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The long-term prevalence of anal incontinence in women with and without obstetric anal sphincter injuries

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ABSTRACT

Introduction: Anal incontinence (AI) is a distressing and common condition for women. Women who sustain obstetric anal sphincter injuries (OASI) have been reported to have a higher prevalence of long-term AI than those who do not. However, the data is wide ranging, inconsistent and seldom comparative, with rates quoted varying between 13.1% and 74%.

Methods: 139 women who sustained OASI at their first delivery and 44 women who did not (controls) were contacted at 6 weeks and then a mean of 8 years following their first delivery. They were asked to complete a questionnaire including the St Mark's Incontinence Score (SMIS) to assess the prevalence and severity of anal incontinence.

Results: There was a high prevalence of anal incontinence (SMIS ≥1) in both women with OASI (62%) and controls (59%) (NS). The prevalence of severe AI (SMIS ≥ 6) was 27% in the OASI group and 18% in controls (NS). Higher grade tears and operative delivery were associated with higher rates of severe AI. In women with OASI, the mode of subsequent delivery did not impact the prevalence of AI.

Conclusion: Women with OASI have a similar prevalence of long-term AI compared with women who do not sustain OASI. Some degree of AI is common 8 years post-partum, with our study demonstrating a high prevalence of 59% in controls.

Brief summary: Women with and without OASI were contacted 5–15 years post-partum, and there was no difference in the rates of anal incontinence between the two groups.

1. Introduction

Obstetric anal sphincter injury (OASI) affects 0.6%–9% of all vaginal births, with a higher incidence in primiparous women, and is considered the most common cause of anal incontinence in women [1]. Anal incontinence (AI) is the involuntary loss of solid, liquid stool or flatus, and can have a significant impact on a woman's quality of life. The existing literature suggests that women with OASI have worse AI outcomes than those without OASI, but the data is wide-ranging with the incidence of AI varying from 19% to 74% [2,3]. However, not all AI after childbirth can be directly attributed to OASI, with other factors being potentially causative including instrumental delivery, and pudendal neuropathy [4].

As well as prevalence of AI, mode of delivery in subsequent births following OASI has been the subject of much investigation. It is difficult to counsel women regarding their subsequent mode of delivery, due to conflicting data regarding the effect of vaginal birth [5–7]. Therefore, following on from our previous study, the aim of this study was to assess the long-term rates of AI in women with and without OASI [8]. The purpose of this study is to gain further information in order to better counsel women regarding their risk of long-term AI as well as the effect of subsequent births.

2. Methods

This is a longitudinal cohort study conducted in a tertiary centre in Sydney, Australia.

Ethics approval was obtained for this study from SESLHD HREC: **2019/STE00147**. In this centre, all women who sustain OASI are reviewed in a dedicated clinic at 6 weeks and 6 months post-partum

Abbreviations: AI, anal incontinence; OASI, Obstetric anal sphincter injury; SMIS, St Mark's incontinence score

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for anorectal manometry testing, endoanal ultrasound and advice regarding future mode of delivery.

Recruitment and consent

Women were sent an initial introduction letter, then contacted by phone to invite them to participate in the study. Those who consented completed a questionnaire either via email or by mail with a postage paid return envelope. The questionnaire included information regarding their current health, subsequent deliveries, and any incontinence symptoms using the St Mark's faecal incontinence score. Questionnaires were also translated into the most common languages seen in our clinic including Mandarin and Arabic. If women did not respond they were sent another questionnaire and a reminder two months afterwards.

OASI group

Data were collected from women with OASI who attended the pelvic floor unit at 6 weeks and 6 months post-partum. Any woman who was seen in this OASI clinic from its inception in 2006 until 2014 was contacted, to allow at least 5 years from delivery and sufficient time for symptom development. At the 6-week postnatal appointment women were seen by a women's health physiotherapist and pelvic floor muscle training and defecatory training is offered to women if pelvic floor muscle assessment reveals weak pelvic floor strength or symptoms are significant. Conservative treatments to manage seepage and constipation are also offered. This clinic follows the RCOG guidelines and recommends vaginal birth for women who are asymptomatic, and caesarean delivery for women who are symptomatic or have significant defects on endoanal ultrasound or anorectal manometry performed at 6 months post-partum.

Written consent for ongoing data collection was obtained from the women at the 6-week visit. A dedicated database was completed prospectively. All OASI were diagnosed by routine per-rectal examination and graded according to the Sultan classification at the time of delivery [9]. Repairs were performed in the operating theatre with end-to-end or overlapping technique by a trained registrar, fellow or consultant, with routine post-operative aperients and antibiotics as standard care. Ultrasound was not used during diagnosis or surgical repair of OASI.

Control group

Data from the control group was obtained from a database created for a previous study published by this research group in 2014 investigating factors contributing to OASI. This group included nulliparous women giving birth between 2010 and 2013 in our hospital. They were recruited from the delivery suite on a sequential basis by a single investigator, where nulliparous women were approached in early labour and included if they did not sustain more than a second-degree perineal injury [10]. Written consent for the study was obtained. A previous study published by this research group in 2020 analysed 6-week AI outcomes between the OASI and control groups thus 6-week data was available for comparison [8].

Outcome measures

The St Mark's Incontinence Score (SMIS) was used to assess the severity of AI. The SMIS is a validated tool for measuring severity of AI, with a total score ranging from 0 (complete continence) to 24 (complete incontinence) based on seven questions [11]. The SMIS was analysed as a categorical variable as it was not normally distributed. SMIS $\geq$ 1 was anal incontinence, and severe anal incontinence was defined as SMIS $\geq$ 6. These definitions are in keeping with other studies within this patient group [2,11,12]. Subgrouping of results was based on the severity of the OASI at the time of delivery.

3. Statistical analysis

Data were analysed using SPSS v.26 (IBM Corp., Armonk, NY). Binary logistic regression analysis was undertaken for independent continuous and categorical variables with significance designated as less than 0.05. All continuous variables underwent the Box–Tidwell test (1962) to confirm the assumption of linearity [13]. The Mann–Whitney U test was used where means were compared. Multivariate analysis was performed using logistic regression. Due to low numbers of grade 3c and 4 tears, these two grades were combined into one grade of severe tears for statistical analysis.

A power calculation was performed to assess sample size required. Assuming a rate of incontinence of 11% in controls based on Handa et al. (2011), and an incontinence rate of 35% in women with OASI based on Evers. et al. (2012), an alpha value of 0.05, and a ratio of cases to controls 1:1, 94 patients were required to achieve 80% power, with a minimum of 47 women required in each group [3,14].

4. Results

From the prospective pelvic floor data clinic database, 419 women were identified between 2006 and 2014, and 139 in the control group from our previous study [10]. Responses to the survey were obtained in 136 (32%) women with OASI and 44 (32%) women in the control group. The response rate was 32% for both groups ($p = 0.89$). There was a significantly higher proportion of Asian women in the non-responding group (38% vs. 23%, $p = 0.001$), possibly due to the language barrier despite the offer of translated materials and a translator. The group who responded was more likely to have had AI (SMIS $\geq$ 1) than non-responders at 6 weeks post-partum ($p = 0.04$), but not severe AI (SMIS $\geq$ 6) ($p = 0.06$). Thus, there was a slight bias of our data towards women who were symptomatic.

The demographics for those who responded are displayed in Table 1. Mean follow-up time was slightly longer in OASI group, and there was a higher proportion of Asian women in the OASI group compared to controls ($p = 0.002$). There was a similar number of subsequent births in both groups after the first birth ($p = 0.35$). However, women with OASI were significantly more likely to have had a caesarean delivery for subsequent births than controls. There was a similar rate of pre-pregnancy bowel symptoms in both groups.

There was no difference in the rate of AI or severe AI between women with OASI and controls when all Sultan classifications are examined collectively (Table 2). Women with 3c and 4th degree tears reported more severe AI compared to controls ($p = 0.03$).

Regardless of OASI, 23/174 (13.2%) women reported severe AI at the 6-week postpartum visit. The women who had severe AI at 6 weeks post-partum were significantly more likely to report long-term severe AI compared to those who had SMIS < 6 at that same early post-partum period ($P = 0.002$) (Table 3).

The rate of AI was further analysed by type of leakage — solid stool, liquid stool and/or gas. When analysing these variables, women who reported any incontinence of solid stool (rarely, sometimes, weekly or daily) were combined, as were women who reported any incontinence of liquid stool or gas combined as one variable (Table 4). There was no difference in the rate of incontinence to either solid stool, or liquid/gas between women with OASI and controls.

The effect of subsequent deliveries after OASI on long-term AI was assessed according to the mode of subsequent birth (no further births versus only vaginal births versus at least one caesarean delivery) (Table 5). There was no difference in the rate of long-term AI based on mode of delivery. Women who had 3a tears were significantly more likely have had all vaginal births ($p = 0.01$). A greater percentage of women with 3c/4 tears had a caesarean delivery although this was not statistically significant ($p = 0.06$).

The impact of mode of first delivery was assessed in all women with OASI and controls combined, comparing non-instrumental with vacuum and forceps deliveries. Women undergoing forceps delivery and vacuum delivery were more likely to have severe AI in the long-term compared with women who had a non-instrumental delivery ($P = 0.03$ and 0.04 respectively) (Table 6).

Table 1
Demographics for women who responded.

	OASI (n = 136)	Control (n = 44)	P-value
Mean follow-up period in years (sd)	8.91 (2.48)	7.35 (1.36)	**0.00**
Mean current age in years (sd)	39.2 (4.9)	37.6 (4.8)	0.07
Mean Birthweight in g (sd)	3377 (629)	3243 (449)	**0.03**
Mean Gestation in weeks (sd)	39.6 (1.4)	39.3 (1.3)	0.23
Menopausal (%)	15 (11)	7 (15.9)	0.43
Asian race (%)	40 (29)	3 (6.8)	**0.002**
South-Asian race (%)	13 (9.6)	2 (4.5)	0.29
Operative delivery (%)	60 (44.1)	12 (27.3)	**0.05**
Vacuum (%)	34 (25.2)	8 (18.2)	0.35
Forceps (%)	26 (19.3)	4 (9.1)	0.11
Episiotomy (%)	43 (32.6)	18 (40.9)	0.31
Mean no. of total births (sd)	1.88 (0.75)	2.02 (0.85)	0.35
Mean no. of vaginal births (sd)	1.45 (0.69)	1.86 (0.82)	**0.001**
No. of women who had an LSCS at some point (%)	48 (35.6)	7 (15.9)	**0.01**
Pre-pregnancy bowel symptoms (%)[a]	9 (11)	2 (7.4)	0.59
Current bowel condition (%)[b]	14 (10.3)	1 (2.3)	0.13

[a]Pre-pregnancy bowel symptoms included any history of AI, constipation, straining, incomplete emptying or haemorrhoids.
[b]Current bowel conditions included Inflammatory bowel disease, irritable bowel syndrome and constipation.

Table 2
Rate of long-term AI in OASI and controls.

	SMIS ≥ 1 (%)	p-value	OR (95% CI)	SMIS ≥ 6 (%)	p-value	OR (95% CI)
Control (n = 46)	26 (59)			8 (18)		
OASI (n = 136)	84 (62)	0.71	1.14 (0.57–2.28)	36 (27)	0.26	1.64 (0.70–3.85)
3a tear (n = 47)	24 (51)	0.44	0.72 (0.32–1.66)	7 (15)	0.67	0.79 (0.26–2.39)
3b tear (n = 49)	34 (69)	0.30	1.57 (0.67–3.67)	15 (31)	0.17	1.99 (0.74–5.28)
3c/4 tear (n = 32)[a]	23 (72)	0.25	1.77 (0.67–4.70)	13 (41)	**0.03**	3.08 (1.09–8.72)

All variables were compared with controls.

Table 3
Predictive value of long-term AI from symptoms at 6-weeks post-partum in pooled OASI and controls.

	No AI 6-weeks (n = 115)	AI 6-weeks (n = 59)	p-value	OR (95% CI)	SMIS < 6 at 6-weeks (n = 151)	Severe AI at 6 weeks (n = 23)	p-value	OR (95% CI)
SMIS ≥ 1 longterm	64 (55.7)	42 (71.2)	**0.048**	1.97 (1.01–3.86)	88 (58.3)	18 (78.3)	0.21	1.58 (0.78–3.21)
SMIS ≥ 6 longterm	25 (21.7)	18 (30.5)	0.08	2.58 (0.91–7.31)	31 (20.5)	12 (52.2)	**0.002**	4.22 (1.7–10.48)

Table 4
Rate of long-term AI based on type of leakage in OASI and controls.

	OASI (n = 136)	Controls (n = 44)	p-value	OR (95% CI)
Solid stool	28 (20.7%)	6 (13.6%)	0.30	1.66 (0.64–4.31)
Liquid stool and gas	73 (54.1%)	23 (52.3%)	0.83	1.08 (0.54–2.13)

NB: Women with any incontinence of solid stool on the SMIS were combined, and the same for liquid stool and gas.

Table 5
Effect of subsequent deliveries on long-term AI in women with OASI.

	SMIS ≥ 1 (%)	p-value	OR	SMIS ≥ 6 (%)	p-value	OR
No further births (n = 41)	27 (66)			9 (22)		
All vaginal (n = 44)	23 (53)	0.21	0.57 (0.24–1.36)	7 (16)	0.48	0.67 (0.23–2.01)
At least 1 caesarean (n = 49)	33 (67)	0.88	1.07 (0.44–2.58)	19 (39)	0.09	2.25 (0.88–5.75)

Table 6
Impact of first delivery mode on long-term AI (all women combined).

	SMIS ≥ 1 (%)	p-value	OR (95% CI)	SMIS ≥ 6 (%)	p-value	OR (95% CI)
Non-instrumental (n = 107)	61 (57)	–	–	19 (17.8)	–	–
Vacuum delivery (n = 41)	26 (63.4)	0.48	1.31 (0.62–2.75)	14 (34.1)	**0.04**	2.4 (1.06–5.42)
Forceps delivery[a] (n = 30)	23 (76.7)	0.06	2.48 (0.98–6.27)	11 (36.7)	**0.03**	2.68 (1.10–6.55)

[a]Forceps delivery included women who had a failed vacuum/forceps delivery.

In order to assess the benefit of a 6-month follow-up for women with OASI following ano-rectal physiology testing, the long-term AI rates was compared between the 98 women who attended and the 38 women who did not attend the 6-month ano-rectal physiology and counselling. There was no significant difference for any long-term AI or severe AI between women who attended this follow-up and those that did not (p = 0.75 and 0.37 respectively).

Multivariate analysis was performed to examined control or OASI, sultan classification, mode of delivery, subsequent deliveries, maternal age, and birthweight. The only significant factor associated with long-term AI was AI at 6-weeks post-partum (p = 0.048). For severe AI, the significant factors were forceps delivery (p = 0.05), mode of subsequent delivery (caesarean delivery, p = 0.03) and having severe AI at 6-weeks (p = 0.03).

5. Discussion

This study found a prevalence of long-term AI in women of approximately 60% at a mean of 8 years after the first vaginal birth regardless of whether women sustained OASI or not. The study is a longitudinal comparative trial, with systematic and prospective collection of data from birth, the early post-partum period and timepoints at 6 months and finally 8 years after the first birth following the years where most women continue to grow their families. Data collected included the serial use of the same validated AI severity measurement tool at all timepoints.

While our study did not include a quality-of-life instrument, it is known that women with a higher SMIS (≥ 6 in this study) have a poorer quality of life [14]. In our study, 27% of women with OASI reported severe AI long-term compared with 18% of controls (p = 0.26) suggesting that factors other than just OASI account for severe AI in women. This is consistent with other studies that report a prevalence of AI at 12%–21% at 6–12 months post-partum [14].

We found the independent predictors for long-term severe AI included severe AI at the 6- week postnatal visit, and forceps birth. In absolute terms, the chance of a woman with severe AI symptoms at the 6- week postpartum visit having severe AI long-term was 52%. If she was asymptomatic or had mild AI symptoms at the 6-week visit, the chance of severe AI long-term was significantly less at 21%. When the groups were examined independently, 16% of controls who were asymptomatic at 6-weeks post-partum had severe AI long-term, compared to 24% of women with OASI (p = 0.38).

Retrospective studies have demonstrated high rates of incontinence in all women 30 years after OASI compared with controls and caesarean deliveries [15]. They found no difference in the rates of flatal and faecal incontinence, however women with OASI were more likely to suffer bothersome flatal incontinence. Faltin et al. (2006) found that women with OASI had slightly more fecal incontinence than controls at 18 years post-delivery (51.0% vs. 40.2%), but the impact on quality of life was the same in both groups [16].

A number of other studies have found higher rates of long-term AI in women with OASI compared with controls [3,7,17–20]. A systematic review by LaCross et al. (2015) found that OASI are significantly associated with AI after vaginal birth with an odds ratio of 2.66 [21]. Soerensen et al. (2013) found that women with OASI were twice as likely to have AI (51%) compared with controls (26%), however they only included women with complete anal sphincter tears rather than partial. Our study concurs with these results that demonstrate women with more severe (or complete) tears were found to have higher rates of AI in the long-term [1].

Overall, similar to other studies, we found that AI is common in all women who have had a vaginal birth, regardless of OASI [2,22,23]. Another study in women with OASI a median of 6 years post-delivery performed in a Sydney hospital found a similar rate of anal incontinence to our study with 61% having a SMIS ≥ 1 though it lacked controls [23]. Linneberg et al. (2016) also found a high rate of AI (74%) in women with OASI at 5 year follow-up [2]. Other studies however have found lower rates of AI such as Guzman Rojas (2018) et al. who reported fecal incontinence in 16.7% of women with OASI and 8.9% in controls however they used a more severe definition of fecal incontinence to be involuntary loss of loose or solid stool [24,25]. The high rate of AI in our study may be due to the chosen definition of AI as SMIS ≥ 1, however flatal incontinence can also be distressing to women. The high prevalence of AI in our study would suggest that some degree of mild AI is normal for post-partum women.

The only Sultan Classification group in our study that had a higher risk of long-term severe AI was women with higher grade 3c and 4 tears, suggesting that severe tears are the main concern rather than all OASI. Several other studies support this finding [2,26]. However, fortunately women with more common 3a and 3b tears can be reassured that they are likely to have similar rates of AI than women without OASI. We found that AI 6-weeks post-partum was a significant predictor for long-term AI, which was also seen by Pollack et al. (2004) [20]. Women with AI at this early stage should be counselled that they may continue to suffer some AI long-term, and future research must focus on measures to prevent severe tears from occurring. Thus in the early post-partum period we can reassure those without AI that they are less likely to suffer long-term AI.

Othman et al. (2022) assessed faecal incontinence in non-pregnant nulliparous women aged 25–64 years and found that isolated leakage of solid stool was rare, with leakage of liquid and gas much more common, postulating that stool consistency rather than the pelvic floor itself plays a significant role in incontinence in these women [27]. Our chosen cut-off of SMIS ≥ 1 does include all incontinence, and thus could cause some overlap with women who have irritable bowel syndrome or issues with stool consistency as a possible cause of their incontinence which may contribute to the lack of difference between the two groups. Nilsson et al. demonstrated higher rates of AI in women with OASI, and particularly with 2 or more OASI over a 20-year period. They analysed the different types of leakage — solid and liquid stool vs. gas leakage only, and found no difference in rates of gas incontinence, but a more pronounced difference for solid and liquid stool [28]. When analysing for types of leakage, our data did not show any difference in AI between the two groups, however it was likely underpowered to properly assess this.

Our study found that the mode of subsequent delivery did not have an impact on the rates of long-term AI as shown in several other studies [5,6,23]. Jango. et al. (2016) found no significant impact of subsequent delivery on rate of long-term AI. However, they did find women who had a subsequent vaginal delivery had a higher risk of deterioration of AI symptoms compared with an elective CS [6]. Jordan et al. (2018) used a comprehensive protocol including SMIS, anal manometry and endoanal ultrasound to decide on mode of subsequent delivery in women with OASI. Women with minor symptoms or asymptomatic with minor EAS defect were recommended a vaginal delivery, while all other women were recommended a caesarean delivery, similar to our protocol. They found no significant worsening of AI in the vaginal delivery group compared with caesarean delivery [5]. In our study, women with caesarean deliveries had significantly more long-term severe AI than women who only had vaginal births. This is likely because women with more severe AI symptoms were recommended a caesarean delivery, while women who were asymptomatic were recommend vaginal births. Turel et al. (2019) found that women who had caesareans were more likely to have a residual EAS defect on ultrasound [23]. While our sample size was small, our data supports the advice that women who are asymptomatic and have more common 3a and 3b tears are generally safe to have a vaginal delivery and can be reasonably confident that they will not have worse incontinence than women without OASI, however women should be warned that a caesarean delivery is not protective for AI. Further research is needed in this area.

Our study demonstrated a higher rate of long-term severe AI in women who had an operative delivery, either vacuum or forceps which is a finding well documented by other research [14,16,25]. Forceps delivery is associated with a higher rate of occult sphincter defects, as well as neurological damage [24]. The risk of higher grade OASI is also present with forceps and following from this, higher grade tears are associated with more anal incontinence. There was a higher rate of operative delivery in our OASI group compared with controls, which will likely confound the risk of AI, however interestingly while forceps and vacuum deliveries were both significantly associated with long-term severe AI, OASI was not. Steps to decrease the prevalence of instrumental birth could decrease the anal incontinence rate, as could variations in the mix of vacuum versus forceps, but this issue is multifaceted and would not be resolved easily without a randomised controlled trial.

It is likely that some of the AI seen in the control group is due to occult sphincter defects as ultrasound was not used to identify

OASI. Occult sphincter injury is reported in 16%–53% of primiparous women [4]. Guzman Rojas et al. (2018) found that 15% of women had sphincter defects on ultrasound 15–24 years after their primary delivery, and 79% of the defects were not identified at the time. They also found that women with ongoing anal sphincter defects were more likely to have fecal incontinence [24]. Other factors such as levator avulsion also contribute to AI and may have been present in some of our participants, however this was not investigated [23].

6. Limitations

A significant limitation of this study was the poor response rate. This is common for long-term studies and has been seen in other similar studies likely due to the high mobility of this young population [2,14,23,25,29]. It is possible that those women who do not respond are less symptomatic as women who respond are more likely to be symptomatic, and this was demonstrated in Table 1. This may have also produced a bias in our results and contributed to the high rate of AI. Thus we expect the true rates of FI to be substantially lower than reported.

Further, while our follow-up time of 8 years was a long period, AI is known to worsen over time, and follow-up over 20 or 30 years may reveal different results.

Unfortunately, we did not quite achieve our power calculation in the control group thus were slightly underpowered. This may have affected our ability to see a statistically significant difference between OASI and controls. As discussed in the methods, as there is no agreed cut-off SMIS to define severe AI, we chose an arbitrary value of ≥6. Use of another cut-off value may have resulted in different outcomes. However, a strength of our study was the use of a validated tool to assess incontinence.

7. Conclusion

AI is common in all women who have vaginal births, and our data suggests that women who sustain OASI are not at higher risk unless they sustain a high-grade tear. Women who sustain OASI can be reassured that they will not be worse off than other women who have vaginal deliveries, however perhaps all women should be warned of the risk of AI. Instrumental delivery, and particularly the use of forceps is associated with higher rates of long term anal incontinence.

CRediT authorship contribution statement

Rebecca Everist: Protocol development, data collection, Data analysis, Manuscript writing. **Madeline Burrell:** Data collection. **Katrina Parkin:** Data collection. **Vicki Patton:** Data collection, Manuscript editing. **Emmanuel Karantanis:** Project development, Manuscript editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethics statement

Local ethics approval was obtained for this study: 2019/ETH00113 and 2019/STE00147.

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References

[1] M.M. Soerensen, S. Buntzen, K.M. Bek, S. Laurberg, Complete obstetric anal sphincter tear and risk of long-term fecal incontinence: A cohort study, Dis. Colon Rectum 56 (8) (2013) 992–1001, http://dx.doi.org/10.1097/DCR.0b013e318299c209.

[2] S. Linneberg, S. Leenskjold, K. Glavind, A five year follow-up of women with obstetric anal sphincter rupture at their first delivery, Eur. J. Obstet. Gynaecol. Reprod. Biol. 203 (2016) 315–319, http://dx.doi.org/10.1016/j.ejogrb.2016.06.018.

[3] E.C. Evers, J.L. Blomquist, K.C. McDermott, V.L. Handa, Obstetrical anal sphincter laceration and anal incontinence 5-10 years after childbirth, Am. J. Obstet. gynaecol. 207 (5) (2012) 425, http://dx.doi.org/10.1016/j.ajog.2012.06.055, e421-426.

[4] L.K.G. Persson, A. Sakse, J. Langhoff-Roos, H. Jangö, Anal incontinence after two vaginal deliveries without obstetric anal sphincter rupture, Arch. Gynaecol. Obstet. 295 (6) (2017) 1399–1406, http://dx.doi.org/10.1007/s00404-017-4368-y.

[5] P.A. Jordan, M. Naidu, R. Thakar, A.H. Sultan, Effect of subsequent vaginal delivery on bowel symptoms and anorectal function in women who sustained a previous obstetric anal sphincter injury, Int. Urogynaecol. J. 29 (11) (2018) 1579–1588, http://dx.doi.org/10.1007/s00192-018-3601-y.

[6] H. Jangö, J. Langhoff-Roos, S. Rosthøj, A. Sakse, Mode of delivery after obstetric anal sphincter injury and the risk of long-term anal incontinence, Am. J. Obstet. Gynecol. 214 (6) (2016) http://dx.doi.org/10.1016/j.ajog.2015.12.030, p. 733.e1–733.e13.

[7] E.U. Fornell, L. Matthiesen, R. Sjödahl, G. Berg, Obstetric anal sphincter injury ten years after: subjective and objective long term effects, BJOG: Int. J. Obstet. Gynaecol. 112 (3) (2005) 312–316, http://dx.doi.org/10.1111/j.1471-0528.2004.00400.x.

[8] M. Burrell, S. Dilgir, V. Patton, K. Parkin, E. Karantanis, Risk factors for obstetric anal sphincter injuries and postpartum anal and urinary incontinence: a case-control trial, Int. Urogynaecol. J. 26 (3) (2015) 383–389, http://dx.doi.org/10.1007/s00192-014-2478-7.

[9] R. Everist, M. Burrell, K.A. Mallitt, K. Parkin, V. Patton, E. Karantanis, Postpartum anal incontinence in women with and without obstetric anal sphincter injuries, Int. Urogynaecol. J. 31 (11) (2020) 2269–2275, http://dx.doi.org/10.1007/s00192-020-04267-8.

[10] A.H. Sultan, Editorial: Obstetrical perineal injury and anal incontinence, AVMA Med. Legal J. 5 (6) (1999) 193–196, http://dx.doi.org/10.1177/135626229900500601.

[11] A.M. Roos, A.H. Sultan, R. Thakar, St. Mark's incontinence score for assessment of anal incontinence following obstetric anal sphincter injuries (OASIS), Int. Urogynaecol. J. 20 (4) (2009) 407–410, http://dx.doi.org/10.1007/s00192-008-0784-7.

[12] D. Gommesen, E.A.A. Nohr, N. Qvist, V. Rasch, Obstetric perineal ruptures - risk of anal incontinence among primiparous women 12 months postpartum: a prospective cohort study, Am. J. Obstet. Gynecol. (2019) http://dx.doi.org/10.1016/j.ajog.2019.08.026.

[13] G.E.P. Box, P.W. Tidwell, Transformation of independent variables, Technometrics 4 (4) (1962) 531, http://dx.doi.org/10.2307/1266288.

[14] V.L. Handa, J.L. Blomquist, L.R. Knoepp, K.A. Hoskey, K.C. McDermott, A. Muñoz, Pelvic floor disorders 5-10 years after vaginal or cesarean childbirth, Obstet. Gynaecol. 118 (4) (2011) 777–784, http://dx.doi.org/10.1097/AOG.0b013e3182267f2f.

[15] I.E. Nygaard, S.S. Rao, J.D. Dawson, Anal incontinence after anal sphincter disruption: a 30-year retrospective cohort study, Obstet. Gynaecol. 89 (6) (1997) 896–901, http://dx.doi.org/10.1016/s0029-7844(97)00119-1.

[16] D.L. Faltin, M. Otero, P. Petignat, M.R. Sangalli, L.A. Floris, M. Boulvain, O. Irion, Women's health 18 years after rupture of the anal sphincter during childbirth: I. Fecal incontinence, Am. J. Obstet. Gynecol. 194 (5) (2006) 1255–1259, http://dx.doi.org/10.1016/j.ajog.2005.10.797.

[17] T.K. Halle, K. Salvesen, I. Volløyhaug, Obstetric anal sphincter injury and incontinence 15-23 years after vaginal delivery, Acta Obstet. Gynaecol. Scand. 95 (8) (2016) 941–947, http://dx.doi.org/10.1111/aogs.12898.

[18] M. Mous, S.A. Muller, J.W. de Leeuw, Long-term effects of anal sphincter rupture during vaginal delivery: faecal incontinence and sexual complaints, BJOG: Int. J. Obstet. Gynaecol. 115 (2) (2008) 234–238, http://dx.doi.org/10.1111/j.1471-0528.2007.01502.x.

[19] D.N. Samarasekera, M.T. Bekhit, Y. Wright, R.H. Lowndes, K.P. Stanley, J.P. Preston, P. Preston, C.T. Speakman, Long-term anal continence and quality of life following postpartum anal sphincter injury, Colorectal Dis.: Off. J. Assoc. Coloproctol. Great Britain Ireland 10 (8) (2008) 793–799, http://dx.doi.org/10.1111/j.1463-1318.2007.01445.x.

[20] J. Pollack, J. Nordenstam, S. Brismar, A. Lopez, D. Altman, J. Zetterstrom, Anal incontinence after vaginal delivery: a five-year prospective cohort study, Obstet. Gynaecol. 104 (6) (2004) 1397–1402, http://dx.doi.org/10.1097/01.AOG.0000147597.45349.e8.

[21] A. LaCross, M. Groff, A. Smaldone, Obstetric anal sphincter injury and anal incontinence following vaginal birth: a systematic review and meta-analysis, J. Midwifery Womens Health 60 (1) (2015) 37–47, http://dx.doi.org/10.1111/jmwh.12283.

[22] T.H. Lee, A.E. Bharucha, How to perform and interpret a high-resolution anorectal manometry test, J. Neurogastroenterol. Motil. 22 (1) (2016) 46–59, http://dx.doi.org/10.5056/jnm15168.

[23] F.D. Turel, S. Langer, K.L. Shek, H.P. Dietz, Medium- to long-term follow-up of obstetric anal sphincter injury, Dis. Colon Rectum 62 (3) (2019).

[24] R.A. Guzmán Rojas, K. Salvesen, I. Volløyhaug, Anal sphincter defects and fecal incontinence 15-24 years after first delivery: a cross-sectional study, Ultrasound Obstet. Gynaecol.: Off. J. Int. Soc. Ultrasound Obstet. Gynaecol. 51 (5) (2018) 677–683, http://dx.doi.org/10.1002/uog.18827.

[25] L.M. Dolan, P. Hilton, Obstetric risk factors and pelvic floor dysfunction 20 years after first delivery, Int. Urogynaecol. J. 21 (5) (2010) 535–544, http://dx.doi.org/10.1007/s00192-009-1074-8.

[26] A.H. Sultan, M.A. Kamm, C.N. Hudson, C.I. Bartram, Third degree obstetric anal sphincter tears: risk factors and outcome of primary repair, BMJ (Clin. Res. Ed.) 308 (6933) (1994) 887–891, http://dx.doi.org/10.1136/bmj.308.6933.887.

[27] J. Al-Mukhtar Othman, S. Åkervall, I.E.K. Nilsson, M. Molin, I. Milsom, Gyhagen, Fecal incontinence in nonpregnant nulliparous women aged 25 to 64 years-a randomly selected national cohort prevalence study, Am. J. Obstet. Gynaecol. 226 (2022) 706, e1-23.

[28] I.E.K. Nilsson, S. Åkervall, M. Molin, I. Milsom, M. Gyhagen, Symptoms of fecal incontinence two decades after no, one, or two obstetrical anal sphincter injuries, Am. J. Obstet. Gynaecol. 224 (2021) 276, e1-23.

[29] S. Cornelisse, L.P. Arendsen, S.M.J. van Kuijk, K.B. Kluivers, J. van Dillen, M. Weemhoff, Obstetric anal sphincter injury: a follow-up questionnaire study on longer-term outcomes, Int. Urogynaecol. J. 27 (10) (2016) 1591–1596, http://dx.doi.org/10.1007/s00192-016-3017-5.