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CLINICAL ARTICLE

A comparative study of the non-pneumatic anti-shock garment for the treatment of obstetric hemorrhage in Egypt

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ABSTRACT

Objective: To assess the impact of the non-pneumatic anti-shock garment (NASG) on maternal outcome following severe obstetric hemorrhage. **Methods:** A non-randomized pre-intervention/intervention study was conducted in 2 tertiary hospitals in Egypt from June 2006 to May 2008. Women with obstetric hemorrhage (estimated blood loss ≥ 1000 mL and/or ≥ 1 sign of shock [systolic blood pressure < 100 mm Hg or pulse > 100 beats per minute]) were treated with either a standardized protocol (pre-intervention) or a standardized protocol plus the NASG (intervention). The primary outcome was extreme adverse outcome (EAO), combining maternal mortality and severe morbidity (cardiac, respiratory, renal, or cerebral dysfunction). Secondary outcomes were measured blood loss, urine output, emergency hysterectomy, and (individually) mortality or morbidity. Analyses were performed to examine independent association of the NASG with EAO. **Results:** Mean measured blood loss decreased from 379 mL pre-intervention to 253 mL in the intervention group ($P < 0.01$). In a multiple logistic regression model, the NASG was associated with reduced odds of EAO (odds ratio 0.38; 95% confidence interval, 0.17–0.85). **Conclusion:** The NASG, in addition to standardized protocols at tertiary facilities for obstetric hemorrhage and shock, resulted in lower measured blood loss and reduced EAO.

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1. Introduction

Obstetric hemorrhage is a major cause of maternal morbidity and mortality. Severe obstetric hemorrhage leads to hypovolemic shock, which—if it is not reversed with intravenous fluids, blood transfusions, and control of the source of hemorrhage—can lead to tissue hypoxia and organ death. Definitive management of hypovolemic shock is often delayed in low-resource settings [1].

The non-pneumatic anti-shock garment (NASG; Zoex Corporation, Ashland, OR, USA) is used to reverse shock and control hemorrhage during delays in either transport from home to hospital or obtaining appropriate care at the facility level [2]. The NASG is a lower-body pressure device made from neoprene and hook-and-loop-fastening tape (Velcro, Manchester, NH, USA) that is placed on the legs, pelvis, and abdomen [3] (Fig. 1). Its mechanisms of action include decreasing the transmural pressure and radius of blood vessels in the lower body, decreasing blood flow to and within the area compressed, and reversing shock by redirecting blood to the core organs [4–10].

In smaller pre-intervention/intervention studies, the NASG was associated with significantly reduced measured blood loss and more rapid recovery times [3–7,9]. The aim of the present study was to analyze the impact of the NASG on extreme adverse outcome (EAO)—a combination of mortality and severe end-organ morbidity—in a larger study in Egypt.

2. Materials and methods

A non-randomized pre-intervention/intervention study was conducted at 2 tertiary-care facilities in Egypt: El Galaa Maternity Teaching Hospital, Cairo; and Assiut University Women's Health Center, Assiut. These comprehensive emergency obstetric care (CEmOC) facilities receive referrals from lower-level facilities and following home delivery, and are staffed by consulting obstetricians and residents. The pre-intervention phase was from June 2006 to May 2007, and the NASG intervention phase was from June 2007 to May 2008.

The participants were pregnant (with a non-viable fetus), birthing, or postpartum women experiencing hypovolemic shock secondary to obstetric hemorrhage from any etiology (Table 1). Additional inclusion criteria were estimated blood loss of at least 1000 mL and/or

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Fig. 1. A non-pneumatic anti-shock garment fully applied.

at least 1 clinical sign of hypovolemic shock (systolic blood pressure [SBP] <100 mm Hg or pulse >100 beats per minute [BPM]). Women were eligible regardless of whether they began hemorrhaging outside the facility and were transferred in, or if they began hemorrhaging in the facility. Estimated revealed blood loss was used—together with urine output, vital signs, and level of consciousness—as further indication of the severity of status on study admission. If blood loss was concealed (e.g. ectopic) but women were in shock, the providers were trained to use a chart, adapted from Martel [11], to classify shock and determine the amount of concealed blood loss.

The study protocol was approved by the University of California, San Francisco (UCSF) Committee on Human Research (approval number H6899-23524) and the Institutional Review Boards of El Galaa Maternity Teaching Hospital and Assiut University Women's Health Center. In the pre-intervention phase, women gave verbal informed consent for their data to be used; all women in the intervention phase provided written informed consent for use of the NASG and their data. A US Federal waiver of consent/authorization for minimal-risk research (45 CFR 46, 45 CFR 164.512) was obtained so that women who were unconscious or confused at study entry were enrolled and treated; however, their data were used only after they subsequently provided informed consent or if a relative gave consent on their behalf.

During the pre-intervention phase, women were treated with a standardized evidence-based protocol for hemorrhage and shock. The intervention phase used the same protocol, in addition to the NASG. In both phases, the women's vital signs and blood loss were monitored while the hemorrhage etiology was identified and resuscitation/treatment initiated. Blood loss after enrollment in both phases was measured using a closed-end calibrated drape (Excellent Fixable Drapes, Madurai, India), which was placed immediately upon study entry. In the intervention phase, the NASG was applied at the same time as the drape. The protocol used in both phases included: administration of crystalloid intravenous fluids (≥ 1500 mL in the first hour following study admission); administration of uterine massage and uterotonic medications for uterine atony (intravenous or intramuscular oxytocin, intramuscular ergometrine, and rectal misoprostol); vaginal procedures; provision of blood transfusions (standard in the 2 study sites for women who lost ≥ 1000 mL of blood); and surgery. The NASG was left in place during vaginal procedures. If laparotomy was required, the abdominal and pelvic segments were opened immediately before the incision was made, and closed as soon as the surgery was completed.

Women remained in the study and—in the intervention phase—in the NASG until their vital signs had stabilized (SBP >100 mm Hg and pulse <100 BPM) for at least 2 hours and blood loss had decreased to approximately 25–50 mL per hour. Each facility had 6 NASGs, and each NASG used in the study was decontaminated, laundered, hung to dry, and stored between uses.

Indicators of severity of condition at study entry were mean arterial pressure (MAP: $[2 \times \text{diastolic blood pressure} + \text{SBP}] / 3$) lower than 60 mm Hg; hemoglobin level (measured with a HemoCue

photometer [HemoCue, Anglehölm, Sweden]); vital signs; and level of consciousness (unconscious, confused/agitated, or normal).

The primary outcome was EAO prior to discharge from hospital. The definition by Mantel et al. [12] for severe maternal morbidities was used, with the following organ system dysfunctions related to severe obstetric hemorrhage: heart failure (impairment of cardiac function according to New York Heart Disease Classification [13]); acute respiratory distress syndrome (impairment of respiratory function requiring oxygen supplementation, ventilation, or limiting physical activity compared with pre-pregnancy status); renal failure (oliguria; <120-mL output in 4-hour intervals, serum creatinine >1.5 mg/dL or increased >1.0 mg/dL above baseline); and cerebral impairment (seizures, unconsciousness, or motor/cognitive loss) lasting more than 24 hours after resuscitation. The secondary outcomes were urine output, cumulative blood loss (measured hourly after study admission), rates of emergency hysterectomy for uterine atony, and maternal mortality or severe maternal morbidity (as individual indicators).

Hospital staff had been trained in the standardized protocol for management of obstetric hemorrhage and shock, blood collection and measurement, and completion of data collection forms. Following the pre-intervention phase, clinician data collectors were trained to use the NASG (including a video of placement and removal; instructions on placement, removal, and use during vaginal procedures and surgery;

Table 1
Demographics, diagnoses, and condition on study entry.^a

	Pre-intervention (n = 432)	Intervention (n = 558)	P value
<i>Characteristic</i>			
Age, y	28.7 \pm 6.2	28.9 \pm 6.0	0.65
Parity	2.4 \pm 2.1	2.6 \pm 2.0	0.18
Duration of pregnancy, wk ^b	37.1 \pm 4.0	37.7 \pm 3.7	0.06
Where bleeding began			<0.01
Outside hospital	298 (69.0)	307 (55.0)	
In hospital	134 (31.0)	251 (45.0)	
<i>Primary definitive diagnosis^c</i>			
Uterine atony	147 (34.0)	246 (44.1)	<0.01
Ectopic pregnancy	80 (18.5)	71 (12.7)	<0.01
Complications of abortion	38 (8.8)	77 (13.8)	0.02
Abruption of placenta	59 (13.7)	52 (9.3)	0.03
Vaginal/cervical lacerations	22 (5.1)	43 (7.7)	0.10
Retained placenta or tissue	18 (4.2)	20 (3.6)	0.64
Ruptured uterus	30 (6.9)	18 (3.2)	<0.01
Placenta previa	23 (5.3)	8 (1.4)	<0.01 ^d
Placenta accreta	5 (1.2)	7 (1.3)	1.00 ^d
Molar pregnancy	7 (1.6)	9 (1.6)	0.99 ^d
<i>Condition on study entry</i>			
MAP <60 mm Hg ^e	72 (16.7)	106 (19.0)	0.35
MAP \geq 60 mm Hg ^e	359 (83.3)	452 (81.0)	
Level of consciousness ^f			0.58
Unconscious	12 (2.8)	19 (3.4)	
Other	414 (97.2)	533 (96.6)	
Measured hemoglobin at study entry, g/dL ^g	6.8 \pm 1.7	6.8 \pm 1.2	0.67
Mean systolic blood pressure, mm Hg	93.8 \pm 15.8	90.9 \pm 12.3	<0.01
Mean pulse, BPM	119.6 \pm 7.4	120.5 \pm 8.4	0.08

Abbreviations: BPM, beats per minute; MAP, mean arterial pressure.

^a Values are given as mean \pm SD or number (percentage) unless otherwise indicated.

^b Excludes cases with diagnoses of ectopic or molar pregnancies, or complications of abortion.

^c Data not shown for 3 cases in the pre-intervention phase and 5 cases in the intervention phase in which there was a primary definitive diagnosis of "other," and 2 cases in the intervention phase in which there was a diagnosis of inverted uterus.

^d Two-sided Fisher exact test used because of small cell sizes.

^e Includes women for whom there was non-palpable blood pressure. Data missing for 1 woman in the pre-intervention phase.

^f Data missing for 12 women (6 in each phase).

^g Data missing for 30 women. Because of skewed distributions, the natural logarithm of mean hemoglobin was used in a *t* test; because the results of this test were similar to those using the original values, results with original values are shown.

and hands-on practice of placement and removal [10]), and the standardized protocol was reviewed. All data were collected by the clinicians during or immediately after treatment of the patient in shock. All data forms were reviewed by the Egyptian principal investigators, who then sent the forms electronically—via a data fax system (Clinical DataFax Systems, Ontario, Canada)—to the data team at UCSF.

Demographic characteristics, condition on study entry, and treatment received during the 2 phases were compared. Two-sided *t* tests of the differences in the means of continuous variables (assuming unequal variances in the 2 populations) and χ^2 tests (or Fisher exact tests, as required) of the independence of dichotomous variables by study phase were used. Relative risk (RR) and 95% confidence interval (CI) were determined for the primary outcome and (individually) for the following secondary outcomes: urine output, emergency hysterectomy (in cases of uterine atony), and mortality and severe morbidity. Mean cumulative measured volume of blood loss in the drape was compared between the phases via *t* test. Multiple logistic regression was used to estimate the independent effect of the NASG on EAO, controlling for other characteristics predictive of this outcome. The independent variables included in the model—in addition to study phase—were selected on the basis of their significant association with EAO in bivariate analyses (*t* tests and logistic regression): severity of shock (MAP <60 mm Hg); level of hemoglobin at study entry; whether the woman began bleeding outside the CEMOC facility; and primary definitive diagnosis of uterine atony versus another diagnosis. In addition, the facility was included as a control variable to hold constant the effect (on EAO) of any unmeasured systematic differences in the characteristics of the 2 clinical populations or in the quality of care provided in the 2 settings. The confidence level for all tests was set to 95%. Data were analyzed using STATA 10 (StataCorp, College Station, TX, USA). *P*<0.05 was considered to be statistically significant.

3. Results

At El Galaa Maternity Teaching Hospital, there were 20 132 deliveries in the pre-intervention period and 19 073 during the intervention period; at Assiut University Women's Health Center, there were 11 858 deliveries during the pre-intervention period and 12 103 in the intervention period.

There were 432 women in the pre-intervention phase and 558 in the intervention phase (women in this phase were in the NASG for an average of 4 hours 29 minutes). Participants in the 2 phases were similar in terms of age, parity, and duration of pregnancy (Table 1). Primary definitive diagnoses varied between the phases; significantly fewer women in the pre-intervention phase than in the intervention phase had uterine atony (34.0% vs 44.1%; *P*<0.01), although there was a significantly higher incidence of ectopic pregnancy, complications of abortion, placental abruption, ruptured uterus, and placenta previa in

the former phase. All women had estimated blood loss of at least 1000 mL (data not shown). A significantly larger proportion of women in the pre-intervention phase than in the intervention phase were transferred to hospital after bleeding had started (69.0% vs 55.0%; *P*<0.01). The majority of women in both phases were in mild–moderate shock. There were no significant differences at study entry in the proportion of women with MAP lower than 60 mm Hg, the level of consciousness, the mean hemoglobin level, or the mean pulse.

Most women with uterine atony received prophylactic uterotonics, although a higher proportion received them in the intervention phase (86.6% vs 78.7%; *P*=0.03) (Table 2). Similarly, more women with atony received uterotonics in the first hour after study admission in the intervention phase (99.3% vs 95.9%; *P*=0.03), whereas significantly fewer women in this phase received at least 1500 mL of crystalloid intravenous fluids in the first hour after study admission (67.7% vs 95.1%; *P*<0.01). Nearly all women in both phases received a blood transfusion; significantly fewer women in the intervention phase received blood in the first hour after study admission (83.0% vs 96.8%; *P*<0.01).

The distribution of outcome by study phase and bivariate tests of association of outcome by phase are shown in Table 3 and Fig. 2. The primary outcome, EAO, was significantly lower in the intervention phase than in the pre-intervention phase: 11 (2.0%) versus 27 (6.3%) (RR 0.32; 95% CI, 0.16–0.63). Secondary outcomes were also lower in the intervention phase; morbidity (primarily renal failure) decreased from 4.0% to 0.9% (RR 0.22; 95% CI, 0.08–0.60). The reduction in maternal mortality, by itself, was not significant. In the intervention phase, there was a reduction in the proportion of women with urine output below 30 mL in the first hour; the decrease in the proportion with urine output less than 60 mL in the second hour was statistically significant (RR 0.55; 95% CI, 0.31–0.99). Compared with the pre-intervention phase, there was a reduction in the intervention phase among women with uterine atony in terms of emergency hysterectomy performed for intractable hemorrhage: 11.8% versus 4.8% (RR 0.41; 95% CI, 0.21–0.80). The total mean measured blood loss was significantly lower in the intervention phase than in the pre-intervention phase (253.2 mL vs 378.9 mL; *P*<0.01). At hourly intervals, mean blood loss was consistently lower among the women treated with the NASG than among those treated during the pre-intervention phase only (Fig. 2).

Two measures of the severity of condition upon study admission were strongly associated with subsequent EAO, after controlling for the other variables in the model (Table 4). Women with MAP lower than 60 mm Hg had almost 5 times the odds of EAO (odds ratio [OR] 4.58; 95% CI, 1.91–11.02). Following bivariate analysis, lower mean hemoglobin level at study admission was associated with EAO (data not shown), and this association remained significant in the multivariate model; each unit increase in hemoglobin level reduced

Table 2
Treatments for shock and hemorrhage administered during the study phases.^a

Treatment	Pre-intervention (n = 432)	Intervention (n = 558)	<i>P</i> value
Prophylactic uterotonics ^b	133 (78.7)	233 (86.6)	0.03
Any uterotonics ^b	165 (97.6)	268 (99.6)	0.08 ^c
Uterotonics within first hour ^b	162 (95.9)	267 (99.3)	0.03 ^c
Mean number of doses of uterotonics received ^b	3.4 ± 1.2	2.5 ± 1.2	<0.01
≥1500 mL intravenous fluids within first hour	411 (95.1)	378 (67.7)	<0.01
≥1500 mL intravenous fluids within 90 minutes	422 (97.7)	497 (89.1)	<0.01
≥1500 mL intravenous fluids within 2 hours	424 (98.1)	510 (91.4)	<0.01
Blood transfusion within first hour	418 (96.8)	463 (83.0)	<0.01
Blood transfusion any time after study admission	430 (99.5)	550 (98.6)	0.20 ^c
Mean volume of blood products transfused, mL ^d	1066.3 ± 487.7	737.1 ± 405.9	<0.01

^a Values are given as number (percentage) or mean ± SD unless otherwise indicated.

^b Data are for 438 cases with a primary or a secondary diagnosis of uterine atony: 169 in the pre-intervention phase and 269 in the intervention phase.

^c Two-sided Fisher exact test used.

^d Whole blood or packed red blood cells. Data are from 980 cases.

Table 3
Outcomes following standard management for hemorrhage and shock (pre-intervention) and standard management plus NASG (intervention).^a

Outcome	Pre-intervention (n = 432)	Intervention (n = 558)	Relative risk	95% confidence interval
Measured vaginal blood loss in drape, mL ^b	378.9 ± 234.3	253.2 ± 205.3	<i>t</i> = 7.79	<i>P</i> < 0.01
Urine output				
<30 mL in first hour ^c	15 (5.5)	10 (2.8)	0.51	0.23–1.12
<60 mL in second hour ^c	25 (5.8)	18 (3.2)	0.55	0.31–0.99
Emergency hysterectomy (for uterine atony cases) ^d	20 (11.8)	13 (4.8)	0.41	0.21–0.80
Extreme adverse outcomes	27 (6.3)	11 (2.0)	0.32	0.16–0.63
Mortality	10 (2.3)	6 (1.1)	0.46	0.17–1.27
Morbidity ^e	17 (4.0)	5 (0.9)	0.22	0.08–0.60

Abbreviation: NASG, non-pneumatic anti-shock garment.

^a Values are given as mean ± SD or number (percentage) unless otherwise indicated.

^b For cases in which the calibrated blood collection drape was used and in which there were data for blood loss (n = 771).

^c Data are for 624 cases with measured urine output in the first hour (271 in the pre-intervention phase and 353 in the intervention phase) and 986 cases with measured urine output in the second hour (428 in the pre-intervention phase and 558 in the intervention phase).

^d Data are for 438 cases with a primary or a secondary diagnosis of uterine atony: 169 in the pre-intervention phase and 269 in the intervention phase.

^e Includes heart failure, acute respiratory distress syndrome, renal failure, and cerebral impairment (seizures, unconsciousness, and motor/cognitive loss) lasting more than 24 hours after resuscitation from shock.

the odds of EAO by 53% (OR 0.47; 95% CI, 0.33–0.66). In addition, women with uterine atony had more than 3 times the odds of EAO than did women with other primary diagnoses (OR 3.40; 95% CI, 1.47–7.85). Neither the facility nor where the bleeding began (within or outside the hospital) was significantly associated with EAO in the adjusted model. However, the NASG remained significantly associated with reduced odds of EAO (OR 0.38; 95% CI, 0.17–0.85).

4. Discussion

In the intervention phase, women who entered the study already suffering from hypovolemic shock secondary to obstetric hemorrhage had significantly reduced EAO rates, total mean measured blood loss, hourly mean measured blood loss, decreased urine output 2 hours after study entry, emergency hysterectomy for intractable hemorrhage from uterine atony, and severe morbidity compared with women in the pre-intervention phase; they also had reduced maternal mortality, although this was not statistically significant. The findings of the multiple logistic regression analysis—controlling for the effects of the severity of conditions on study admission, diagnostic categories, facility differences, and where bleeding began—demonstrated that the NASG reduced the odds of EAO by 62%.

In the present study, the evidence regarding the benefits of the NASG was stronger than in the earlier Egypt NASG pilot study [6]. In both studies, the measured blood loss was significantly lower for women treated with the NASG, and in the current study EAO was significantly reduced—with the exception of mortality as an individual outcome. The findings were also similar to those from a smaller study

in Nigeria [3] among women with more severe shock. The present study had the advantages of equivalent periods for the pre-intervention and the intervention phases, larger sample size, more power to demonstrate statistically significant differences in EAO, use of emergency hysterectomy and urine output as further indicators of positive effects of the NASG, and use of logistic regression to hold constant the effect of potential confounding variables.

An interesting finding was that approximately 80% (more in the intervention phase) of women with uterine atony had received prophylactic uterotonics; this increase was probably due to the protocol of prophylaxis having gained more acceptance during the mid-2000s. Despite the increasing rate of prophylaxis, women continue to experience postpartum hemorrhage caused by uterine atony. At least 97% of women with uterine atony in each phase received uterotonics for treatment; however, women who were treated with the NASG and uterotonics received significantly fewer doses of uterotonics. Furthermore, fewer women with uterine atony underwent emergency hysterectomy, perhaps because of the decrease in bleeding and faster recovery from shock—which would

Table 4
Multiple logistic regression model of factors predictive of extreme adverse outcome.^a

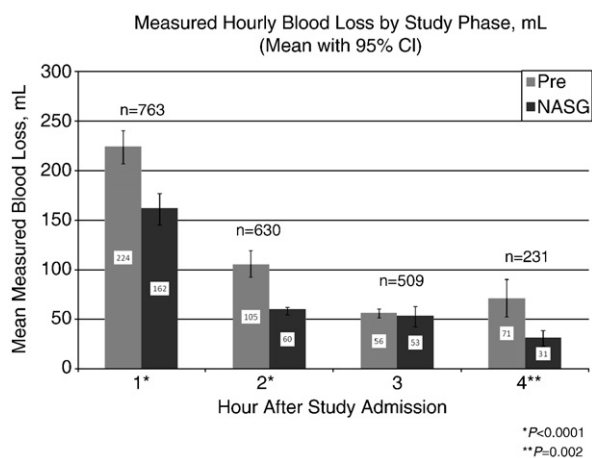
	Odds ratio	<i>P</i> value	95% confidence interval
Severity of shock			
MAP ≥ 60 mm Hg ^b	1.00	<0.01	1.91–11.02
MAP < 60 mm Hg	4.58		
Hemoglobin, g/dL ^c	0.47	<0.01	0.33–0.66
Hospital			
Assiut ^b	1.00	0.98	0.34–3.05
El Galaa	1.01		
Where bleeding began			
In hospital ^b	1.00	0.89	0.35–3.39
Outside hospital	1.09		
Primary diagnosis			
Other condition ^b	1.00	<0.01	1.47–7.85
Uterine atony	3.40		
Study phase			
Pre-intervention ^b	1.00	0.02	0.17–0.85
Intervention	0.38		

Abbreviation: MAP, mean arterial pressure.

^a n = 989 (1 case lacked data on blood pressure).

^b Reference group.

^c Thirty cases lacked data on hemoglobin at study entry. The hemoglobin value was imputed for those 30 cases; missing data were replaced with the mean value of the distribution of non-missing cases. Mean measured hemoglobin upon study entry was the only continuous independent variable.

**Fig. 2.** Measured hourly blood loss by study phase.

have enabled medical care providers to manage hemorrhage more methodically. Another interesting finding was the high percentage of etiologies not caused by uterine atony; although the literature states that uterine atony causes the majority of obstetric hemorrhage [14,15], this condition was responsible for less than 40% of severe hemorrhage with hypovolemic shock in the present study. This seemingly smaller role of uterine atony (despite still being the largest single contributor to obstetric hemorrhage) may be related to increased use of prophylactic uterotonics, better treatment of uterine atony, and/or previous overestimates.

The proportion of women who received at least 1500 mL of intravenous fluids in the first hour after study admission, the proportion who received a blood transfusion in the first hour after study admission, and the volume of blood products transfused were all significantly lower in the intervention phase than in the pre-intervention phase. There was also a statistically significant decrease in the number of uterotonic doses given to women with uterine atony in the intervention phase. These variables were not significantly related to EAO, although this finding, given the similarity in women's condition on study entry, raises the issue of possible complacency on the part of providers with regard to the introduction of a beneficial technology—with important implications for training in the introduction of the NASG. However, it may be that the more rapid recovery and decreased bleeding of the women in the intervention phase led to a clinical decision to reduce the level of fluid replacement, the volume and initiation of blood replacement therapy, and the amount of uterotonics administered.

Major limitations to the study were inherent in the non-randomized, non-blinded, and pre-intervention/intervention design. Selection bias is always possible in a non-randomized study in which enrollment is dependent upon clinician choice. It is possible that some women with hemorrhage and shock were not enrolled during the study and that the worst cases were not enrolled during the intervention phase; however, hospital-level statistics for both facilities showed 15 obstetric hemorrhage deaths in the pre-intervention phase and 6 obstetric hemorrhage deaths in the intervention phase. Also, because the pre-intervention phase occurred first, it is possible that clinicians' skills in terms of the management of hemorrhage improved over time. There were statistically significant differences in etiologies between the 2 phases, with more women with ectopic pregnancy, complications of abortion, placental abruption, ruptured uterus, and placenta previa in the pre-intervention phase. This imbalance was adjusted for by including uterine atony versus other etiologies in the multiple logistic regression model, but a larger study comparing individual etiologies may be warranted. Finally, the study was conducted only at the referral/CEmOC level. The public health implications for NASG use might be greater at the community level, given the lack of access to even basic emergency obstetric care below referral level and the number of women who still deliver at home with unskilled attendants.

Obstetric hemorrhage remains a leading cause of maternal mortality. Despite advances in the prevention and treatment of uterine atony, this condition is not the only etiology of obstetric hemorrhage. Furthermore, hemorrhagic shock is experienced by women delivering in their communities or homes who have limited access to immediate

definitive treatment. The NASG is a new technology that can afford women—particularly those delivering at home or at the community level—time until they can access CEmOC facilities. The present study in Egypt demonstrated the contribution of the NASG toward reducing EAO at the referral hospital level. An experimental study that begins with women at the primary-healthcare level who are transferred in the NASG to a CEmOC facility is essential for a comprehensive assessment of the potential of this technology.

5. Conflict of interest

The authors have no conflicts of interest.

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References

- [1] Hofmeyr GJ, Mohlala BK. Hypovolaemic shock. *Best Pract Res Clin Obstet Gynaecol* 2001;15(4):645–62.
- [2] Miller S, Turan JT, Dau K, Bray E, Fathalla MF, Mourad M, et al. Innovation for managing hypovolemic shock, uterine atony, and obstetric hemorrhage in low-resource settings: the non-pneumatic anti-shock garment (NASG). Washington, DC: 33rd Annual International Conference for Global Health 2006.
- [3] Miller S, Ojengbede O, Turan JM, Morhason-Bello IO, Martin HB, Nsima D. A comparative study of the non-pneumatic anti-shock garment for the treatment of obstetric hemorrhage in Nigeria. *Int J Gynecol Obstet* 2009;107(2):121–5.
- [4] Brees C, Hensleigh PA, Miller S, Pelligra R. A non-inflatable anti-shock garment for obstetric hemorrhage. *Int J Gynecol Obstet* 2004;87(2):119–24.
- [5] Hensleigh PA. Anti-shock garment provides resuscitation and haemostasis for obstetric haemorrhage. *BJOG* 2002;109(12):1377–84.
- [6] Miller S, Hamza S, Bray EH, Lester F, Nada K, Gibson R, et al. First aid for obstetric haemorrhage: the pilot study of the non-pneumatic anti-shock garment in Egypt. *BJOG* 2006;113(4):424–9.
- [7] McSwain NE. PASG in the 90s. *Emergency* 1994(26):28–33.
- [8] Miller S, Hensleigh P. Non-pneumatic Anti-shock Garment for Obstetric Hemorrhage. In: B-Lynch C, Keith L, LaLonde A, Karoshi M, editors. *Postpartum Hemorrhage: New Thoughts, New Approaches*. London, UK: Sapiens Publications; 2006. p. 136–46.
- [9] Miller S, Turan JM, Dau K, Fathalla M, Mourad M, Sutherland T, et al. Use of the non-pneumatic anti-shock garment (NASG) to reduce blood loss and time to recovery from shock for women with obstetric haemorrhage in Egypt. *Glob Public Health* 2007;2(2):110–24.
- [10] Miller S, Martin HB, Morris JL. Anti-shock garment in postpartum haemorrhage. *Best Pract Res Clin Obstet Gynaecol* 2008;22(6):1057–74.
- [11] Martel MJ, MacKinnon KJ, Arsenault MY, Bartellas E, Klein MC, Lane CA, et al. Clinical Practice Obstetrics Committee and Executive and Council, Society of Obstetricians and Gynaecologists of Canada. Hemorrhagic shock. *J Obstet Gynaecol Can* 2002;24(6):504–20.
- [12] Mantel GD, Buchmann E, Rees H, Pattinson RC. Severe acute maternal morbidity: a pilot study of a definition for a near-miss. *BJOG* 1998;105(9):985–90.
- [13] The Criteria Committee of the New York Heart Association. *Nomenclature and Criteria for Diagnosis of Disease of the Heart and Great Vessels*. Ninth ed. Boston, Massachusetts, USA: Little, Brown; 1994.
- [14] Derman RJ, Kodkany BS, Goudar SS, Geller SE, Naik VA, Bellad MB, et al. Oral misoprostol in preventing postpartum haemorrhage in resource-poor communities: a randomised controlled trial. *Lancet* 2006;368(9543):1248–53.
- [15] McCormick ML, Sanghvi HC, Kinzie B, McIntosh N. Preventing postpartum hemorrhage in low-resource settings. *Int J Gynecol Obstet* 2002;77(3):267–75.