SHOE SIZE, STATURE AND CAESAREAN DELIVERY

To the Editor: Van Bogaert's analyses of shoe size,[1] foot length[2] and stature[1,2] as indicators of pelvic (in)adequacy, assume that their statistical relationships with caesarean delivery reflect their allometric relationships with pelvic size. Yet caesarean deliveries are the result of decisions taken by clinician(s) responsible for assessing the risk (to mother and fetus) of alternative delivery procedures, i.e. non-operative and/or unassisted delivery. Anthropometric measurements (of height, weight or shoe size) comprise only one component of the information on which such decisions are based. These measurements therefore have to compete with a variety of additional maternal, fetal and social factors, many of which influence the need (or desire) for operative delivery. For example, older and primiparous mothers as well as those experiencing gestational diabetes, pregnancy-induced hypertension (PIH) or pre-eclampsia, have an increased risk of caesarean delivery, as do mothers who have had one or more previous caesarean deliveries. Likewise, macrosomia (birth weight ≥ 4 000 g), prematurity and (mal)presentation all increase the risk of caesarean delivery, while the social identity (class and ethnicity), negotiating skills and personal preferences of both mothers and clinicians are likely to influence their decision to undergo/undertake an operative delivery.[3] To establish whether small shoe size or shorter stature might increase the risk of caesarean delivery it is therefore necessary to control for these potential (maternal, fetal and social) confounders.

Using data abstracted from the obstetric notes of Birth-to-Ten mothers who delivered at Chris Hani Baragwanath Hospital in Soweto[4,5] it was possible to assess whether small shoe size or shorter stature were associated with an increased risk of caesarean delivery both before and after controlling for a number of alternative risk factors (maternal age, parity, body mass index (BMI), previous caesarean delivery, PIH, proteinuria, glycosuria, prematurity, macrosomia and fetal sex). Obstetric notes were available for 539 of the 2 120 Birth-to-Ten mothers who delivered at Baragwanath Hospital.[5] Maternal shoe size had been recorded in the obstetric records of 280 mothers, with height measurements available for 214 of the 280. These anthropometric measurements were highly correlated (Fig. 1) and, among this sample of mothers, height explained more than three times as much variance in shoe size

Table I. Sociodemographic, anthropometric, obstetric and neonatal risk factors for caesarean delivery

	Model 1 — bivariate		Model 2 — multivariate		Model 3 — multivariate		Model 4 — multivariate	
Risk factor (referent)	*N*	OR (95% CI)	*N*	OR (95% CI)	*N*	OR (95% CI)	*N*	OR (95% CI)
Sociodemographic								
Maternal age (20 - 29 y)	280							
< 20 y		0.94 (0.47, 1.86)		-		-		-
30 - 35 y		1.03 (0.56, 1.88)		-		-		-
≥ 36 y		1.12 (0.48, 2.57)		-		-		-
Parity (not primiparous)	280	1.09 (0.67, 1.77)		-		-		-
Anthropometric								
Body mass index (26 - 29 kg/m²)	214		205		205		205	
< 26 kg/m²		1.63 (0.65, 4.08)		1.55 (0.60, 4.00)		1.50 (0.59, 3.85)		1.51 (0.58, 3.90)
> 29 kg/m²		3.16 (1.50, 6.66)†		2.79 (1.27, 6.11)†		3.00 (1.37, 6.61)†		2.98 (1.35, 6.61)†
Height (> 1.60 m)	214		205				205	
1.51 - 1.60 m		1.35 (0.61, 2.98)		1.62 (0.67, 3.92)		-		1.48 (0.60, 3.62)
≤ 1.50 m		1.57 (0.76, 3.22)		1.96 (0.88, 4.36)		-		1.62 (0.68, 3.85)
Shoe size (≥ size 7)	280	1.34 (0.76, 2.34)		-	205	1.89 (0.89, 4.00)	205	1.60 (0.70, 3.62)
Obstetric								
Previous caesarean (none)	269	2.00 (1.07, 3.74)*	205	2.71 (1.11, 6.61)*	205	2.55 (1.06, 6.10)*	205	2.72 (1.11, 6.67)*
PIH (absent)	253	1.65, (0.91, 3.00)		-		-		-
Proteinuria (absent)	270	0.54 (0.20, 1.44)		-		-		-
Glycosuria (absent)	269	0.43 (0.09, 2.17)		-		-		-
Neonatal								
Prematurity (≥ 36 weeks)	276	0.48 (0.20, 1.13)		-		-		-
Macrosomia (< 4 000 g)	280	8.50 (1.87, 38.8)†	205	4.08 (0.82, 20.34)	205	4.67 (0.93, 23.39)	205	4.36 (0.86, 22.07)
Male fetal sex (female)	280	0.71 (0.44, 1.14)		-		-		-

*$P < 0.05$
†$P < 0.01$
OR = odds ratio; PIH = pregnancy-induced hypertension.

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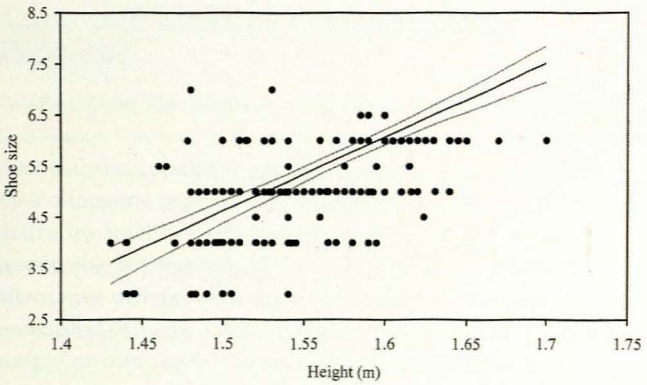

Fig. 1. Scatter plot of maternal shoe size against maternal height. The solid line represents the relationship predicted by the regression: y = 14.0 (SEE 1.1) · x –16.3 (SEE 1.8); F = 151.42, df = 1 and 213, P < 0.001; adjusted r^2 = 0.41. The dotted lines represent the 95% confidence intervals of the regression line.

(r^2 = 0.41) as that observed in Van Bogaert's study (r^2 = 0.12).[1] Nevertheless, there was only a modest tendency towards an increased risk of caesarean delivery for mothers with smaller shoe sizes and those with shorter statures (model 1, Table I) — even selecting those cut-offs for shoe size (below size 7) and stature (below 1.60 m) that displayed the strongest bivariate associations with caesarean delivery (analyses not shown). The statistical strength of these associations did increase after controlling for maternal and neonatal factors that were significantly associated with an increased risk of caesarean delivery (BMI, previous caesarean delivery and macrosomia; models 2 and 3, Table I), but none achieved statistical significance (models 2, 3 and 4, Table I). Instead, caesarean deliveries were up to three times more common among mothers who were obese (> 29 kg/m^2) before delivery, while mothers who had already had at least one previous caesarean delivery were more than twice as likely to have another.

These findings suggest that neither shoe size nor stature were important (or useful) risk factors for caesarean delivery in this population. Either they had a limited predictive value for pelvic (in)adequacy, or they were perceived as irrelevant by those deciding whether to perform a caesarean delivery. Given that only half of the obstetric records examined in the present study contained records of maternal shoe size, and fewer of these also contained maternal height measurements, it is likely that the clinicians responsible for collecting this information share the view of the World Health Organistation (WHO) collaborative panel, which recently concluded that stature did not meet the screening criteria for assisted delivery.[6]

George T H Ellison

Social Science Research Unit
Institute of Education
University of London
London

Thea de Wet

Department of Anthropology and Development Studies
Rand Afrikaans University

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PLANT STEROL/STEROLIN SUPPLEMENT USE IN A COHORT OF SOUTH AFRICAN HIV-INFECTED PATIENTS — EFFECTS ON IMMUNOLOGICAL AND VIROLOGICAL SURROGATE MARKERS

To the Editor: It has been demonstrated that micronutrient supplementation may be an important prophylactic and therapeutic measure for HIV-1-infected patients, and is possibly one of the few potential interventions for low-income countries.[1] In sub-Saharan countries facing the bulk of new infections worldwide, the use of highly active antiretroviral therapy (HAART) is out of reach of most patients because of the cost in the private sector and the lack of provision of any therapies by the health departments of these countries. In recent years many groups have investigated the outcomes of this infection in patients supplemented with vitamin B_2 or multivitamin supplementation including/excluding vitamin A during pregnancy.[3] Some studies have shown that high doses of vitamin B_6 supplements were associated with improved survival of patients, while zinc supplementation was associated with poorer survival.[4]

A supplement containing a mixture of plant sterols and sterolins has been developed and investigated by our group in the treatment of many diseases. This mixture has been shown to have immune modulating activities — the addition of this mixture to T-cells *in vitro* leads to the enhanced secretion of interleukin 2 (IL2) and gamma interferon (INF-γ)[5,6] and further tests revealed that the mixture preferentially targets CD4 cells of T_{H1} phenotype and leaves T_{H2} CD4 cells unaffected.[7] This mixture was tested in a double-blind, placebo-controlled manner as an adjuvant in patients with pulmonary tuberculosis and it was shown to have a positive effect on disease recovery in such patients.[8] The mixture was also tested in a double-blind, placebo-controlled trial in healthy marathon runners where it prevented exercise-induced inflammation and post-event transient immune suppression.[9] Furthermore, the beneficial effects of this mixture in an animal model of