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Prognostic Impact of the Indexation of Left Ventricular Mass in Patients Undergoing Dialysis

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Abstract. Left ventricular hypertrophy (LVH) is exceedingly frequent in patients undergoing dialysis. Cardiac mass is proportional to body size, but the influence of various indexing methods has not been studied in patients with end-stage renal disease. The issue is important because malnutrition and volume expansion would both tend to distort the estimate of LV mass (LVM) in these patients. In a cohort of 254 patients, the prognostic impact on all-cause mortality and cardiovascular outcomes of LVH values, calculated according to two established methods of indexing, either body surface area (BSA) or height$^{2.7}$, was assessed prospectively. When LVM was analyzed as a categorical variable, the height$^{2.7}$-based method identified a larger number of patients with LVH than the corresponding BSA-based method. One hundred and thirty-seven fatal and nonfatal cardiovascular events occurred during the follow-up period. Overall, 90 patients died, 51 of cardiovascular causes. In separate Cox models, both the LVM/height$^{2.7}$ and the LVM/BSA index independently predicted total and cardiovascular mortality ($P < 0.001$). However, the height$^{2.7}$-based method coherently produced a closer-fitting model ($P \leq 0.02$) than did the BSA-based method. The height$^{2.7}$ index was also important for the subcategorization of patients according to the presence of concentric or eccentric LVH because the prognostic value of such subcategorization was apparent only when the height$^{2.7}$-based criterion was applied. In conclusion, LVM is a strong and independent predictor of survival and cardiovascular events in patients undergoing dialysis. The indexing of LVM by height$^{2.7}$ provides more powerful prediction of mortality and cardiovascular outcomes than the BSA-based method, and the use of this index appears to be appropriate in patients undergoing dialysis.

Left ventricular hypertrophy (LVH) is now regarded as a major component of the cardiovascular burden of patients with end-stage renal disease (ESRD) (1–11). A number of studies have documented the exceedingly high frequency of this multifactorial complication in patients on renal replacement treatment. LV mass (LVM) increases progressively as renal function deteriorates with duration of dialysis treatment (4) and only in part regresses after renal transplantation or after long-term control of arterial hypertension or uremic anemia (8). In the general population, LVH, as determined by echocardiography, is a strong predictor of adverse cardiovascular events independent of conventional risk factors (12). The ominous prognosis entailed by LVH in patients with chronic renal failure who are undergoing regular dialysis treatment was consistently demonstrated in two prospective analyses (1,3), and the measurement of LVM by echocardiography is now used as a main outcome measure in intervention studies in patients undergoing dialysis (13).

Because LVM is proportional to body size, appropriate indexing is fundamental for the definition of LVH. LVH is currently identified by calculation of LVM indexed for body surface area (BSA) (12,14–16) or for BSA$^{1.5}$ (17), height$^{2.0}$ (18), or height$^{2.7}$ (17,19,20). However, controversy still exists regarding the optimal method for indexing LVM to body size in the clinical setting (21). Indexing in patients with ESRD is even more critical because weight, *i.e.*, a basic parameter for the calculation of BSA, is dependent not only on nutritional status, which is very often compromised in these patients, but also on the body fluid volume status. Yet, with the exception of a study in children (22), in virtually all studies in patients with ESRD, LVM was indexed for body surface area (1–11).

This study was designed to compare the predictive value of echocardiographically determined LVM for mortality and cardiovascular outcomes by use of two established methods of indexing for LVM in a large cohort of patients undergoing dialysis.

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Materials and Methods

Protocol

The protocol conformed to the ethical guidelines of our institutions, and informed consent was obtained from each participant. All studies were performed between 8 a.m. and 1 p.m..

Study Cohort

Two hundred and fifty-four patients with ESRD (144 men and 110 women) who had been on regular dialysis treatment (203 on hemodialysis [HD] and 51 on chronic ambulatory peritoneal dialysis) for at least 6 mo, with LV ejection fraction >35% and without a history of clinical evidence of circulatory congestion (23), were eligible for the study. The main demographic and clinical characteristics of the patients included in the study are detailed in Table 1. All participants were in sinus rhythm at the time of the study. These patients represented ~70% of the whole dialysis population of four dialysis units. The remaining 30% of patients were excluded because of the presence of circulatory congestion or major infections (20%) or because they were hospitalized for intercurrent illnesses or for logistic reasons/unwillingness to participate in the study (10%). The prevalence of diabetes mellitus in this cohort was 15% (*i.e.*, 37 patients out of 254).

Table 1. Demographic, anthropometric, biochemical, hemodynamic, and echocardiographic characteristics of the study population[a]

Characteristic	Value
Age (yr)	60.3 ± 15.4
Men/women	144/110
BMI (kg/m^2)	24.9 ± 4.3
Duration of dialysis treatment (mo)	42 (18 to 97)
Hemoglobin (g/dl)	10.6 ± 1.9
Serum albumin (g/dl)	4.01 ± 0.56
Serum C-reactive protein (mg/L)	7.4 (3.4 to 16.3)
Serum cholesterol (mg/dl)	206.5 ± 54.4
Serum phosphate (mmol/L)	1.96 ± 0.46
Serum calcium (mmol/L)	2.26 ± 0.26
Serum iPTH (pg/ml)	145 (61 to 331)
Homocysteine (μmol/L)	27.0 (19.8 to 40.2)
Kt/V	
patients undergoing hemodialysis	1.22 ± 0.27
patients undergoing CAPD	1.67 ± 0.31
Arterial pressure and heart rate	
systolic pressure (mmHg)	133.2 ± 22.5
diastolic pressure (mmHg)	75.0 ± 12.3
heart rate (beats/min)	80.6 ± 12.1
Echocardiography	
LV end diastolic diameter (cm)	5.06 ± 0.67
interventricular septum thickness (cm)	1.12 ± 0.20
posterior wall thickness (cm)	1.12 ± 0.20
ejection fraction (%)	58.1 ± 9.8
LV end diastolic volume (ml)	142.6 ± 58.6
left atrial volume (ml)	39.4 ± 18.6
LVMI (g/m^2)	137.0 ± 39.6
LVMI (g/height$^{2.7}$)	63.8 ± 19.9

[a] Data are expressed as mean ± SD or as median (interquartile range), as appropriate. *Kt/V* denotes fractional urea clearance, which was calculated according to the method of Sargent and Gotch (25) for patients undergoing hemodialysis and by a standard formula (26) for patients undergoing chronic ambulatory peritoneal dialysis. BMI, body mass index; PTH, parathyroid hormone; LVM, left ventricular mass.

All patients undergoing HD were virtually anuric (24 h urine volume <200 ml/d), whereas a minority ($n = 6$) of patients undergoing chronic ambulatory peritoneal dialysis had a 24-h diuresis >500 ml/d. Patients undergoing HD were being treated three times weekly with standard bicarbonate dialysis (Na 138 mmol/L, HCO3 35 mmol/L, K 1.5 mmol/L, Ca 1.25 mmol/L, and Mg 0.75 mmol/L) and cuprophan or semisynthetic membranes (dialysis filter surface area, 1.1 to 1.7 m2). Dry weight was established for each patient on a trial-and-error basis and was defined as the weight below which the patient suffered frequent hypotensive episodes during the latter part of the dialysis session and experienced malaise, cramps, and dizziness postdialysis. One hundred and seven patients were habitual smokers (22 ± 17 cigarettes/d). One hundred and thirty-five patients were on treatment with erythropoietin. One hundred and eleven patients were on antihypertensive treatment (78 on monotherapy with angiotensin-converting enzyme [ACE] inhibitors, AT1 antagonists, calcium channel blockers, and alpha and beta blockers, and 33 on double or triple therapy with various combinations of these drugs).

After the initial assessment, patients were followed up for 29 ± 12 mo. During the follow-up, cardiovascular events (echocardiogram-documented anginal episodes, peripheral artery disease, venous thrombosis, artery thrombosis, myocardial infarction, heart failure, echocardiogram-documented arrhythmia, transient ischemic attacks, and stroke), and death were accurately recorded. Each death was reviewed and assigned an underlying cause by a panel of five physicians. As a part of the review process, all available medical information about each death was collected. This information always included study and hospitalization records. In the case of an out-of-hospital death, family members were interviewed by telephone to better ascertain the circumstances surrounding death.

Laboratory Measurements

A fasting blood sample for serum lipids, albumin, hemoglobin, calcium and phosphate, C-reactive protein, homocysteine, and serum parathyroid hormone was obtained between 8.00 and 12.00 A.M.

Echocardiography

These studies were performed within 2 h after blood sampling during a midweek nondialysis day in patients undergoing HD and at empty abdomen in patients undergoing chronic ambulatory peritoneal dialysis. All echocardiographic measurements were carried out according to the recommendations of the American Society of Echocardiography by an observer unaware of biochemical results. LVM was calculated according to the Devereux formula and separately indexed to BSA (12) and to height$^{2.7}$ (20). The rationale for indexing to height$^{2.7}$ rather than to BSA is that the use of BSA assumes a linear relationship with LVM (*i.e.*, first power) with near-zero intercept, although it was demonstrated that linearity is approached only by the use of appropriate exponents (called "allometric signals") of measures of body size. The height$^{2.7}$ index optimizes the capacity of LVM to predict cardiovascular morbidity (20). Partition values for the definition of LVH based on BSA- and height$^{2.7}$-indexed LVM were obtained from a pooled analysis of 228 normal-weight normotensive adults, including 87 southern Italian individuals (20). These partition

values coincided with the 97.5th percentile of the distribution of either, the BSA-indexed LVM (gender-specific, men 117 g/m^2; women, 104 g/m^2) or height$^{2.7}$-indexed LVM (gender-independent, 51 g/m $^{2.7}$; gender-specific, men 50 g/m^2; women, 47 g/m^2) (20). The gender-independent BSA-based partition value (cutoff 125 g/m^2) was derived from the Framingham cohort (12). The relative wall thickness (two times the posterior wall thickness/LV end diastolic diameter) was calculated as an index of the LV geometric pattern (concentric LVH, relative wall thickness [RWT] $\geq$ 0.45; eccentric LVH, RWT $<$0.45).

Statistical Analyses

Data are reported as mean $\pm$ SD or as median and interquartile range. Variables that showed a positively skewed distribution were log transformed ($\log_{10}$). Relationships between paired parameters were analyzed by linear regression analysis.

The sensitivity, specificity, and positive and negative prediction value of each LVM index for the prediction of cardiovascular outcomes was calculated by standard methods.

The performance of LVM indexed for height$^{2.7}$ and BSA in the prediction of cardiovascular events (fatal and not) and survival was further tested with multivariate Cox proportional hazards. Variables that had an independent influence on these outcomes were identified by a backward elimination strategy starting with age, male gender, duration of regular dialysis treatment, smoking, diabetes, previous cardiovascular events, treatment modality, systolic pressure, hemoglobin, calcium times phosphate product, C-reactive protein, homocysteine, albumin, cholesterol, *Kt/V*, and parathyroid hormone. After the definition of a basic model of prognostic variables, we tested the predictive power for survival of LVM as normalized for BSA and height$^{2.7}$ (expanded models, see Table 2). To compare different models, we used the -2 log likelihood (-2 log L) statistics. This procedure compares different models fitted to the same set of survival data, and the smaller the -2 log L value, the better the agreement between the model and the observed data (24). The difference between the -2 log L of the models, which are being compared, gives a statistical estimate as to which of them provides a better fit to the data. A 3.841 difference in -2 log L coincides with a significance level of 0.05 in a χ^2 distribution with 1 df and indicates a better prediction of risk estimate provided by the method leading to the lowest -2 log L value. Hazard ratios (HR) and their 95% confidence intervals were calculated with the use of the estimated regression coefficients and their SEM in the Cox regression analysis. All calculations were made by use of a standard statistical package (SPSS for Windows Version 9.0.1, March 11, 1999, Chicago, IL).

Results

The detailed results of echocardiographic measurements as well as the main demographic, anthropometric, biochemical, and hemodynamic characteristics of the study population are shown in Table 1.

As expected, the LVM indexed for BSA and height$^{2.7}$ were highly interrelated ($r = 0.89$, $P < 0.0001$). The prevalence of LVH in patients undergoing dialysis, established in accordance to population-based specific cutoffs (see Materials and Methods section), is shown in Table 3. The height$^{2.7}$-based method (either gender-independent or -specific) identified a larger number of patients with LVH than the corresponding BSA-based method. The prevalence of concentric and eccentric hypertrophy was higher when LVH was defined by the height$^{2.7}$ method than by the BSA method (Table 3).

Assessment of Ability of LVM as a Continuous Variable (BSA- and Height$^{2.7}$-Indexed) to Predict Survival and Cardiovascular Outcomes

The ability of LVM as a continuous variable to predict death and cardiovascular outcomes was tested by Cox's model. One hundred and thirty-seven fatal and nonfatal cardiovascular events occurred during the follow-up period (Table 4). Overall, 90 patients died, 51 (57%) of cardiovascular causes. The LVM as indexed to BSA as well as the LVM as normalized to height$^{2.7}$ were much higher in patients who died during the follow-up than in those who survived ($P < 0.0001$), and in separate Cox models they significantly and independently added predictive power to the basic models of overall mortality and cardiovascular mortality (Table 2). However, the height$^{2.7}$-based method coherently produced a closer-fitting data than did the BSA-based method because the -2 log L of models based on height$^{2.7}$ were all significantly higher than those of the corresponding models based on BSA. Neither concentric hypertrophy nor eccentric hypertrophy identified by the BSA indexing contributed to explaining neither all-cause mortality ($P = 0.06$ and $P = 0.26$) nor cardiovascular outcomes ($P = 0.19$ and $P = 0.96$), whereas concentric hypertrophy by the height$^{2.7}$ indexing was an independent predictor of all-cause mortality (HR, 2.96 [1.40 to 6.26], $P = 0.004$) as well as of cardiovascular outcomes (HR, 2.20 [1.12 to 4.30], $P = 0.02$). Eccentric hypertrophy by this criterion predicted all-cause death (HR, 2.48 [1.16 to 5.33], $P = 0.02$) but failed to predict cardiovascular outcomes ($P = 0.19$).

Sensitivity, Specificity, and Positive and Negative Prediction power of LVM (BSA- and Height$^{2.7}$-Indexed) in the Prediction of Cardiovascular Outcomes

Table 5 shows the sensitivity, specificity, and positive and negative prediction power for cardiovascular events (fatal and nonfatal) of the BSA- and height$^{2.7}$-based partition values of LVM. The height$^{2.7}$-based method (either gender-independent or -specific) was consistently more sensitive than the BSA method for the identification of patients who developed cardiovascular complications, but the BSA-based method appeared to be more specific. The positive prediction value of the two methods was very close, but, again, the height$^{2.7}$-based method displayed a consistently higher negative prediction power.

Discussion

This study shows that indexing LVM by height$^{2.7}$ increases the power to predict mortality and cardiovascular outcomes in patients undergoing dialysis. Such specificity is probably due to the fact that this method is independent of body weight, a parameter that is critically influenced by malnutrition and body fluid volume status in patients undergoing dialysis.

The interest in LVM as a surrogate end point has grown exponentially during the past 20 yr, mostly because it has

Table 2. Cox proportional hazards models for overall mortality, cardiovascular death, and fatal and nonfatal cardiovascular events[a]

Model	Units of Increase	Hazard Ratio (95% CI)	*P*	
Overall mortality				
basic model				−2 log L = 858.319
significant predictors				
age	1 yr	1.05 (1.02 to 1.07)	0.00001	
gender	0 = female; 1 = male	2.46 (1.45 to 4.18)	0.0008	
cholesterol	1 mg/dl	1.01 (1.00 to 1.01)	0.002	
albumin	1 g/dl	0.41 (0.24 to 0.73)	0.002	
previous CV events	0 = no; 1 = yes	1.83 (1.15 to 2.90)	0.01	
CRP	1 mg/L	1.01 (1.00 to 1.01)	0.04	
expanded models				
LVMI (g/m^2)-based model	1 g/m^2	1.01 (1.01 to 1.02)	0.0001	842.961
LVMI ($g/ht^{2.7}$)-based model	1 $g/m^{2.7}$	1.03 (1.02 to 1.05)	0.00001	837.788 ($P < 0.02$ *versus* LVMI [g/m^2])
Cardiovascular death				
basic model				−2 log L = 461.790
significant predictors				
gender	0 = female; 1 = male	3.95 (1.88 to 8.31)	0.0003	
age	1 yr	1.05 (1.02 to 1.08)	0.0006	
cholesterol	1 mg/dl	1.01 (1.00 to 1.01)	0.004	
Kt/V		0.15 (0.04 to 0.59)	0.007	
previous CV events	0 = no; 1 = yes	2.31 (1.23 to 4.33)	0.009	
homocysteine	1 μmol/L	1.01 (1.00 to 1.02)	0.01	
calcium phosphate	1 $mmol^2/L^2$	1.40 (1.05 to 1.86)	0.02	
albumin	1 g/dl	0.45 (0.21 to 0.99)	0.049	
expanded models				
LVMI (g/m^2)-based model	1 g/m^2	1.01 (1.00 to 1.02)	0.0005	450.274
LVMI ($g/ht^{2.7}$)-based model	1 $g/m^{2.7}$	1.03 (1.02 to 1.05)	0.00001	442.835 ($P < 0.01$ *versus* LVMI [g/m^2])
Cardiovascular events (fatal and nonfatal)				
basic model				−2 log L = 947.967
significant predictors				
age	1 yr	1.04 (1.02 to 1.06)	0.0002	
duration of RDT	1 mo	1.01 (1.00 to 1.01)	0.002	
treatment modality	1 = HD; 2 = CAPD	2.79 (1.33 to 5.85)	0.007	
calcium*phosphate	1 $mmol^2/L^2$	1.31 (1.07 to 1.61)	0.01	
previous CV events	0 = no; 1 = yes	1.64 (1.06 to 2.54)	0.03	
homocysteine	1 μmol/L	1.01 (1.00 to 1.02)	0.05	
expanded models				
LVMI (g/m^2)-based model	1 g/m^2	1.00 (1.00 to 1.01)	NS	946.125
LVMI ($g/ht^{2.7}$)-based model	1 $g/m^{2.7}$	1.02 (1.01 to 1.03)	0.004	939.877 ($P < 0.02$ *versus* LVMI [g/m^2])

[a] Basic models were established starting with the risk factors detailed in the Materials and Methods section. The two indexes of LVM (body surface area [BSA]-based and height$^{2.7}$-based) were then separately introduced into the basic models. The lower −2 log L, the better the prediction of risk estimate of the corresponding model (see Materials and Methods). Factors that did not contribute significantly to the models may be deduced from the description of the multivariable strategy adopted in this study (see Statistical Analysis). ERP, C-reactive protein; CV, cardiovascular; RDT, regular dialysis treatment.

become clear that LVH is accompanied by a marked increase in cardiovascular morbidity and mortality (12,16).

The issue whether LVH is a risk factor in patients undergoing dialysis has been investigated in only two studies (1,3), which coherently demonstrated the high risk of death and cardiovascular events in patients with raised LVM. Herein we

Table 3. Prevalence of LVH by the BSA- and height$^{2.7}$-based methods[a]

	Patients with LVH *n* (%)	Patients without LVH *n* (%)
Gender-independent criteria		
LV mass/BSA	145 (57.1)	109 (42.9)
LV mass/ht$^{2.7}$	178 (70.1)	76 (29.9)
Gender-specific criteria		
LV mass/BSA	178 (70.1)	76 (29.9)
LV mass/ht$^{2.7}$	196 (77.2)	58 (22.8)
Geometric pattern of LVH		
BSA-based method		
concentric	78 (30.7)	
eccentric	67 (26.4)	
ht$^{2.7}$-based method		
concentric	95 (37.4)	
eccentric	83 (32.7)	

[a] The partition values used to define LVH are reported in the Materials and Methods section.

Table 4. Cardiovascular events (fatal and nonfatal) and causes of death in the study cohort

Outcome	*n*
Nonfatal cardiovascular events	
stroke	13
arrhythmia	20
heart failure	9
myocardial infarction	4
angina pectoris	24
peripheral artery disease	2
transient ischemic attack	9
major venous thrombosis	4
retinal artery thrombosis	1
total	86
Fatal cardiovascular events	
stroke	15
arrhythmia	5
heart failure	8
myocardial infarction	11
sudden death	6
pulmonary embolism	2
mesenteric infarction	4
total	51
Other causes of death	
sepsis/infection	13
cachexia	11
neoplasia	5
hyperkalemia	5
gastrointestinal haemorrage	2
diabetes, hyperosmolar coma	1
treatment withdrawal	1
respiratory failure	1
total	39

confirm that, however indexed, LVM adds important prognostic information to cardiovascular risk models, including Framingham risk factors like age, gender, serum cholesterol, and smoking and nontraditional risk factors like C-reactive protein and homocysteine.

To identify abnormalities of ventricular mass, the relation between heart and body size should be thoroughly considered. Although the relations between BSA, height, and weight are not linear, the division of LVM by these variables implicitly assumes linear relations with zero intercepts. It was noted that the relations of LVM to measures of body size would approximate the mathematical relations among variables with different dimensions (27,28). Thus the relation between LV mass and weight (which are both three-dimensional measurements) should be a first-power relation ($3/3 = 1$) that, between LVM (three-dimensional) and BSA (two-dimensional), should be between the first and second power and that between LV mass and height (monodimensional) near the third power (*e.g.*, height$^{2.7}$. These considerations are important because the BSA and the height$^{2.7}$ methods may identify different prevalences of LVH in diseases that affect the nutritional status and, more importantly, may underlie a different ability to predict cardiac risk (28).

The problem of appropriately indexing LVM may be of particular importance in ESRD. Malnutrition is very frequent in patients undergoing dialysis (26), and body weight is substantially reduced at all height groupings in these patients in comparison to normal individuals, and the difference is magnified at taller heights (29). Body weight loss in patients undergoing dialysis may produce an overestimate of LVM when the BSA indexing (LVM/BSA) is applied. On the other hand, body weight in patients undergoing dialysis depends to an important extent on body fluid volume status, which is notoriously expanded in most cases. Substantial fluid accumulation, up to 10% of body weight, may occur in patients undergoing dialysis without any evidence of peripheral edema, and any surfeit in body fluid volume will produce an underestimate of LVM by the LVM/BSA index. Thus, the estimate of LVM may be distorted in an unpredictable way and in opposite directions by malnutrition and extracellular volume expansion when the BSA index is used in patients undergoing dialysis.

The ability to predict the outcome is the basic requirement of any factor (risk factor) that is suspected to influence a given outcome. Thus, comparing the prognostic ability of the BSA- and height$^{2.7}$-based indexes of LVM provides a reasonable and objective way of comparing their usefulness in clinical practice. Data by De Simone (20) in patients with essential hypertension showed a better prognostic performance of the height$^{2.7}$-based method. In contrast, the prediction of the mortality risk estimated by different methods of indexing (including BSA and height$^{2.7}$ was substantially similar in two recent studies in African American (30) and in Italian (31) hypertensives, which suggests that LVH carries a high risk indepen-

Table 5. Sensitivity, specificity, positive and negative prediction power for cardiovascular events based on BSA or height$^{2.7}$ indexing[a]

Criteria	Sensitivity (%)	Specificity (%)	Positive Prediction Value (%)	Negative Prediction Value (%)
Gender specific				
LVM/BSA	84.0	38.6	45.5	80.2
LVM/height$^{2.7}$	91.7	31.6	44.9	86.2
Gender-independent				
LVM/BSA	69.8	50.6	46.2	73.4
LVM/height$^{2.7}$	84.0	38.6	45.5	80.2

[a] The partition values used to define LVH are reported in the Materials and Methods section.

dently of the method of indexing. Patients with ESRD are a high-risk population, and, in our study cohort, during an average follow up of ~30 mo, we registered 137 cardiovascular events. Our data in patients with ESRD confirmed that LVM, however indexed, has a strong and independent power to predict all-cause mortality and cardiovascular complications. Yet the height$^{2.7}$-based method yielded a better (and statistically more significant) model of all-cause mortality, cardiovascular mortality, and cardiovascular events. Accordingly, the height$^{2.7}$ index, although not superior to the BSA index for the identification of patients who will develop cardiovascular events, proved to be a stronger criterion for the identification of event-free patients (Table 5). It is important to note that the height$^{2.7}$ index is also important for the subcategorization of patients according to concentric and eccentric LVH, because we found that the prognostic value of this subcategorization became apparent only when the height$^{2.7}$-based criterion was applied. In theory, both malnutrition and fluid retention may both contribute to distort the prognostic power of the BSA-based method. However, the distortion attributable to malnutrition (*i.e.,* an overestimation of LVM) is unlikely to be a major factor in explaining the relatively lower prognostic performance of the BSA-based method, because this method yielded a lower prevalence of LVH than the height$^{2.7}$-based method and the difference was similar to that reported in a recent large survey among hypertensive patients (32). On the other hand, fluid volume expansion in patients undergoing dialysis would (mathematically) produce an underestimation of the "true" LVM according to the BSA method. The situation is further confounded by the fact that sodium and water retention is a well-established (biologic) trigger of LV hypertrophy. Thus, weight excess attributable to fluid retention (biologically) triggers LV hypertrophy but (numerically), by increasing the denominator of the BSA-based index, it also tends to decrease the estimate of LVM, thereby reducing the prognostic strength of the LVM/BSA index.

In summary, it seems likely that the apparent superiority of the height$^{2.7}$-based method is mainly due to the fact that this index renders the estimate of LVM independent of volume expansion. To properly address this issue, detailed information on body composition and extracellular volume quantification is required. Bioelectric impedance, a technique that is increasingly used in the dialysis population, appears to be a promising method (33) that can be applied to better understand and to refine the prognostic value of LVM in patients undergoing dialysis.

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