard deviation. The review was approved by the Institutional Review Board. To simplify presentation of the data, comparisons between the clinical classes were analyzed on the basis of each limb. However, patient-specific data, age, and BMI were also compared by assigning each patient's "worst" or most severely involved limb on the basis of clinical class. The worst limb was selected because it was the most likely to limit patient behavior. Because VCSS and pressure aesthesiometry scores are limb specific, these data were evaluated with a comparison of limbs.

RESULTS

Twenty male patients (39 symptomatic limbs) had a mean age of 62 years, a mean height of 178 cm (70 in), and a mean weight of 164 kg (361 lbs). All 20 had class III morbid obesity, with a mean BMI of 52. All but one had bilateral symptoms consistent with apparent CVI. Diabetes mellitus was present in 10 patients (50%), coronary artery disease in eight, congestive failure in eight, and sleep apnea in five. Hepatic insufficiency was minimal, and only one individual had renal insufficiency on the basis of a creatinine level of 2.5 (Table I).

Twenty-five limbs had healed or active ulcerations. Mean ulcer size was 29 cm^2, and a mean of 7 months was necessary to heal. Two have never healed, with a mean follow-up of 3 years. Recurrent ulceration has occurred in 13 limbs; in addition, during preparation of the manu-

Table III. DU: Distribution of findings in 39 limbs

CEAP clinical class	*Duplex (+): 15 limbs with 28 sites of reflux*							*Duplex(-): (normal)*	*Total*
	Superficial		*Deep*		*Perforating veins*	*Total sites*	*No. of limbs*	*No. of limbs*	*No. of limbs*
	GSV	*LSV*	*CFV*	*POP*					
CEAP 4	1	—	—	2	2	5	4	10	14 (36%)
CEAP 5	8	3	—	3	4	18	8	6	14 (36%)
CEAP 6	2	1	1	1	—	5	3	8	11 (28%)
Total	11	4	1	6	6	28	15 (38%)	24 (62%)	39 (100%)

No limbs were identified with evidence of active or previous thrombosis; no profunda, superficial femoral vein, or tibial vein incompetence was detected.
GSV, Greater saphenous vein; *LSV,* Lesser saphenous vein; *CFV,* common femoral vein; *POP,* popliteal.

Table IV. Comparison of obesity (BMI), clinical severity (VCSS), and neuropathy according to CEAP clinical class

	BMI			*VCSS*			*Neurosensory Score*			
	No. of limbs	*BMI*	P *value*	*No. of limbs*	*VCSS*	P *value*	*No. of Limbs*	*Neurosensory score*	P *value*	*Groups compared*
CEAP 4	14	47 ± 7	.075	9	10 ± 4	NS	7	5.20 ± 0.8	NS	CEAP 4 versus 5
CEAP 5	14	52 ± 8	NS	11	11 ± 5	NS	10	5.06 ± 0.7	NS	CEAP 5 versus 6
CEAP 6	11	56 ± 7	.0053	10	15 ± 5	.018	10	5.38 ± 0.9	NS	CEAP 4 versus 6
CEAP 5 + 6	25	54 ± 7	.0094	21	13 ± 5	.05	20	5.22 ± 0.8	NS	CEAP 4 versus 5 + 6
All limbs	39	52 ± 8	—	30	12 ± 5	—	27	5.21 ± 0.8	—	—

Values expressed as mean ± standard deviation.
NS, Not significant.

script, one patient with C4 disease had progression to C6, active ulceration. Location of the ulceration was often atypical; the ulcers were located in the proximal anterior or lateral calf of 10 limbs. These data are summarized in Table II.

The limb distribution of CEAP clinical class is summarized in Table III. The distribution was 14 C4 (36%), 14 C5 (36%), and 11 C6 (28%). Limbs without noninvasive evidence of anatomic venous insufficiency had a similar CEAP distribution 10 C4 (42%), six C5 (25%), and eight C6 (33%).

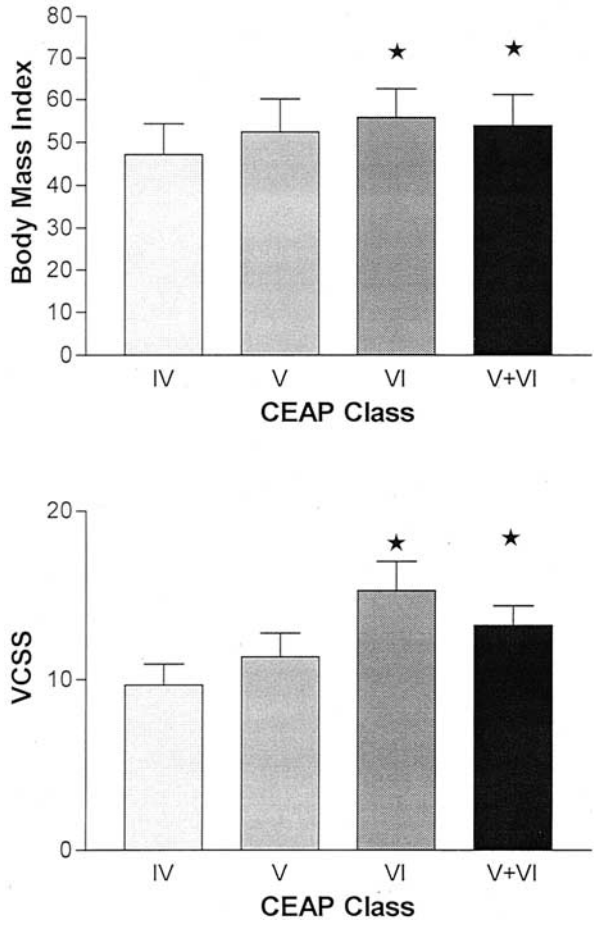
Ultrasound scan examinations of the lower extremity veins were normal in 24 of the 39 limbs (62%); neither reflux nor scarred vein walls nor obstruction was observed with DU in either the deep or superficial system of either limb in these patients. Thus, with only physical findings to evaluate, CEAP characterization of etiology, anatomy, and pathophysiology could not be assigned further in these 24 limbs.

Ultrasound scan examinations identified 28 sites of venous valvular incompetence in the remaining 15 limbs. Two subjects accounted for 14 of the 28 abnormal sites (50%). Bilateral saphenous and popliteal reflux was observed in one individual who had no DU evidence of old thrombus, despite a prior history of bilateral deep venous thrombosis. The second individual, whose ulcer recurred after a 20-lb weight gain, also had bilateral superficial incompetence. Both declined operative intervention. The remaining 11 limbs had venous incompetence in a total of 14 sites with DU. These were remarkable for a paucity of findings; eight limbs had only a single site of incompetence. The distribution was typical with a predominance of superficial reflux mixed with the deep and perforating veins. These findings are detailed in Table III.

BMI, VCSS, and neurosensory scores were compared between clinical classes C4, C5, and C6 and with the aggregate data of C5 with C6 (C5+6). These data are summarized in Table IV. VCSS scores were obtained on 30 of the 39 limbs, with a mean of 12 ± 5. An increase in BMI and VCSS was observed with increases in CEAP classification. Differences were observed in the comparisons between C4 and C6 and between C4 and C5+6 for both BMI and VCSS (Table IV; Fig). Compliance with elastic compression garments was poor at best; only two individuals claimed successful routine use. Pressure aesthesiometry was obtained in 27 of the 39 limbs; the mean neurosensory score was 5.21 ± 0.8 (range, 4.08 to 6.45). No differences were detected between CEAP classes in these obese patients.

In nine individuals, the two lower limbs differed with respect to CEAP clinical class. Therefore, to accurately present patient-specific variables (age and BMI), we also analyzed the data according to the patients worst limb (Table V) as opposed to analysis on the basis of each limb. No age differences were identified between groups. Again, as with the individual limb analysis, significant differences between C4 and C6 and between C4 and C5+6 were observed for BMI (Table V).

Age, BMI (Table V), VCSS, and neurosensory scores (Table VI) were compared between those with no anatomic



Graphic comparison of CEAP clinical class for all 39 limbs, as represented in Table IV. In addition, as noted in text and Table V, when comparing BMI between clinical classes for worst limb, the same differences were observed. **A,** BMI. **B,** VCSS.

findings (DU−) and those with observed reflux (DU+). No differences were observed between age, BMI, or VCSS; a difference was detected in sensory thresholds between those with (4.84 ± 0.6) and without (5.56 ± 0.8) venous abnormalities with DU (Table VI).

DISCUSSION

Severe limb symptoms, typical of CVI, increased in severity (CEAP class and VCSS) with the magnitude of the obesity (BMI). Approximately two thirds of these limbs had no anatomic evidence of venous disease. In contrast, published surveys of C4, C5, and C6 patients with similar venous symptoms reported that approximately 50% to 88% will have superficial incompetence and 48% to 72% will have deep vein incompetence.[1-4] Postthrombotic abnormalities were present in 44% of a C5 population evaluated with contrast venography and in 38% of the C4, C5, and C6 population reported by the North American Subfascial Endoscopic Perforator Surgery registry.[1,2] Both BMI and venous severity scores increased with CEAP clinical class, and no relationship to findings with DU was observed. These data suggest that class III morbid obesity should be considered a major contributing factor to severe symptomatology, whether it is venous in origin or not.

There are several alternative explanations for this discrepancy. First, there appears to be a correlation between the magnitude of the obesity and the severity of these presumably venous symptoms. Massive obesity probably produces a relative obstruction to lower extremity venous return similar to the effects of ascites. Although many of these individuals also had cardiac failure, this common comorbidity of morbid obesity does not explain the findings in those patients who did not have cardiac failure. The combined effects of increased demand on cardiac output may be additive to diminished venous return to produce central arteriovenous dysfunction.[14] In view of the excellent renal and hepatic function in these subjects, these would appear to be minimal factors. Another likely potential cause is lymphatic obstruction. Repetitive episodes of cellulitis would be expected to lead to scarring of the lymphatic channels. Perhaps more important, however, lymphatic return flow may also be compromised by the same adverse intraabdominal pressure hemodynamics as venous return flow. Perhaps it is a combined effect of lymphovenous hypertension.

We have also noted a strong association between recurrent ulceration and sensory impairment. Although localized sensory impairment has been associated with diabetes mellitus, leprosy, and now CVI, the relationship between the sensory abnormality and obesity remains undefined.[7,13] Clearly, there is considerable opportunity for additional investigations regarding the role of sensory impairment in this population.

Morbid obesity is now regarded as second only to cigarette smoking as the underlying cause of death in the United States, producing an estimated mortality rate of 280,000/y.[8,15] Obesity is becoming an increasingly common problem throughout the world, affecting both adult and pediatric populations, industrialized and developing nations.[8,9] The prevalence of class III morbid obesity doubled from 1.3% to 2.9% in the decades of the 1980s and 1990s.[8] Obesity, defined as a BMI of more than 30, is now estimated to have reached a prevalence of 19.8% (or 1/5) in the population of the United States.[8] Individuals considered to be at a particularly high risk are those with comorbidities of coronary artery disease, type 2 diabetes, and sleep apnea.[9] The risk of development of heart failure in relationship to obesity was recently characterized in men. The risk increased by 5% for each increment of 1 in BMI.[16]

In addition to the apparent manifestations of CVI, morbid symptoms attributed to severe obesity include gynecologic abnormalities, stress incontinence, hypertension, esophageal reflux, cirrhosis, cholelithiasis, intertriginous dermatitis, depression, pseudotumor cerebri, and advanced osteoarthritis of the hip and knee.[8,9,17] Limb-threatening popliteal arterial injuries have resulted from deceivingly innocuous mechanisms of injury.[18] Severe reductions in the

Table V. Comparison of severity of obesity and severity of CVI according to CEAP clinical class and DU findings (+ or −) for individual patients

Worst limb	*No. of patients*	*Age (y)*	P *value*	*BMI*	P *value*	*Groups compared*
	20	62 ± 8	—	51 ± 8	—	—
CEAP 4	4	67 ± 11	NS	44 ± 3	NS	C4:5
CEAP 5	7	62 ± 7	NS	49 ± 8	.06	C5:6
CEAP 6	9	59 ± 8	NS	56 ± 7	.001	C4:6
C5 + C6	16	61 ± 7	NS	53 ± 8	.042	C4:5 + 6
Duplex findings						
DU+	9	63 ± 8	NS	50 ± 8	NS	DU+:DU−
DU−	11	61 ± 8	—	52 ± 9	NS	—

Because age and BMI are patient-specific variables, patients were assigned according to their worst limb for this comparison.
Values expressed as mean ± standard deviation.
NS, Not significant.

Table VI. Comparison of severity of CVI and DU findings (+ or −)

All limbs	*No. of limbs*	*VCSS*	P *value*	*No. of limbs*	*Neurosensory*	P *value*	*Groups compared*
DU+	13	12 ± 5	NS	13	4.84 ± 0.6	.014	DU+:DU−
DU−	17	12 ± 5	—	14	5.56 ± 0.8	—	—
All limbs	30	12 ± 5	—	27	5.21 ± 0.8	—	—

Because VCSS and venous sensory neuropathy are limb specific, they were compared in groups defined by DU result.
Values expressed as mean ± standard deviation.
NS, Not significant.

quality of life and increased symptoms of bodily pain accompany morbid obesity.[19]

Individuals with excess body mass had their ulcerations heal more slowly and had recurrence more frequently than that reported for other venous patients. The 7-month mean time to healing greatly exceeded the 10-week interval reported by Marston et al[3] for 24-cm^2 ulcerations and the 54 days by the North American Subfascial Endoscopic Perforator Surgery registry.[2] If one accepts the premise that diminished protective sensation predisposed to injury in the gaiter area, the elevated sensory threshold of 5.21 offers one explanation for the frequency of ulceration in the morbidly obese population. Although many patients kept regular attendance, most were incapable of donning regular stockings, much less standard footwear, or a graduated compression garment dispensed from our clinic. Many obese individuals are physically incapable of reaching their feet, and most were unable to see the foot. A key clue was the slip-in sandal with no heel. The relative difficulty and slower deliberate pace of simple bodily functions (standing, sitting, lying down) may be additive to visual and sensory impairment in fostering skin breakdown leading to ulceration.

The normal methods for management of CVI are poorly used in this patient population for a variety of different reasons. Compression therapy was probably appropriate, but compliance was poor. Physical inability to place compression garments can be achieved by enlisting the support of a family member but is still limited by the need for custom measurement. An adjustable velcro garment is easier to apply but also requires additional personnel to don effectively. With diligence and persistence, most of these ulcers could be healed, but 50% recurred within 3 years. For comparison, the recurrence rate of venous ulceration was characterized by various reports as 29% at 5 years, 38% at 3 years, and 28% at 2 years.[2,3,20] Although decreased activity was likely associated with decreased calf pump function, little data existed to prove this supposition. Despite the major risks associated with gastric bypass, the reported results indicate that this may be one of the most effective management tools available.

Because of the fatal and morbid complications associated with uncontrolled obesity, surgical gastric bypass has become an increasingly common procedure.[8,9] Sugarman et al[17] suggested that secondary complications in the obese population were related to the effects of chronically elevated abdominal compartment pressures. They have presented evidence, in the form of bladder pressure measurements (mean, 19 cm H_2O) to corroborate this theory. Of direct relevance was the clinical reversal of venous symptoms (edema, ulceration) in individuals who achieved successful weight loss after surgical gastric bypass therapy.[17] Notably, their patients with venous stasis were the most obese (mean BMI of 61) and had greater comorbidity.

Our report compares anatomic results from duplex scan examinations to clinical findings but would be strengthened with the addition of hemodynamic data. We attempted to obtain physiologic data with air plethysmography but considered these examinations too variable and unreliable to justify reporting. Although duplex scan examinations of extremity venous structures were surprisingly

accessible, another potential weakness of this report was the indirect assessment of suprainguinal venous pathophysiology. On the other hand, other investigators have also suggested an effect of increasing BMI on severity of CVI independent of measured venous reflux.[21]

Finally, one could reasonably inquire whether these typical clinical findings of venous insufficiency satisfactorily represent bona fide venous disease. Should these individuals even be classified with the CEAP system? Or should the CEAP system be modified to include morbid obesity as an additional etiologic factor?

The usual anatomic causes of venous insufficiency were not sufficient to explain the severe extremity symptoms. Localized sensory impairment may contribute to the frequency of ulceration. These data suggest that class III obesity itself contributed to the apparent venous insufficiency. In support of this consideration were the magnitude of the venous symptoms, bilaterality, atypical location, and the association between increasing weight and severity of CVI.

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DISCUSSION

Dr Enrique Criado (Baltimore, Md). I have the impression that you are really dealing with venous insufficiency. But secondary to uncommon etiologic factors, those being, chronically sustained venous hypertension, secondary to increased hydrostatic pressure throughout the day. These patients have unusual habits. They very rarely lay flat, and they sleep typically with their heads elevated and dangling their limbs, thus rarely relieving the hydrostatic column from their lower limbs.

And secondly, you have to consider chronic failure of the calf muscle pump mechanism. These patients may have intact and effective calf muscles with normal ejection fractions as measured by air plethysmography, but they do not activate their calf muscle pump properly throughout the day. They walk very little, they tend to walk with their hips, barely using their calf musculature. Unfortunately, we do not have any means to measure these factors during prolonged periods of time, such as we can do for 24-hour EKG or blood pressure monitoring.

My question to you is, whether you agree with these two comments and whether you have any suggestions on how we could measure these factors over time in this particular population?

Dr Frank Padberg, Jr. I agree with you. I suspect that we will probably find an element of chronic venous hypertension. One method with which we could access this information would be by ambulatory venous pressures. And as you realize, that is going to be a bit difficult in this population.

We have tried. However, I cannot give you any results as yet. We also tried with air plethysmography. We were able to study a few limbs, but the results were relatively unreliable, for many reasons, one of which was that the test does require that the patient be somewhat facile in getting out of bed and onto the floor in a standing position while the reflux is occurring.

The chronic calf pump and the poor walking, I would agree, we see many of these folks that still do spend a lot of time sitting in

chairs. And I think the habit, and mobility, and a sedentary lifestyle all contribute to this, too.

Dr Karl A. Illig (Rochester, NY). Your data are fascinating in light of our own anecdotal experience. We have done superficial ablation and SEPS in seven or eight morbidly obese patients and really noticed a dramatic, almost immediate, healing of their ulcers. We have never taken this beyond anecdote, but my sense has always been that there is something unusual in the etiology of chronic venous disease in the morbidly obese, and whatever it is responds unusually well to surgery.

You report a very low incidence of surgically correctable disease. Have you operated on any of these or similar patients, and if so, how did they do?

Dr Padberg. That is an interesting question. I can recall at least one patient from our series of superficial ablations who was obese enough to qualify for this series, but his operation preceded this time frame. However, his course was consistent with the observations reported here—recurrence of ulceration and localized sensory impairment, despite improved venous hemodynamics.

Dr Andre M. van Rij (Dunedin, New Zealand). We have done a study in which we have looked at 1000 limbs and correlated weight against some of the functions that you talked about, particularly APG. One of the interesting features is that the heavier you are, the better your muscle pump. But the point might well be that in the very obese, despite a good muscle pump, sedentary activity may negate this and have something to do with worse venous function. However I do wonder, is this really venous disease, or is it something else?

Dr Padberg. So do we.

Dr Alan R. Koslow (Des Moines, Iowa). Apropos to some of the studies from Oxford in the 1980s that showed that lymphatic failure was actually the cause of a lot of venous stasis disease after DVT, I do lymph scintigrams on my patients who are obese like this, who have it, and I am finding marked lymphedema tartar in these patients.

The question I have for you is, if they do lose adequate weight, are you finding that the failure of lymphatic drainage is improving, or is this something that we need to do a study on?

And the other thing, I agree with your comment. When I see the patients, I tell them that there is nothing we can do to treat you until you lose weight, and I give them a packet from the University of Iowa Gastric Stapling Center.

Dr Padberg. I think you may be referring Mortimer's work that associated lymphatic disease with chronic venous insufficiency. Like you, I suspect there probably is a lymphatic role here as well. We have not studied this, so I cannot make any specific comments in relationship to this group of patients. However, if there is an obesity-related chronic venous hypertension, then there is probably also an obesity-related chronic lymphatic hypertension as well.

Like you, I believe that one of the options for these patients is weight reduction by surgical means. One series that has shown relief and improvement in the venous complications is referenced in the manuscript.