# Hemodynamic monitoring in obese patients: The impact of body mass index on cardiac output and stroke volume*

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***Objective:*** **Interpreting hemodynamic parameters in critically ill obese patients can be difficult as the effects of body mass index (BMI) on cardiac output (CO) and stroke volume (SV) at the extremes of body size remains unknown. We examined the relationship between BMI and both CO and SV for patients with varying body sizes.**

***Design:*** **Retrospective cohort analysis.**

***Setting:*** **A large tertiary care academic medical center.**

***Patients:*** **A total of 700 consecutive adults who were found to have disease-free coronary arteries and a cardiac output measurement (thermodilution or Fick method) during coronary angiography between July 1, 2000, and July 31, 2004.**

***Measurements and Results:*** **We examined the relationship between BMI (mean, 28 kg/m$^2$; range, 10.6–91.6 kg/m$^2$) and cardiac hemodynamics after adjusting for demographic (age, sex) and clinical (diabetes, smoking status, valvular heart disease, medications, indications for catheterization) characteristics using multivariable regression. Body mass index was positively correlated with CO and SV. Each 1 kg/m$^2$ increase in BMI was associated with a 0.08 L/min (95% confidence interval [CI], 0.06–0.10; $p < .001$) increase in CO and 1.35 mL (95% CI, 0.96–1.74; $p < .001$) increase in SV. There was no significant association between BMI and both cardiac index (0.003 L/min/m$^2$; 95% CI, −0.008–0.014; $p = .571$) and stroke volume index (0.17 mL/m$^2$; 95% CI, −0.03–0.37; $p = .094$).**

***Conclusion:*** **Variations in BMI translate into predictable but only modest differences in CO and SV, even at the extremes of body size. Indexing hemodynamic measurements to body surface area attenuates the effects of BMI. Body habitus should not appreciably complicate the interpretation of hemodynamic measurements. (Crit Care Med 2006; 34:1243–1246)**

**KEY WORDS: hemodynamic monitoring; body mass index; obesity; cardiac output; stroke volume**

Obesity is a major public health problem. Currently, more than forty million American adults are obese (1), and each year an estimated 300,000 adults die of causes related to obesity (2). Increasing numbers of obese patients are treated in intensive care units (ICUs). Management of the critically ill obese patient may present considerable practical challenges (3, 4), including assessment of intravascular volume status and cardiac performance (5). Conventional teaching is that obesity is characterized by an increase in total blood volume and resting cardiac output (CO), resulting in dilation and hypertrophy of the left ventricle (6, 7). Body size appears to directly correlate with CO and stroke volume (SV) (8, 9). However, weight is a continuous trait, and moderate weight excess has a much higher prevalence than overt obesity. Most studies have focused on the physiologic effects of moderate obesity. Little data are available to guide the clinician in practically evaluating CO and SV over a wide range of body sizes. Accordingly, we examined the relationship between cardiac hemodynamics and body mass index (BMI). We asked two specific questions. Does BMI correlate with CO and SV over a wide range of body sizes? Should CO and SV be indexed to body surface area (BSA) when interpreting hemodynamic parameters?

## MATERIALS AND METHODS

*Patients.* We identified consecutive adults who underwent diagnostic cardiac catheterization between July 1, 2000, and July 31, 2004, at the Massachusetts General Hospital. Patients were selected if their coronary arteries were found to be free from disease (no stenoses >50%) and a cardiac output measurement was performed during the procedure.

We obtained patient information from two computerized databases containing prospectively collected data. A cardiac catheterization database for all patients undergoing coronary angiography was reviewed to obtain demographic (age, sex, race), clinical (height, weight, smoking status, diabetes, indications for catheterization), and hemodynamic (mean arterial pressure, heart rate, ejection fraction, cardiac output, stroke volume, valvular disease) data. The electronic medical record was reviewed for each patient's medications at the time of cardiac catheterization. Height and weight were determined at the time of cardiac catheterization and BMI, calculated as weight in kilograms divided by the square of height in meters. Patients with either type 1 or type 2 diabetes were categorized as diabetic. Smoking history was elicited at the time of cardiac catheterization. Hemodynamic measurements were determined by a computer algorithm and manually overread. Left ventricle (LV) and coronary angiograms were visually reviewed by the attending staff for routine clinical reporting. Left ventricular ejection fraction (LVEF) was determined by LV angiogram. Car-

*See also p. 1289.

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diac output was measured using either thermodilution or the Fick method.

*Statistical Analyses.* Body mass index was treated as both a continuous and categorical variable. Patients were classified into six categories of BMI according to the National Institutes of Health guidelines: below normal (<18.5 kg/m$^2$), normal (18.5–24.9 kg/m$^2$), overweight (25–29.9 kg/m$^2$), obese class 1 (30–34.9 kg/m$^2$), obese class 2 (35–39.9 kg/m$^2$), and obese class 3 (≥40 kg/m$^2$) (10). Common practice dictates that cardiac parameters be indexed to BSA. However, controversy surrounds the appropriateness and ideal method of indexing cardiac hemodynamics (11). In addition, different measures of body size (e.g., height, weight, BSA, BMI) are correlated, and indexing hemodynamics to a measure of body size (e.g., BSA) may introduce bias into the analysis. Therefore, we measured SV and CO and subsequently also indexed these values to BSA (cardiac index and stroke volume index [SVI]) as per convention.

The primary analysis tested associations between BMI and cardiac hemodynamics. We developed multivariable linear regression models to determine the relationship between patient BMI and cardiac hemodynamics (CO, SV, cardiac index, SVI). All analyses were performed in a similar fashion by forcing patients' BMIs into the model. Demographic (age, sex, race), clinical (smoking status, diabetes, medications, indication for catheterization), and hemodynamic factors (mean arterial blood pressure, valvular disease) were first examined using univariate analyses. Variables that were significant at $p < .20$ were included in the multivariable analyses. Variables were selected by means of backward stepwise regression and comparison of the regression sum of squares. A partial $F$-test was used to compare linear models with quadratic models. Statistical analyses were performed using Stata (version 8.2; Stata, College Station, TX) with two-tailed significance levels of .05. The institutional review board approved this study.

## RESULTS

*Patient Characteristics.* We identified 1,764 patients who underwent diagnostic cardiac catheterization and were found to have disease-free coronary arteries. Seven hundred patients had a cardiac output measurement during their angiogram and were included in our study cohort (thermodilution in 615 patients, Fick method in 39 patients, both methods in 46 patients [Spearman's correlation coefficient, 0.70]). Eight patients had missing body size data (height in five patients, weight in three patients). Body mass index could be calculated for the remaining 692 patients (99%). There were significant differences with respect to age, diabetes, mean arterial pressure, indications for catheterization, and valvular disease among BMI categories (Table 1). All pa-

**Table 1.** Patient characteristics

| Characteristics | Body Mass Index Categories | | | | | | $p$ Value for Trend |
|---|---|---|---|---|---|---|---|
| | <18.5 kg/m$^2$ (n = 16) | 18.5–24.9 kg/m$^2$ (n = 211) | 25–29.9 kg/m$^2$ (n = 250) | 30–35.9 kg/m$^2$ (n = 119) | 35–39.9 kg/m$^2$ (n = 64) | ≥40 kg/m$^2$ (n = 32) | |
| Demographic | | | | | | | |
| Age, mean (SD), yrs | 67 (12) | 66 (15) | 65 (13) | 61 (13) | 62 (11) | 59 (12) | <.001 |
| Female, n (%) | 12 (75) | 114 (54) | 109 (44) | 50 (42) | 35 (55) | 22 (69) | .925 |
| Nonwhite race, n (%) | 2 (12) | 42 (20) | 37 (15) | 15 (13) | 10 (16) | 5 (16) | .281 |
| Clinical | | | | | | | |
| Height, mean (SD), m | 1.7 (.1) | 1.7 (.1) | 1.7 (.1) | 1.7 (.1) | 1.7 (.1) | 1.6 (.1) | .035 |
| Weight, mean (SD), kg | 44 (9) | 64 (10) | 79 (11) | 93 (13) | 102 (14) | 118 (20) | <.001 |
| BMI, mean (SD), kg/m$^2$ | 16 (2) | 23 (2) | 27 (1) | 32 (1) | 37 (1) | 46 (10) | <.001 |
| BSA, mean (SD), m$^2$ | 1.5 (.2) | 1.7 (.2) | 1.9 (.2) | 2.0 (.2) | 2.1 (.2) | 2.2 (.3) | <.001 |
| Smoking history, n (%) | 10 (62) | 113 (54) | 153 (61) | 83 (70) | 35 (55) | 23 (72) | .054 |
| Diabetes, n (%) | 1 (6) | 14 (7) | 43 (17) | 30 (25) | 17 (27) | 11 (34) | <.001 |
| Medications, n (%) | | | | | | | |
| Beta blockers | 10 (62) | 103 (49) | 119 (48) | 60 (50) | 37 (58) | 12 (37) | .881 |
| Calcium channel blockers | 2 (12) | 32 (15) | 43 (17) | 15 (13) | 13 (20) | 3 (9) | .884 |
| Angiotensin pathway blockers | 5 (31) | 76 (36) | 80 (32) | 38 (32) | 19 (30) | 14 (44) | .915 |
| Vasodilators | 1 (6) | 12 (6) | 18 (7) | 14 (12) | 3 (5) | 3 (9) | .337 |
| Nitrates | 6 (37) | 25 (12) | 52 (21) | 20 (17) | 10 (16) | 6 (19) | .740 |
| Digoxin | 4 (25) | 50 (24) | 45 (18) | 24 (20) | 7 (11) | 7 (22) | .121 |
| Diuretics | 4 (25) | 81 (38) | 86 (34) | 42 (35) | 26 (41) | 15 (47) | .364 |
| Antiarrhythmics | 0 (0) | 12 (6) | 15 (6) | 3 (2) | 4 (6) | 0 (0) | .369 |
| Indications for catheterization, n (%) | | | | | | | |
| Angina or positive stress test | 8 (50) | 72 (34) | 101 (40) | 64 (54) | 34 (53) | 17 (53) | .001 |
| Congestive heart failure | 2 (12) | 39 (19) | 38 (15) | 19 (16) | 10 (16) | 7 (22) | .987 |
| Structural heart disease | 3 (19) | 73 (35) | 83 (33) | 25 (21) | 9 (14) | 4 (12) | <.001 |
| Preoperative evaluation | 3 (19) | 23 (11) | 24 (10) | 10 (8) | 8 (12) | 2 (6) | .430 |
| Other | 0 (0) | 4 (2) | 4 (2) | 1 (1) | 3 (5) | 2 (6) | .078 |
| Hemodynamic | | | | | | | |
| Heart rate, mean (SD), beats/min[a] | 74 (15) | 70 (15) | 70 (14) | 69 (16) | 72 (19) | 70 (13) | .981 |
| MAP, mean (SD), mm Hg[a] | 88 (18) | 87 (17) | 89 (19) | 93 (17) | 94 (19) | 97 (27) | <.001 |
| Ejection fraction, mean (SD), % | 53 (15) | 56 (21) | 58 (23) | 59 (23) | 59 (21) | 62 (26) | .067 |
| Valvular disease, n (%) | | | | | | | |
| Mitral stenosis | 1 (6) | 7 (3) | 8 (3) | 2 (2) | 0 (0) | 0 (0) | .050 |
| Mitral regurgitation | 1 (6) | 49 (23) | 23 (9) | 6 (5) | 5 (8) | 0 (0) | <.001 |
| Aortic stenosis | 1 (6) | 17 (8) | 32 (13) | 15 (13) | 4 (6) | 1 (3) | .734 |
| Aortic insufficiency | 0 (0) | 13 (6) | 9 (4) | 1 (1) | 1 (2) | 0 (0) | .017 |

BMI, body mass index; BSA, body surface area; MAP, mean arterial pressure.

[a]Heart rate and mean arterial pressure were measured continuously throughout cardiac catheterization and averaged over the duration of the procedure.

Table 2. Determinants of cardiac output and stroke volume[a, b]

| Predictor | Cardiac Output β coefficient (95% CI) | *p* Value | Cardiac Index β Coefficient (95% CI) | *p* Value | Stroke Volume β Coefficient (95% CI) | *p* Value | Stroke Volume Index β Coefficient (95% CI) | *p* Value |
|---|---|---|---|---|---|---|---|---|
| BMI, kg/m$^2$ | 0.08 (0.06, 0.10) | <.001 | 0.003 (−0.008, 0.014) | .571 | 1.35 (0.96, 1.74) | <.001 | 0.17 (−0.03, 0.37) | .094 |
| Age, yrs | −0.03 (−0.04, −0.02) | <.001 | −0.02 (−0.02, −0.01) | <.001 | −0.21 (−0.38, −0.04) | .018 | −0.09 (−0.18, 0.00) | .048 |
| Female sex | −0.62 (−0.88, −0.38) | <.001 | NS | NS | −11.40 (−16.07, −6.72) | <.001 | NS | NS |
| Smoking history | −0.36 (−0.61, −0.11) | .005 | −0.19 (−0.32, −0.06) | .004 | −6.07 (−10.77, −1.36) | .012 | −3.06 (−5.43, −0.69) | .012 |
| Diabetes mellitus | 0.37 (0.04, 0.71) | .030 | 0.17 (0.00, 0.36) | .053 | NS | NS | NS | NS |
| Beta blocker | −0.37 (−0.62, −0.13) | .003 | −0.25 (−0.38, −0.12) | <.001 | −4.43 (−9.01, 0.14) | .057 | −2.87 (−5.23, −0.51) | .017 |
| Vasodilators | NS | NS | NS | NS | −11.38 (−19.63, −3.14) | .007 | −5.22 (−9.44, −0.99) | .016 |
| Nitrates | −0.42 (−0.75, −0.09) | .013 | −0.20 (−0.38, −0.03) | .021 | NS | NS | NS | NS |
| Digoxin | −0.29 (−0.60, 0.01) | .061 | −0.20 (−0.36, −0.04) | .012 | NS | NS | −2.14 (−5.06, 0.77) | .149 |
| Indications for catheterization | | | | | | | | |
| Angina/positive stress test[c] | 1.00 | | 1.00 | | 1.00 | | 1.00 | |
| Congestive heart failure | −0.18 (−0.53, 0.17) | .315 | −0.08 (−0.27, 0.10) | .363 | −7.61 (−14.12, −1.09) | .022 | −3.32 (−6.69, 0.04) | .053 |
| Structural heart disease | 0.32 (0.01, 0.63) | .041 | 0.22 (0.06, 0.38) | .003 | 2.07 (−3.71, 7.85) | .482 | 1.92 (−1.04, 4.88) | .204 |
| Preoperative evaluation | 0.61 (0.17, 1.06) | .007 | 0.38 (0.15, 0.61) | .001 | 4.76 (−3.55, 13.07) | .261 | 3.45 (−0.80, 7.71) | .112 |
| Other | 0.09 (−0.76, 0.95) | .828 | 0.06 (−0.39, 0.51) | .860 | −3.89 (−19.92, 12.14) | .633 | –0.69 (−8.90, 7.53) | .870 |
| Mean arterial pressure | NS | NS | NS | NS | −0.13 (−0.25, 0.00) | .045 | −0.07 (−0.13, −0.01) | .032 |
| Mitral regurgitation | −0.58 (−0.97, −0.19) | .003 | −0.34 (−0.55, −0.14) | .001 | −10.54 (−17.77, −3.32) | .004 | −5.83 (−9.54, −2.12) | .002 |
| Aortic stenosis | −0.48 (−0.89, −0.07) | .022 | −0.28 (−0.50, −0.07) | .011 | −8.85 (−16.57, −1.13) | .025 | −4.93 (−8.88, −0.98) | .014 |

CI, confidence interval, BMI; body mass index, NS; variables not selected into the multivariable model.

[a]The variables with the best predictive power in the linear regression models are shown. Body mass index was forced into the models. Patient factors were determined with use of stepwise regression for selection of variables; [b]each 1-unit change in predictor represents a change in L/min of cardiac output, L/min/m$^2$ of cardiac index, mL of stroke volume, and mL/m$^2$ of stroke volume index; [c]patients with this factor served as the reference group.

tients had normal coronary arteries, and none had complex congenital heart disease.

*Study Questions.* First, we addressed whether BMI correlated with CO and SV over a wide range of body sizes? Each 1 kg/m$^2$ increase in BMI was associated with a 0.08 L/min (95% confidence interval [CI], 0.06–0.10; $p < .001$) increase in CO and a 1.35 mL (95% CI, 0.96–1.74; $p < .001$) increase in SV (Table 2). A similar relationship between BMI and both CO and SV was noted when patients were classified into BMI categories (Fig. 1).

Our second question was should CO and SV be indexed to BSA when interpreting hemodynamic parameters? Indexing attenuated the influence of body size on hemodynamic parameters. Each 1 kg/m$^2$ increase in BMI was not significantly associated with either the cardiac index (0.003 L/min/m$^2$; 95% CI, −0.008–0.014; $p = .571$) or the SVI (0.17 mL/m$^2$; 95% CI, −0.03–0.37; $p = .094$) (Table 2). A similar relationship between BMI and both cardiac index and SVI was noted when patients were classified into BMI categories (Fig. 1).

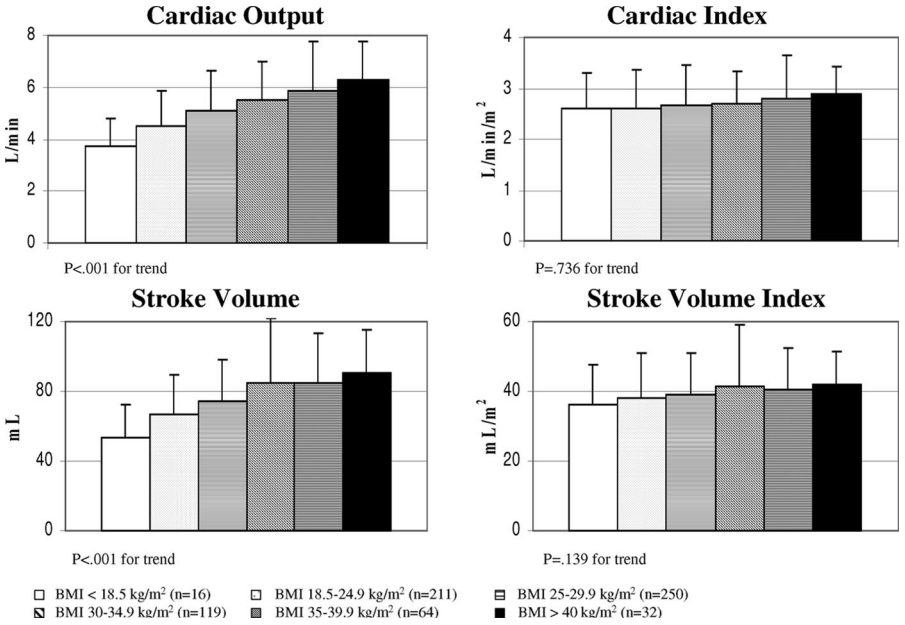


**Figure 1.** Cardiac output and stroke volume according to body mass index (*BMI*). Patients classified into six categories of BMI according to the National Institutes of Health guidelines. *Error bars* represent a single SD.

The correlation between BMI and cardiac hemodynamics was consistent in patients without valvular heart disease (CO: 0.08 L/min; 95% CI, 0.05–0.10; $p < .001$; SV: 1.40 mL; 95% CI, 0.95–1.85; $p < .001$; cardiac index: 0.002 L/min/m$^2$; 95% CI, −0.10–0.015; $p = .710$; SVI: 0.19 mL/m$^2$; 95% CI, −0.04–0.43; $p = .107$) and those not prescribed medications that influence cardiac output and stroke volume (CO: 0.07 L/min; 95% CI, 0.02–0.12; $p = .004$; SV: 1.06 mL; 95% CI, 0.40–1.72; $p = .002$; cardiac index: 0.008 L/min/m$^2$; 95% CI, −0.016–0.032; $p = .498$; SVI: 0.10 mL/m$^2$; 95% CI, −0.23–0.42; $p = .554$).

## DISCUSSION

Our study examined the relationship between BMI and cardiac hemodynamics. The results demonstrate a positive linear relationship between BMI and both CO and SV. Patients with increased BMIs had small increases in CO and SV. Indexing cardiac hemodynamics to BSA attenuated the relationship between BMI and both CO and SV.

Our study provides some of the most clinically relevant data to date on how body habitus affects cardiovascular hemodynamics. First, BMI is significantly associated with CO and SV, but the relationship is unlikely to be clinically relevant in most hemodynamic monitoring circumstances. For example, in our study, the difference in CO and SV between a normal and overweight patient was 0.6 L/min and 8 mL, respectively. Small hemodynamic differences are unlikely to complicate clinical management. Second, the observed relationship between BMI and cardiac hemodynamics extends to the extremes of body habitus. Patients with BMIs below normal had COs and SVs that were in the lower limits of normal, while very obese patients had COs and SVs that were at the upper limits of normal (12). Finally, indexing cardiac hemodynamics to BSA may allow a clinician to remove any uncertainty regarding the interpretation of CO and SV at the extremes of body size. For example, in our study the difference in cardiac index and SVI between a below normal and obese class 3 patient were 0.3 L/min/m$^2$ and 5 mL/m$^2$, respectively. Therefore, our data suggest that even in cachectic and morbidly obese patients, significant deviations in hemodynamic parameters are likely to reflect an underlying medical disorder rather than body habitus. Extremes of body size should therefore not complicate the interpretation of hemodynamics in most clinical scenarios.

The results of our study need to be interpreted within the context of its limitations. First, the study was performed in patients undergoing cardiac catheterization. Although our study patients were found to have disease-free coronaries, they were nevertheless selected for angiography, a potential marker for cardiac dysfunction. We adjusted for clinical variables in our analyses and noted that patients in the normal BMI group had hemodynamic measurements similar to normal values reported in the literature (12). In addition, a significant proportion of patients had valvular heart disease and were prescribed medications that influence cardiac output and stroke volume. However, subgroup analyses of patients without valvular heart disease and not prescribed cardiovascular medications demonstrated a consistent relationship between BMI and cardiovascular hemodynamics. Nevertheless, the relationship between BMI and cardiac hemodynamics in other clinical settings may differ. Second, our study population consisted predominantly of white patients, and the results may not be generalizable to other ethnic populations. For example, the severity of obesity as a risk factor for myocardial dysfunction in the absence of coronary artery disease is greatest in African Americans (13). Finally, only a limited number of patients in our study were identified at the extremes of body habitus. Nevertheless, our measures of cardiac hemodynamics were similarly precise across all BMI subgroups.

## CONCLUSIONS

Differences in patients' BMIs translate into statistically significant, but clinically unimportant, differences in CO and SV. Therefore, body habitus should not appreciably complicate the interpretation of hemodynamic measurements. In the event of uncertainty, indexing hemodynamic measurements to BSA will attenuate the effects of BMI.

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