Obésité, inactivité physique et maladie veineuse

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Obesity and overweight

WHO Fact sheet N311. Updated January 2015

- a BMI greater than or equal to 25 is overweight
- a BMI greater than or equal to 30 is obesity.
- The fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended. Globally, there has been:
  - an increased intake of energy-dense foods that are high in fat; and
  - an increase in physical inactivity due to the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization.

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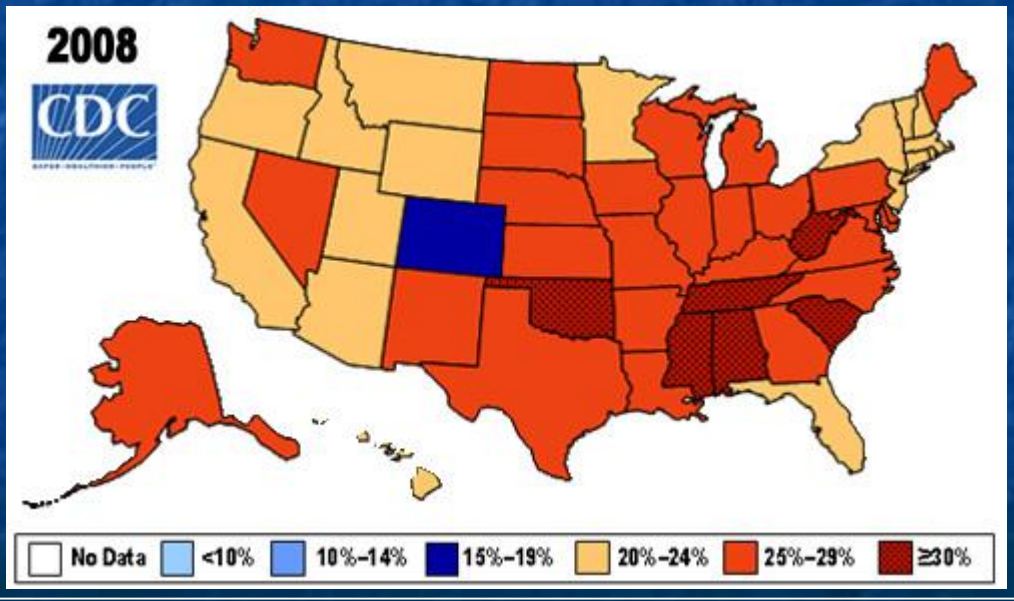
- Worldwide obesity has more than doubled since 1980.
- In 2014, more than 1.9 billion adults, 18 years and older, were overweight. Of these over 600 million were obese.
- 39% of adults aged 18 years and over were overweight in 2014, and 13% were obese.
- Most of the world's population live in countries where overweight and obesity kills more people than underweight.
- 42 million children under the age of 5 were overweight or obese in 2013.
- Obesity is preventable!!!

Increasing obesity rates

- Rates of obesity in Canadian boys have increased from 11% in 1980s to over 30% in 1990s
- During this same time period rates increased from 4 to 14% in Brazilian children.
- Obesity rates in US

  % with BMI > 30



Obesity and overweight

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- Raised BMI is a major risk factor for noncommunicable diseases such as:
  - cardiovascular diseases (mainly heart disease and stroke), which were the leading cause of death in 2012;
  - diabetes;
  - musculoskeletal disorders (especially osteoarthritis - a highly disabling degenerative disease of the joints);
  - some cancers (endometrial, breast, and colon).
- Childhood obesity is associated with a higher chance of obesity, premature death and disability in adulthood.
  - in addition to increased future risks, obese children experience breathing difficulties, increased risk of fractures, hypertension, early markers of cardiovascular disease, insulin resistance and psychological effects.
- Venous diseases are not mentioned

Pulsford et al: Sitting Behavior and Obesity: Evidence from the Whitehall II Study. A J Prev Med 2013;44:132–138

- To investigate cross-sectional and prospective associations between multiple sitting time indicators and obesity and examine the possibility of reverse causality.
- Whitehall II cohort, examine associations between:
  - prevalent obesity (BMI ≥30) at Phase 5 (1997–1999)
  - incident obesity between Phases 5 and 7 (2003–2004)
  - four levels of five sitting exposures (work sitting, TV viewing, non-TV leisure-time sitting, leisure-time sitting, and total sitting).
  - Using obesity data from three prior phases (1985–1988, 1991–1993; and recalled weight at age 25 years)
  - Linear regression models were fitted to examine the association between prior obesity and sitting time at Phase 5
- Sitting time was not associated with obesity cross-sectionally or prospectively.
- Prior obesity was prospectively associated with time spent watching TV per week but not other types of sitting.

Hu FB et al: Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. JAMA 2003;289:1785-91

- During 6 years of follow-up, 3757 (7.5%) of 50 277 women who had a BMI of less than 30 in 1992 became obese (BMI > or =30).
- Time spent watching TV was positively associated with risk of obesity and type 2 diabetes.
- In the multivariate analyses adjusting for age, smoking, exercise levels, dietary factors, and other covariates,
  - each 2-h/d increment in TV watching was associated with a 23% (95% confidence interval [CI], 17%-30%) increase in obesity
  - each 2-h/d increment in sitting at work was associated with a 5% (95% CI, 0%-10%) increase in obesity

Hamilton MT et al: Role of Low Energy Expenditure and Sitting in Obesity, Metabolic Syndrome, Type 2 Diabetes, and Cardiovascular Disease. Diabetes 2007;56:26552667

- Recent observational epidemiological studies strongly suggest that daily sitting time or low nonexercise activity levels may have a significant direct relationship with mortality, cardiovascular disease, type 2 diabetes, metabolic syndrome risk factors, and obesity.

An Epidemiologic Study of Risk Factors for Deep Vein Thrombosis in Medical Outpatients – The Sirius Study

(Samama 2000)

Case-Control study n=1272 (636 DVT, 636 controls)

	Risk	Odds Ratio
Intrinsic	anamnestic DVT	15,60
Factors	CVD	4,45
(basic risk)	cardial insufficiency	2,93
	BMI > 30 kg/m^2	2,39
	standing profession > 6 h	1,85
	> 3 pregnancies	1,74

Risk factors of PTS

Pesavento R, Villalta S, Prandoni P, Intern Emerg Med 2009

- Obesity increases the risk 3-5 X
- Proximal DVT
- Persistant symptoms 1 month after DVT
- Ipsilateral recurrent DVT (10 X)
- Varicose veins prior to DVT
- Insufficient anticoagulation

Lindsey Robertson et al: Risk factors for chronic ulceration in patients with varicose veins: A case control study. J Vasc Surg 2009;49:1490-8

- Aim: to determine, in subjects with varicose veins, the characteristics of venous disease and other factors associated with an increased risk of ulceration.
- Case control study:
  - 120 subjects with VV and C5 or C6
  - 120 controls with VV and no history of VLU.
- Results: increased risk of ulceration
  - severity of clinical venous disease, especially with the presence of skin changes (P <0.0001)
  - history of deep vein thrombosis (DVT) (P = 0.001)
  - higher body mass index (BMI) (P = 0.006)
  - smoking (P = 0.009)
  - reflux in the deep veins (P = 0.0001)
  - low pumping function in photoplethysmography (P < 0.05)
  - limited range of ankle movement (not wholly due to the effects of an active ulcer) (P < 0.05)

Bonn Vein Study
Questions

- Prevalence of venous disorders in urban and rural population
- Frequency of signs and symptoms
- Identification of risk factors

Bonn Vein Study

Risk Analysis for Telangiectases, VV and CVI

adjusted for age, gender, region of living, number of brothers and sisters

effector		Tel	VV	CVI
pos. family history VV		(+)	+	(+)
age		++	++	++
pregnancies		-	+	-
female gender	+	+	+	
obesity		+(f)	-	++
urban inhab.		-	-	+

Bonn Vein Study

Risk of obesity for Tel, VV and CVI

multivariate analysis, OR (95%CI) adjusted for age and region of living

effector	Tel		VV		CVI	
	m	f	m	f	m	f
BMI <18,5	0,7	0,3	*	2,0	*	0,9
BMI 25- < 30	0,9	1,8	0,9	1,3	2,7	1,6
BMI 30- < 40	1,3	2,9	1,3	1,9	6,5	3,1
BMI>40	*	*	1,8	1,6	10,5	7,9

* low number, combined with next category

Bonn Vein Study II

- **Identical population and procedure as in Bonn Vein Study I**
- **Start of follow-up: August 2007**
- **End of investigations: Oktober 2008**
- **Follow-up time: 6.6 years**
- **Response at follow-up: 84.6 % (n=1978)**

Bonn Vein Study II

- incidence of varicose veins -

All VV	**13.7 % (2.1%/year)**
Men	**13.7 % (2.1%/year)**
Women	**13.7 % (2.1%/year)**
<39 years	**7.1 % (1.1%/year)**
40-59 years	**13.2 % (2.0%/year)**
>59 years	**18.5 % (2.8%/year)**

Bonn Vein Study II

- risk factors for new varicose veins -

Risk factor for VV	Relative risk	Confidence interval	p
Women and men			
Age (per year)	1.03	1.01-1.04	<0.0001
Fam. history	1.52	1.10-2.09	0.01
Women only			
Pregnancy 1 vs 0	1.33	0.73-2.43	0.35
Pregnancy 2-3 vs 0	1.23	0.72-2.10	0.44
Pregnancy 4 vs 0	1.65	0.86-3.16	0.14

Bonn Vein Study II

- incidence of CVI (C3-C6) -

All CVI	**13.0 % (2.0%/year)**
Men	**12.7 % (1.9%/year)**
Women	**13.3 % (2.0%/year)**
<39 years	**3.9 % (0.6%/year)**
40-59 years	**10.0 % (1.5%/year)**
>59 years	**22.0 % (3.3%/year)**

Bonn Vein Study II

- risk factors for new CVI (C3-C6) -

Risk factor for CVI	Relative risk	Confidence interval	p
Gender f vs m	1.33	1.02-1.73	0.034
Age 40-59 vs <39	2.62	1.07-6.41	0.035
Age >59 vs <39	4.67	1.92-11.32	0.0007
<140/<90 vs <120/<80	2.27	1.14-4.53	0.019
<160/<100 vs<120/<80	2.10	1.00-4.37	0.049
>160/100 vs <120/<80	2.05	0.86-4.87	0.10
BMI 25-<30 vs <25	2.48	1.54-4.28	< 0.0001
BMI 30-<40 vs <25	3.12	1.65-4.94	< 0.0001
BMI ≥40 vs <25	6.10	1.01-11.93	0.001

Lee AJ, Robertson LA, Boghossian SM, Allan PL, Ruckley CV, Fowkes FGR, Evans CJ: Progression of varicose veins and chronic venous insufficiency in the general population in the Edinburgh Vein Study. J Vasc Surg: Venous and Lym Dis 2015;3:18-26

- Follow-up results of the Edinburgh Vein Study
- N=334 who had trunk VV or CVI at baseline
- 13.4 years follow-up
- Progression rate in trunk VV or CVI : 57.8% (4.3%/year)
- Trunk VV only at baseline: 31.9% progression to CVI
- Risk factors for progression:
  - family history of VV (OR 1.85, 95% CI 1.14-1.30)
  - Previous DVT (OR 4.10, 95% CI 1.07-15.71).
  - Overweight (OR 1.85, 95% CI 1.10-3.12)

Bonn Vein Study I/II

- progression of CVD -

BVS I/II (N)	C2 N (%)	C3 N (%)	C4a N (%)	C4b N (%)	C5 N (%)	C6 N (%)	Any %
C0+C1 (1269)	161 12.7	95 7.5	22 1.7	1 0.1	3 0.2	0 0	22,2
C2 (91) non saphenous		15 16.5	2 2.2	1 1.1	0 0	0 0	19.8 3.0/y
C2 (132) saphenous		28 21.2	14 10.6	0 0	0 0	0 0	31,8 4.8/y
C3 (204)			9 4.4	0 0	0 0	0 0	4,4
C4 (33)					1 3.0	1 3.0	6.1
C5 (3)						0	0,0

Bonn Vein Study II

- risk factors for progression from C2 -

Risk factor for progression of C2	Relative risk	Confidence interval	p
Gender f vs m	1.31	0.89-1.94	0.17
Age (per year)	1.02	1.01-1.04	0.0048
BMI 25-<30 vs <25	2.56	1.54-4.28	0.0003
BMI 30-<40 vs <25	2.86	1.65-4.94	0.0002
BMI ≥40 vs <25	3.47	1.01-11.93	0.048
Swelling sensation	1.68	1.01-2.81	0.047

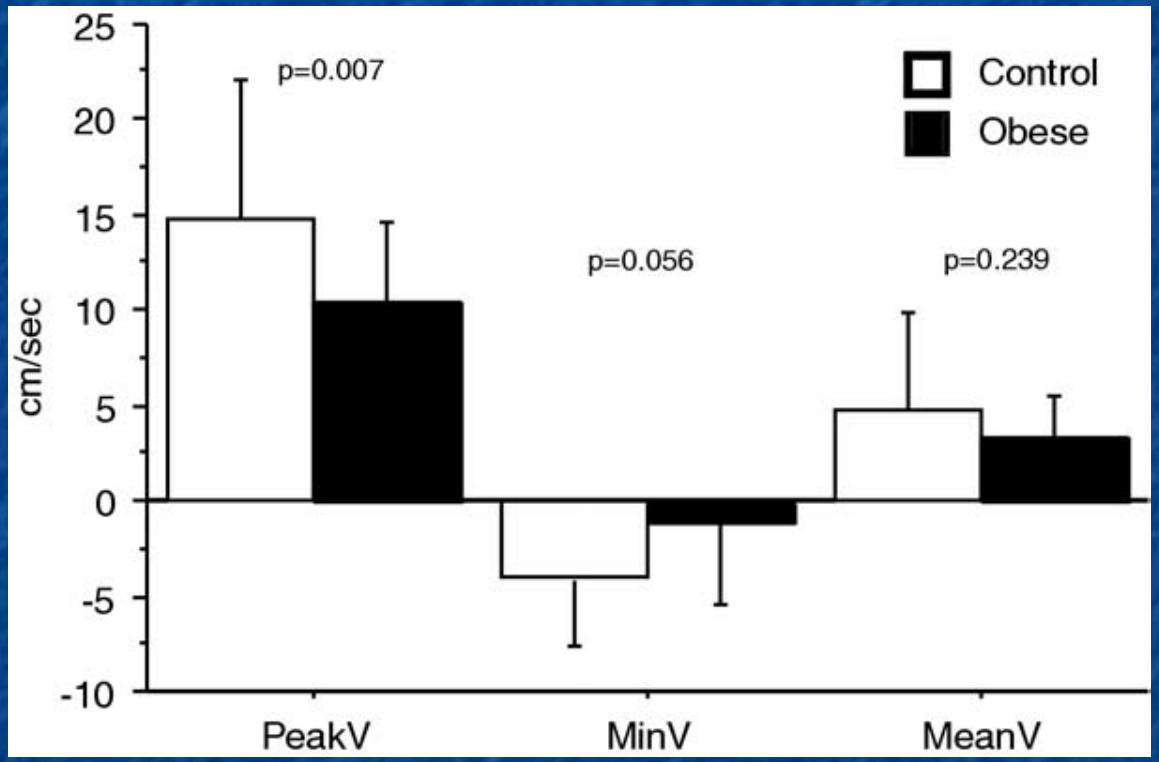
Frank Padberg, Jr: Does severe venous insufficiency have a different etiology in the morbidly obese? Is it venous? J Vasc Surg 2003;37:79-85

- Methods: Patients with severe CVI (CEAP clinical class, 4, 5, and 6), and class III obesity (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.
  - Increasing CEAP class correlated with an increased mean BMI of 47, 52, and 56, respectively ($P < .01$).
- 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

T. Willenberg et al: Impact of obesity on venous hemodynamics of the lower limbs. J Vasc Surg 2010;52:664-8

- ***Methods:*** **Venous hemodynamics were studied in a prospective cohort study in nonobese (BMI <25 kg/m2) and obese individuals (BMI >30 kg/m2). Diameter, flow volume, peak, mean, and minimum velocities were assessed.**
- ***Conclusion:*** **Lower limb venous flow parameters differ significantly between healthy obese and nonobese individuals.**
- **These findings support the mechanical role of abdominal adipose tissue potentially leading to elevated risk for both venous thromboembolism and chronic venous insufficiency.**

T. Willenberg et al: Impact of obesity on venous hemodynamics of the lower limbs. J Vasc Surg 2010;52:664-8



Mean values of peak *(PeakV)*, minimal venous (*MinV*) and mean venous (*MeanV*) blood flow velocities in the femoral vein of nonobese and obese participants. Error bars show the standard deviation.

T. Willenberg et al: The Influence of Abdominal Pressure on Lower Extremity Venous Pressure and Hemodynamics: A Human In-vivo Model Simulating the Effect of Abdominal Obesity Eur J Vasc Endovasc Surg 2011;41:849-855

- Objective: To demonstrate that abdominal pressure impacts venous flow and pres- sure characteristics.
- Results: Intravenous pressure increased with pressure application in all participants
- Conclusions: External abdominal pressure application creates venous stasis in lower limbs.
- Results of this study indicate that abdominal obesity might induce resistance to venous backflow from the lower limbs.

Obesity, sitting and CVD

- Obesity increases venous pressure independently from venous pathology
- Sitting increases this effect by compression of the iliac veins
- In addition to ambulatory venous hypertension in CVD obesity increases the venous pressure permanently during sitting

Summary

- Obesity is an independent risk factor for DVT and developement or progression of CVI
- Obesity may cause functional venous insufficiency independently from reflux
- Sitting for a long time may increase this effect
- Reducing BMI and sitting time may prevent CVI

Thank you very much for listening