



# ISUOG Virtual World Congress

## ON ULTRASOUND IN OBSTETRICS & GYNECOLOGY

15-17 OCTOBER 2021

## Book your place at World Congress 2021

### Confirmed topics include

---

Artificial intelligence

---

Fetal interventions

---

Pelvic pain and endometriosis

---

Ultrasound in labour ward

---

Fetal disease and structural abnormalities

---

Managing Ovarian Masses

---

Foetal and maternal infections

---

### First Confirmed speakers include:

---

Beryl Benacerraf, MD

---

Professor Basky Thilaganathan

---

Professor Christoph Lee

---

Professor Liona Poon

---

Professor Lil Valentin

---

...and more

---

Our platform will give you the opportunity to view and engage with the content and speakers live or at a time and place that suits you.

You will also receive exclusive access to all presentations and lectures for 12 months after the event.

REGISTER TODAY  
[isuog.org](https://isuog.org)



# Anal sphincter defects and fecal incontinence 15–24 years after first delivery: a cross-sectional study

R. A. GUZMÁN ROJAS<sup>1,2</sup>, K. Å. SALVESEN<sup>3,4</sup> and I. VOLLØYHAUG<sup>3,4</sup> 

<sup>1</sup>Departamento de Ginecología y Obstetricia, Facultad de Medicina, Clínica Alemana - Universidad del Desarrollo, Santiago, Chile;

<sup>2</sup>Departamento de Ginecología y Obstetricia, Hospital Clínico de la Universidad de Chile, Santiago, Chile; <sup>3</sup>Department of Clinical and Molecular Medicine, Norwegian University of Science and Technology, Trondheim, Norway; <sup>4</sup>Department of Obstetrics and Gynaecology, Trondheim University Hospital, Trondheim, Norway

**KEYWORDS:** anal sphincter defect; fecal incontinence; forceps delivery; obstetric anal sphincter injury; transperineal ultrasound; vacuum delivery

## ABSTRACT

**Objectives** To establish the prevalence of external (EAS) and internal (IAS) anal sphincter defects present 15–24 years after childbirth according to mode of delivery, and their association with development of fecal incontinence (FI). The study additionally aimed to compare the proportion of women with obstetric anal sphincter injuries (OASIS) reported at delivery with the proportion of women with sphincter defect detected on ultrasound 15–24 years later.

**Methods** This was a cross-sectional study including 563 women who delivered their first child between 1990 and 1997. Women responded to a validated questionnaire (Pelvic Floor Distress Inventory) in 2013–2014, from which the proportion of women with FI was recorded. Information about OASIS was obtained from the National Birth Registry. Study participants underwent four-dimensional transperineal ultrasound examination. Defect of EAS or IAS of  $\geq 30^\circ$  in at least four of six slices on tomographic ultrasound was considered a significant defect and was recorded. Four study groups were defined based on mode of delivery of the first child. Women who had delivered only by Cesarean section (CS) constituted the CS group. Women in the normal vaginal delivery (NVD) group had NVD of their first child and subsequent deliveries could be NVD or CS. The forceps delivery (FD) group included women who had FD, NVD or CS after FD of their first born. The vacuum delivery (VD) group included women who had VD, NVD or CS after VD of their first born. Multiple logistic regression was used to calculate adjusted odds ratios (aORs) for comparison of prevalence of an EAS defect following different modes of delivery and to test its association with FI. Fisher's exact

test was used to calculate crude odds ratios (ORs) for IAS defects.

**Results** Defects of EAS and IAS were found after NVD (n = 201) in 10% and 1% of cases, respectively, after FD (n = 144) in 32% and 7% of cases and after VD (n = 120) in 15% and 4% of cases. No defects were found after CS (n = 98). FD was associated with increased risk of EAS defect compared with NVD (aOR = 3.6; 95% CI, 2.0–6.6) and VD (aOR = 3.0; 95% CI, 1.6–5.6) and with increased risk of IAS defect compared with NVD (OR = 7.4; 95% CI, 1.5–70.5). The difference between VD and NVD was not significant for EAS or IAS. FI was reported in 18% of women with an EAS defect, in 29% with an IAS defect and in 8% without a sphincter defect. EAS and IAS defects were associated with increased risk of FI (aOR = 2.5 (95% CI, 1.3–4.9) and OR = 4.2 (95% CI, 1.1–13.5), respectively). Of the ultrasonographic sphincter defects, 80% were not reported as OASIS at first or subsequent deliveries.

**Conclusions** Anal sphincter defects visualized on transperineal ultrasound 15–24 years after first delivery were associated with FD and development of FI. Ultrasound revealed a high proportion of sphincter defects that were not recorded as OASIS at delivery. Copyright © 2017 ISUOG. Published by John Wiley & Sons Ltd.

## INTRODUCTION

Anal sphincter injury occurring during vaginal delivery is a major risk factor for fecal incontinence (FI) later in life and may have adverse impact on quality of life<sup>1–5</sup>. FI affects between 2% and 24% of women, depending on the population and definition used<sup>6–8</sup>. Obstetric anal

Correspondence to: Dr I. Volløyhaug, NTNU, Faculty of Medicine, Department of Laboratory Medicine, Children's and Women's Health, PO Box 8905, Medisinsk Teknisk Forskningscenter, 7491 Trondheim, Norway (e-mail: ingrid.volloyhaug@ntnu.no)

Accepted: 28 July 2017

sphincter injury (OASIS) is reported after 0.5–20% of vaginal deliveries, with different prevalence between hospitals and modes of delivery<sup>9–12</sup>. Operative vaginal delivery is the strongest risk factor for OASIS, particularly, the use of forceps along with midline episiotomy<sup>10–15</sup>. Studies have shown that 10–30% of women with OASIS will develop symptoms of FI later in life<sup>4,16–18</sup>. Other risk factors for FI are increasing parity, age and body mass index (BMI)<sup>7,8,18–20</sup>.

The prevalence of OASIS reported at the time of delivery is likely to be an underestimate of the true proportion because of occult tears and missed diagnoses<sup>21–23</sup>. Endoanal ultrasound is currently regarded as the gold standard for evaluation of the anal sphincter<sup>24</sup>. The incidence of OASIS, diagnosed using endoanal ultrasound, has been reported as 35% after vaginal delivery and up to 80% after forceps delivery (FD)<sup>14,21</sup>. Transperineal ultrasound imaging is an alternative technique to endoanal ultrasound<sup>25,26</sup>. This approach has wider availability in most gynecology services, and its use for the assessment of anal sphincter defects is increasing<sup>27,28</sup>. Good agreement between endoanal and transperineal detection of anal sphincter defects has been reported<sup>26</sup>. There are few studies analyzing OASIS at delivery in relation to anal sphincter defects on ultrasound and FI with long-term follow-up, and we found no study that used transperineal ultrasound for evaluation of the anal sphincters after different modes of delivery with a long time interval after childbirth<sup>4,20</sup>.

The objective of this study was to establish the prevalence of anal sphincter defects visualized on transperineal four-dimensional (4D) ultrasound 15–24 years after childbirth, according to mode of delivery and their association with development of FI. Additionally, we aimed to compare the proportion of women with OASIS recorded at the time of delivery with the proportion of women with sphincter defect detected on ultrasound 15–24 years later.

## METHODS

This study is a subanalysis of a previous cross-sectional study of women who delivered their first child between 1 January 1990 and 31 December 1997, and was conducted at Trondheim University Hospital, Norway, between 2013 and 2014<sup>29,30</sup>. FD and vacuum delivery (VD) were performed at approximately the same rate during the time period 1990–1997 (FD in 3–4% and VD in 3–5% of all deliveries), and we assumed that doctors were well trained in both delivery methods.

All women with FD, VD and Cesarean section (CS), and 130 consecutive women with normal vaginal delivery (NVD) from January onwards in each year of the study period were invited to participate in the study. Exclusion criteria were stillbirth, breech delivery and birth weight < 2000 g at first delivery. Sample size calculations of the parent study were based on prevalence of detected levator ani injuries for different modes of delivery, and the results have been published previously<sup>29,30</sup>. A total of 847 women who had answered previously a

questionnaire about pelvic floor disorders and agreed to invitation for examination were invited if they still lived close to Trondheim University Hospital, regardless of the symptoms indicated in the questionnaire. Informed consent was obtained from all study participants. The study was approved by the Regional Committee for Medical and Health Research Ethics (REK midt 2012/666).

Four study groups were defined based on mode of delivery of the first child. Women who had delivered only by CS constituted the CS group. Women in the NVD group had NVD of their first child (including delivery with oxytocin augmentation, epidural analgesia, episiotomy and/or perineal tears) and subsequent deliveries could be NVD or CS, but women with FD or VD were excluded from this group. The FD group included women who had FD, NVD or CS after FD of their first born, but no VD. The VD group included women who had VD, NVD or CS after VD of their first born, but no FD. Women who had both VD and FD were excluded.

The women had completed a Norwegian translation of the validated Pelvic Floor Distress Inventory (PFDI), including queries about leakage of stool. A positive response to the questions ‘Do you usually lose stool beyond your control if your stool is well formed?’ or ‘Do you usually lose stool beyond your control if your stool is loose or liquid?’ was used to define FI, regardless of symptom bother. Obstetric variables (mode of delivery, indication for FD or VD, infant birth weight, parity and OASIS) for the first and any subsequent delivery were obtained from the Medical Birth Registry of Norway and cross-checked with information from the hospital’s birth records. BMI was calculated using information from the questionnaire. Information about malposition, first- and second-degree perineal tears and episiotomy was not available for this study, but mediolateral episiotomy was regarded as routine for FD and VD, and rotational forceps or forceps higher than mid-cavity were not part of the clinical practice. It was therefore assumed that mediolateral episiotomy was performed for all FD and VD and that FD was performed only with the head in the occiput anterior or posterior position at the pelvic floor.

All women underwent 4D transperineal ultrasound examination of their anal sphincter. All ultrasound examinations were performed by one of the authors (I.V.), who was blinded to demographic and clinical data at the time of examination. Women were asked to withhold any information regarding symptoms and previous deliveries until the examination had been completed. They were examined in the supine position in a gynecological examination chair with empty urinary bladder and bowel. 4D ultrasound volumes were acquired with a GE Voluson S6 device (GE Medical Systems, Zipf, Austria) using the RAB 4–8RS abdominal three-dimensional (3D) probe and an acquisition angle of 85°. Volumes were acquired on maximal Valsalva maneuver and on pelvic floor muscle contraction. Archived ultrasound volumes were analyzed in 2016 by the first author (R.G.R.), who was blinded to all clinical and demographic data. The

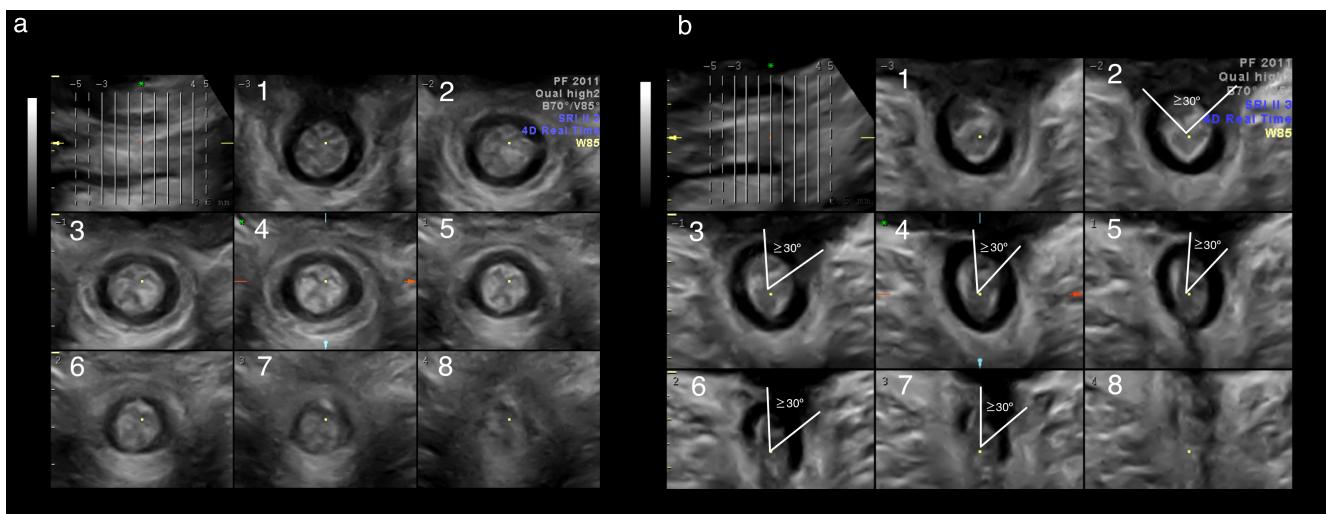


proprietary software 4D View version 14 Ext. 0 (GE Medical Systems) was used for post-processing offline analysis. Tomographic ultrasound imaging (TUI) was used to evaluate the external (EAS) and internal (IAS) anal sphincters on contraction, as described previously<sup>31</sup>. A set of eight slices was obtained in which the entire EAS was encompassed by placing the upper slice cranial to the EAS (at the level of the puborectalis muscle) and the lower slice caudal to the IAS (at the level of the anal verge). The distance between each slice was adjusted depending on the length of the EAS, leaving six slices to delineate the entire muscle (Figure 1a). The IAS was assessed similarly, with the first slice cranial to the IAS (at the level of

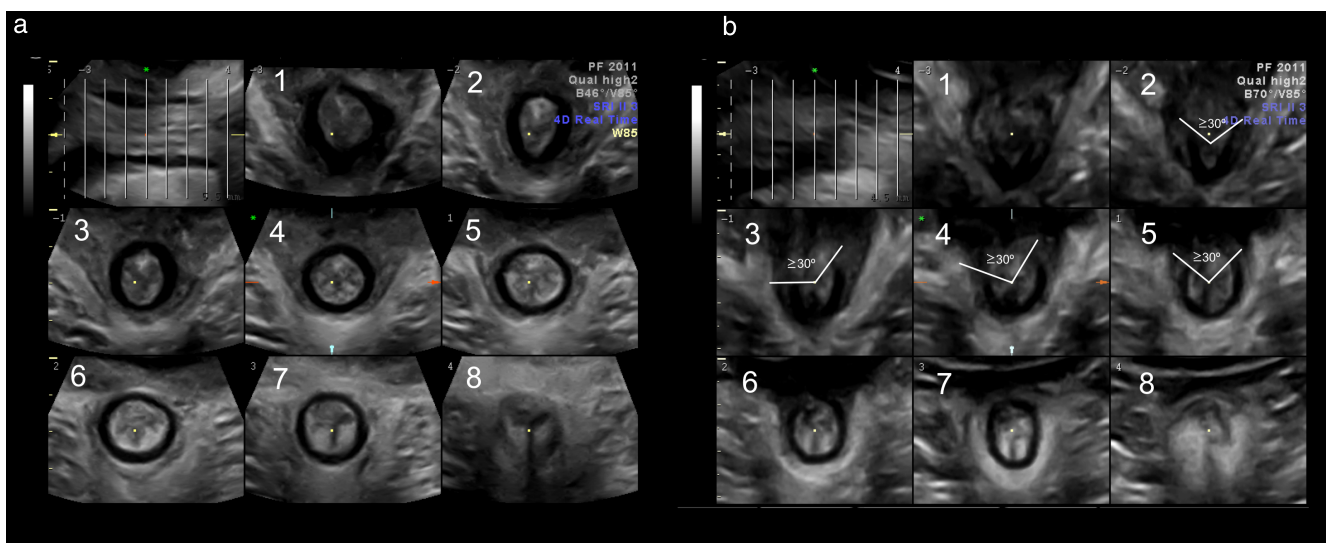
the anorectal junction) and the most distal slice at the level of the subcutaneous portion of the EAS (Figure 2a). Diagnosis of significant injury to the EAS or IAS was defined as  $\geq 30^\circ$  defect of the circumference in at least four of six slices on TUI (Figures 1b and 2b, respectively).

### Statistical analysis

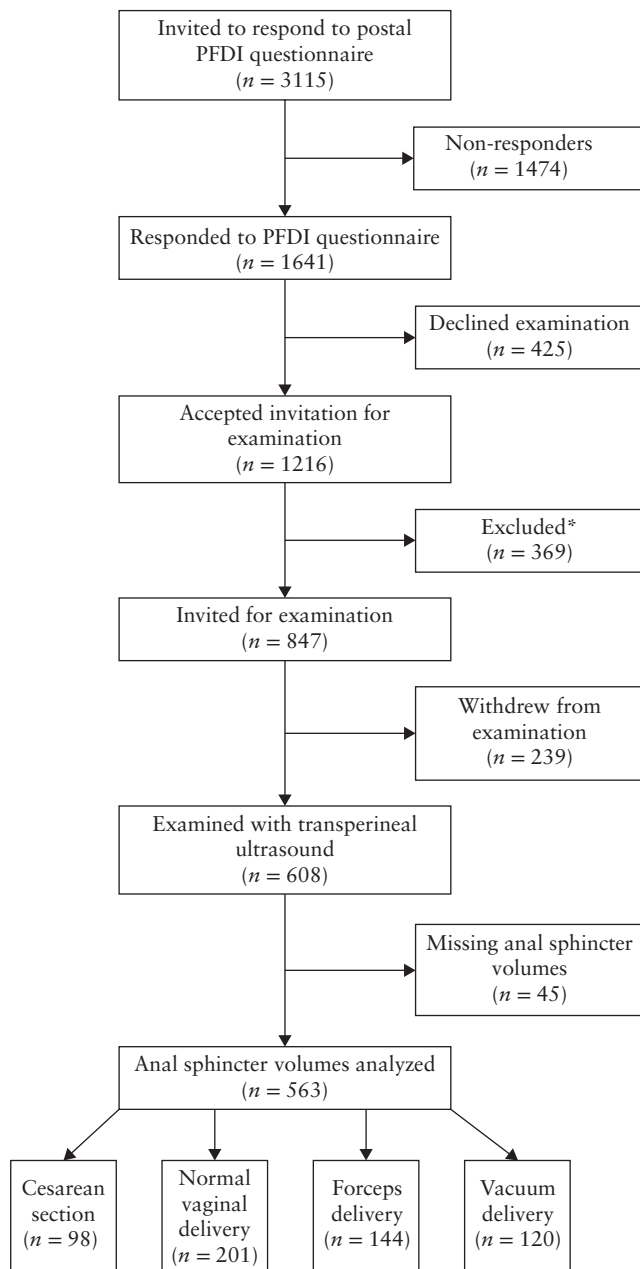
Statistical analysis was performed using IBM SPSS statistics version 23 (IBM SPSS, Armonk, NY, USA). A value of  $P < 0.05$  was considered statistically significant for all analyses. Data were approximately normally distributed, and the two-sample *t*-test was used to analyze differences between modes of delivery in demographics



**Figure 1** Tomographic ultrasound imaging in woman with intact external anal sphincter (EAS) (a) and in woman with EAS defect (b), showing midsagittal view of anal canal (top left) and set of eight slices. Upper slice (1) is at level of puborectalis muscle and lower slice (8) is caudal to internal anal sphincter at level of anal verge, with six slices (2–7) delineating entire EAS muscle. Significant defect of EAS ( $\geq 30^\circ$  of circumference) (b) involved all six slices, as indicated.



**Figure 2** Tomographic ultrasound imaging in woman with intact internal anal sphincter (IAS) (a) and in woman with IAS defect (b), showing midsagittal view of anal canal (top left) and set of eight slices. Upper slice (1) is cranial to IAS at level of anorectal junction and lower slice (8) is at level of subcutaneous portion of external anal sphincter, with six slices (2–7) delineating entire IAS. Significant defect of IAS ( $\geq 30^\circ$  of circumference) (b) involved slices 2–5, as indicated.



**Figure 3** Flowchart of study participants who first delivered between 1990 and 1997 and were examined with transperineal ultrasound 15–25 years later. \*Lived too far from Trondheim or were not available in inclusion period. PFDI, Pelvic Floor Distress Inventory.

and clinical background data. The prevalence of EAS and IAS defects visualized on ultrasound and of OASIS recorded at delivery was established. For EAS and OASIS, crude (OR) and adjusted (aOR) odds ratios with 95% CI for comparison between modes of delivery were calculated using univariable and multivariable logistic regression analysis. Infant birth weight (largest infant delivered vaginally), vaginal parity (total number of infants delivered vaginally) and indication for FD or VD (prolonged second stage of labor or fetal distress) were chosen as potential confounders on the basis of clinical experience and because these variables were different between delivery groups.

The proportion of women reporting FI was calculated for those with EAS and IAS defects seen on ultrasound and for OASIS recorded at delivery. Univariable and multivariable logistic regressions were used to calculate OR and aOR (with adjustment for age, BMI and total parity) for FI in relation to the status of the EAS on ultrasound and OASIS recorded at delivery. Few women had an IAS defect, so Fisher's exact test was used to test the association with mode of delivery and with FI.

Finally, we calculated the proportion of OASIS recorded at first or subsequent deliveries that were still visible on ultrasound, and the proportion of defects detected on ultrasound but not recorded after delivery.

## RESULTS

Of the 847 women invited, 608 (72%) attended the clinical examination. Forty-five women were excluded (ultrasound volume not stored properly ( $n=13$ ) or volume had insufficient quality for assessment of the anal sphincters ( $n=32$ )), leaving 563 women for analysis (Figure 3). The proportion of women examined was similar for all delivery groups (CS = 71%, NVD = 66%, VD = 66%, FD = 65%;  $P > 0.05$ ). A higher proportion of ultrasound volumes of poor quality occurred after FD and a lower proportion after CS compared with NVD (CS = 9%, NVD = 25%, FD = 44% and VD = 22%), and six (16%) were obtained in women for which OASIS was reported after their first delivery.

Clinical background data for the whole study population and comparison between modes of delivery are shown in Table 1. The women who were examined were

**Table 1** Background characteristics according to mode of delivery in 563 women who first delivered between 1990 and 1997

Characteristic	Total ( $n = 563$ )	NVD ( $n = 201$ )	CS ( $n = 98$ )	FD ( $n = 144$ )	VD ( $n = 120$ )	P*			
						CS vs NVD	FD vs NVD	VD vs NVD	FD vs VD
Age (years)	47.9 ± 4.9	46.7 ± 4.5	50.2 ± 4.5	48.2 ± 4.8	47.6 ± 5.1	< 0.01	< 0.01	0.08	0.34
Age at first delivery (years)	28.2 ± 4.5	26.9 ± 4.0	30.1 ± 4.6	28.6 ± 4.5	28.5 ± 4.7	< 0.01	< 0.01	< 0.01	0.86
BMI (kg/m <sup>2</sup> )	25.8 ± 4.6	24.9 ± 4.2	26.7 ± 4.5	26.0 ± 4.3	26.5 ± 5.3	< 0.01	0.02	< 0.01	0.34
Parity ( $n$ )	2.2 ± 0.8	2.5 ± 0.8	1.7 ± 0.7	2.1 ± 0.8	2.2 ± 0.8	< 0.01	< 0.01	0.01	0.33
Vaginal parity ( $n$ )	1.8 ± 1.1	2.4 ± 0.8	NA	2.0 ± 0.7	2.1 ± 0.8	NA	< 0.01	< 0.01	0.20
Birth weight (g)†	3868 ± 483	3851 ± 457	NA	3819 ± 496	3957 ± 500	NA	0.53	0.05	0.03

Data are given as mean ± SD. \*Two-group  $t$ -test. †Of largest infant delivered vaginally. BMI, body mass index; CS, Cesarean section; FD, forceps delivery; NA, not applicable; NVD, normal vaginal delivery; VD, vacuum delivery.

**Table 2** Significant external (EAS) and internal (IAS) anal sphincter defects seen on ultrasound 15–24 years after first delivery, and obstetric anal sphincter injuries (OASIS) reported at first or any subsequent delivery, according to mode of delivery

Type of anal sphincter defect/injury	NVD (n = 201)	FD (n = 144)	VD (n = 120)	FD vs NVD		VD vs NVD		FD vs VD	
				OR	P	OR	P	OR	P
EAS (n = 85)	21 (10.4)	46 (31.9)	18 (15.0)	3.6 (2.0–6.5)	< 0.01*	1.3 (0.7–2.6)	0.45*	3.0 (1.6–5.6)	< 0.01†
IAS (n = 17)	2 (1.0)	10 (6.9)	5 (4.2)	7.4 (1.5–70.5)	< 0.01‡	4.3 (0.7–45.9)	0.11‡	1.7 (0.5–6.6)	0.43‡
OASIS (n = 48)	3 (1.5)	23 (16.0)	22 (18.3)	13.3 (3.8–45.9)	< 0.01*	14.2 (4.1–49.3)	< 0.01*	0.9 (0.5–1.7)	0.75†

Data are given as *n* (%) or odds ratio (OR) (95% CI). \*Adjusted for infant birth weight and vaginal parity. †Adjusted for infant birth weight, vaginal parity and indication for forceps or vacuum. ‡Fisher's exact test. FD, forceps delivery; NVD, normal vaginal delivery; VD, vacuum delivery.

older than the background population of questionnaire responders from whom they were recruited (47.9 years *vs* 47.3 years,  $P = 0.01$ ), but parity, birth weight and BMI were similar. Indications for FD and VD were similar (prolonged second stage of labor 51% *vs* 40% ( $P = 0.07$ ) and fetal distress 57% *vs* 63% ( $P = 0.29$ )).

Defects of EAS and IAS seen on ultrasound were found in 85 (15%) and 17 (3%) women, respectively. Isolated IAS defect was found in only one woman. No ultrasound defects were visualized in women in the CS group. In total, 48 (8.5%) of 563 women had OASIS recorded at delivery. Table 2 shows the prevalence of anal sphincter defects seen on ultrasound and OASIS at delivery according to mode of delivery. ORs demonstrated increased risk of EAS and IAS defects after FD compared with NVD and VD. No statistically significant difference was found between VD and NVD for sphincter defects seen on ultrasound, in spite of a higher proportion of OASIS reported after VD than after NVD.

In total, 562 women answered the question about FI and 54 (9.6%) reported FI, of whom nine (1.6%) were incontinent to well-formed stool. This was similar to the proportion reporting FI in the background population of questionnaire responders (9.1%). The proportion was higher among the 29 women with insufficient image quality for assessment (32.0%;  $P < 0.01$ ). Defects of the EAS and IAS were significantly associated with increased risk of FI (Table 3). A tendency was found toward increased prevalence of FI in women who had OASIS recorded after delivery compared with women for whom no OASIS was reported, but this was not statistically significant.

Only 18 (21%) of 86 sphincter defects visible on ultrasound were recorded as OASIS at first or subsequent delivery. None of the defects detected on ultrasound had been recorded after NVD, compared with seven (39%) of 18 after VD and 11 (23%) of 47 after FD. Eighteen (38%) of 48 OASIS reported after delivery were still visible as sphincter defects on ultrasound (17 (35%) EAS and 11 (23%) IAS).

## DISCUSSION

This study found that anal sphincter defects visualized on transperineal ultrasound, 15–24 years after delivery, are associated with FD and development of FI. Anal sphincter defects detected after this time period that had not been

**Table 3** Prevalence of fecal incontinence in women with external (EAS) or internal (IAS) anal sphincter defect seen on ultrasound 15–24 years after first delivery and association with obstetric anal sphincter injuries (OASIS) reported at first or any subsequent delivery

Type of anal sphincter defect/injury	Fecal incontinence (n (%))	Odds ratio (95% CI)	P
Sphincter defect			
Not significant (n = 477)	38 (8.0)	—	
EAS (n = 84)	15 (17.9)	2.5 (1.3–4.9)	< 0.01*
IAS (n = 17)	5 (29.4)	4.2 (1.1–13.5)	0.02†
OASIS			
Not reported (n = 514)	46 (8.9)	—	
Reported (n = 48)	8 (16.7)	2.2 (0.96–5.0)	0.06*

One woman did not answer the question about fecal incontinence. \*Adjusted for age, body mass index and parity. †Fisher's exact test.

reported as OASIS at the time of first or subsequent delivery were common.

One strength of this study is that women from a normal population were recruited and followed up many years after delivery. A large proportion of women with FD or VD were included. Prior to 1990, few VDs and a larger proportion of FDs were performed, and doctors were therefore potentially better trained in FD than in VD at the beginning of the study period. Both procedures were performed for the same indications, but VD was permitted for all fetal head positions and for higher stations of the fetal head. Episiotomies were not recorded so adjusting for episiotomy as a potential confounder was not possible. However, mediolateral episiotomy was regarded as routine for operative vaginal delivery so rate of this procedure would have been similar for FD and VD.

A higher proportion of women were excluded because of insufficient image quality after vaginal delivery than after CS, and the prevalence of FI was higher among these women. Still, inclusion was similar for NVD, FD and VD, and the comparison of these groups should be valid. The examiner analyzing the ultrasound volumes was experienced in the methodology and was blinded to obstetric data and symptoms, ensuring an unbiased evaluation.

Endoanal ultrasound is the gold standard for examination of anal sphincters. One limitation is that this modality was not available, but we used a similar



definition of the minimal criteria necessary for diagnosing significant sphincter defects<sup>32</sup>. Furthermore, the cross-sectional study design allows us to conclude on only association between outcome measures and mode of delivery; causality cannot be established.

Despite the biases, mentioned above, against VD, we found a higher prevalence of sphincter defects after FD, which is consistent with previous findings both in observational studies and in a randomized trial<sup>14,22,33</sup>. We argue that this is caused by the device itself, and not the indication or level of training of the doctor.

The prevalence of sphincter defects was lower than in some previous studies<sup>14,22,33</sup>. The proportion detected on ultrasound depends on the definition of minimum size and length of discontinuity of the anal sphincters, and could explain different results in studies in which a standardized definition was not used<sup>32</sup>. Another explanation is that the true prevalence of OASIS depends on demographic differences in study populations and different obstetric practices between hospitals and countries<sup>9</sup>. The population of the present study constituted Caucasian women delivering at a university hospital.

Several publications have indicated that OASIS recorded at delivery is associated with FI<sup>3-5,16-18</sup>. We observed a similar tendency, although the number of women with OASIS was small and any association with incontinence did not reach significance. At transperineal ultrasound examination, women had a mean age of 48 years, and incontinence is more common in older age groups; therefore, a longer follow-up could reveal a significant association between OASIS and incontinence<sup>7,8,19</sup>.

Ultrasonographic defects of the EAS and IAS were associated significantly with FI, suggesting that transperineal ultrasound would be better for prediction of FI than would OASIS reported at delivery. In this study, EAS defect doubled the risk, and defect of both anal sphincters was associated with a four-fold increased risk of FI. This is comparable with results from studies using endoanal ultrasound<sup>4,28</sup>.

Transperineal ultrasound examination was well tolerated by the women and a 3D abdominal ultrasound probe is available in many gynecologic units. As sphincter defects seen on ultrasound correlate well with incontinence symptoms, we suggest that this is a good method for examination of the anal sphincters in gynecologic wards.

We found that 80% of sphincter defects visible on ultrasound had not been recorded previously. A similarly high proportion of undetected tears was found in a population of primiparous women examined 5 months after delivery<sup>22</sup>. This could be explained by true occult tears or, more likely, missed diagnosis at the time of delivery<sup>23</sup>.

None of the sphincter defects visible on ultrasound in the NVD group had been recorded as OASIS at delivery, compared with 40% in the VD group. This discrepancy could have been caused by a higher level of attention to OASIS after operative delivery and suggests that increased attention to OASIS is also needed after normal deliveries.

One option would be to perform ultrasound screening of women with intact perineum soon after delivery. A previous study found that ultrasound screening of women without clinically evident OASIS improved the diagnosis and that suturing decreased risk of severe FI 1 year later compared with women who were not screened<sup>34</sup>.

A total of 38% of OASIS recorded after delivery were still seen on ultrasound many years later. A similar proportion was found by Shek *et al.*<sup>27</sup>, 2 months after delivery, and in almost 60% by Valsky *et al.*<sup>28</sup> at 11 months' follow-up. This suggests that, even when OASIS is recognized and sutured, the suture technique often fails or that defects could persist as a result of healing problems. This could explain the increased risk of FI in women with OASIS reported in other studies<sup>3-5,16-18</sup>.

## Conclusion

FD, but not VD, was associated with higher prevalence of anal sphincter defects seen on ultrasound 15–24 years after delivery. Ultrasonographic sphincter defects were associated with FI. This implies that FD should be avoided and VD preferred when operative vaginal delivery is indicated. Ultrasound revealed a high proportion of sphincter defects not detected at delivery, suggesting that increased attention to diagnosis and suturing of OASIS after childbirth is needed to reduce women's risk of FI later in life. Future studies of women soon after delivery are needed to establish if transperineal ultrasound can be used as a screening method to detect OASIS undiagnosed at clinical examination. A follow-up study could determine whether differences in symptoms between women with intact anal sphincter and those with a defect increase over time.

## ACKNOWLEDGMENTS

We thank Christine Østerlie and Tuva K. Halle for help with identifying potential study participants, Berit M. Bjelkaasen and Johan M. Dreier for technical support with Questionnaires, and Guri Kolberg for help with coordination of clinical examinations.

## REFERENCES

1. Rao SS. Pathophysiology of adult fecal incontinence. *Gastroenterology* 2004; 126(1 Suppl 1): S14–22.
2. Sultan AH, Kamm MA, Hudson CN, Bartram CI. Third degree obstetric anal sphincter tears: risk factors and outcome of primary repair. *BMJ* 1994; 308: 887–891.
3. Eason E, Labrecque M, Marcoux S, Mondor M. Anal incontinence after childbirth. *CMAJ* 2002; 166: 326–330.
4. Samarasekera DN, Bekhit MT, Wright Y, Lowndes RH, Stanley KP, Preston JP, Preston P, Speakman CT. Long-term anal continence and quality of life following postpartum anal sphincter injury. *Colorectal Dis* 2008; 10: 793–799.
5. Halle TK, Salvesen KA, Volloyhaug I. Obstetric anal sphincter injury and incontinence 15–23 years after vaginal delivery. *Acta Obstet Gynecol Scand* 2016; 95: 941–947.
6. Macmillan AK, Merrie AE, Marshall RJ, Parry BR. The prevalence of fecal incontinence in community-dwelling adults: a systematic review of the literature. *Dis Colon rectum* 2004; 47: 1341–1349.
7. Wu JM, Vaughan CP, Goode PS, Redden DT, Burgio KL, Richter HE, Markland AD. Prevalence and trends of symptomatic pelvic floor disorders in U.S. women. *Obstet Gynecol* 2014; 123: 141–148.
8. Rømmen K, Schei B, Rydning A, H Sultan A, Mørkved S. Prevalence of anal incontinence among Norwegian women: a cross-sectional study. *BMJ Open* 2012; 2: pii: e001257.

9. Laine K, Gissler M, Pirhonen J. Changing incidence of anal sphincter tears in four Nordic countries through the last decades. *Eur J Obstet Gynecol Reprod Biol* 2009; **146**: 71–75.
10. Baghestan E, Irgens LM, Bordahl PE, Rasmussen S. Trends in risk factors for obstetric anal sphincter injuries in Norway. *Obstet Gynecol* 2010; **116**: 25–34.
11. Gurol-Urganci I, Cromwell DA, Edozien LC, Mahmood TA, Adams EJ, Richmond DH, Templeton A, van der Meulen JH. Third- and fourth-degree perineal tears among primiparous women in England between 2000 and 2012: time trends and risk factors. *BJOG* 2013; **120**: 1516–1525.
12. de Leeuw JW, Struijk PC, Vierhout ME, Wallenburg HC. Risk factors for third degree perineal ruptures during delivery. *BJOG* 2001; **108**: 383–387.
13. Signorello LB, Harlow BL, Chekos AK, Repke JT. Midline episiotomy and anal incontinence: retrospective cohort study. *BMJ* 2000; **320**: 86–90.
14. Sultan AH, Kamm MA, Bartram CI, Hudson CN. Anal sphincter trauma during instrumental delivery. *Int J Gynaecol Obstet* 1993; **43**: 263–270.
15. Shekhar S, Rana N, Jaswal RS. A prospective randomized study comparing maternal and fetal effects of forceps delivery and vacuum extraction. *J Obstet Gynaecol India* 2013; **63**: 116–119.
16. Sangalli MR, Floris L, Faltin D, Weil A. Anal incontinence in women with third or fourth degree perineal tears and subsequent vaginal deliveries. *Aust N Z J Obstet Gynaecol* 2000; **40**: 244–248.
17. Mous M, Muller SA, de Leeuw JW. Long-term effects of anal sphincter rupture during vaginal delivery: faecal incontinence and sexual complaints. *BJOG* 2008; **115**: 234–238.
18. Pollack J, Nordenstam J, Brismar S, Lopez A, Altman D, Zetterstrom J. Anal incontinence after vaginal delivery: a five-year prospective cohort study. *Obstet Gynecol* 2004; **104**: 1397–1402.
19. Whitehead WE, Borrud L, Goode PS, Meikle S, Mueller ER, Tuteja A, Weidner A, Weinstein M, Ye W. Fecal incontinence in US adults: epidemiology and risk factors. *Gastroenterology* 2009; **137**: 512–517, 517 e1–2.
20. Fornell EU, Matthiesen L, Sjadahl R, Berg G. Obstetric anal sphincter injury ten years after: subjective and objective long term effects. *BJOG* 2005; **112**: 312–316.
21. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Engl J Med* 1993; **329**: 1905–1911.
22. Guzman Rojas RA, Shek KL, Langer SM, Dietz HP. Prevalence of anal sphincter injury in primiparous women. *Ultrasound Obstet Gynecol* 2013; **42**: 461–466.
23. Andrews V, Sultan AH, Thakar R, Jones PW. Occult anal sphincter injuries – myth or reality? *BJOG* 2006; **113**: 195–200.
24. Abdool Z, Sultan AH, Thakar R. Ultrasound imaging of the anal sphincter complex: a review. *Br J Radiol* 2012; **85**: 865–875.
25. Peschers UM, DeLancey JO, Schaer GN, Schuessler B. Exoanal ultrasound of the anal sphincter: normal anatomy and sphincter defects. *Br J Obstet Gynaecol* 1997; **104**: 999–1003.
26. Oom DM, West RL, Schouten WR, Steensma AB. Detection of anal sphincter defects in female patients with fecal incontinence: a comparison of 3-dimensional transperineal ultrasound and 2-dimensional endoanal ultrasound. *Dis Colon rectum* 2012; **55**: 646–652.
27. Shek KL, Guzman-Rojas R, Dietz HP. Residual defects of the external anal sphincter following primary repair: an observational study using transperineal ultrasound. *Ultrasound Obstet Gynecol* 2014; **44**: 704–709.
28. Valsky DV, Cohen SM, Lipschuetz M, Hochner-Celnikier D, Yagel S. Three-dimensional transperineal ultrasound findings associated with anal incontinence after intrapartum sphincter tears in primiparous women. *Ultrasound Obstet Gynecol* 2012; **39**: 83–90.
29. Volloyhaug I, Morkved S, Salvesen O, Salvesen KA. Forceps delivery is associated with increased risk of pelvic organ prolapse and muscle trauma: a cross-sectional study 16–24 years after first delivery. *Ultrasound Obstet Gynecol* 2015; **46**: 487–495.
30. Volloyhaug I, Morkved S, Salvesen O, Salvesen K. Pelvic organ prolapse and incontinence 15–23 years after first delivery: a cross-sectional study. *BJOG* 2015; **122**: 964–971.
31. Guzman Rojas RA, Kamisan Atan I, Shek KL, Dietz HP. Anal sphincter trauma and anal incontinence in urogynecological patients. *Ultrasound Obstet Gynecol* 2015; **46**: 363–366.
32. Roos AM, Thakar R, Sultan AH. Outcome of primary repair of obstetric anal sphincter injuries (OASIS): does the grade of tear matter? *Ultrasound Obstet Gynecol* 2010; **36**: 368–374.
33. Sultan AH, Johanson RB, Carter JE. Occult anal sphincter trauma following randomized forceps and vacuum delivery. *Int J Gynaecol Obstet* 1998; **61**: 113–119.
34. Faltin DL, Boulvain M, Floris LA, Irion O. Diagnosis of anal sphincter tears to prevent fecal incontinence: a randomized controlled trial. *Obstet Gynecol* 2005; **106**: 6–13.