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PREVALENCE AND RISK FACTORS OF ANAL INCONTINENCE AT 16 WEEKS OF GESTATION.

Aims of Study
The prevalence of anal incontinence has been little assessed in the younger age groups. In community-based surveys it varies between 2.2% and 5.7% (1,2), the prevalence known to health and social service agencies was 0.04% among women aged 15-64 years (3). Even though childbirth is considered to be the commonest cause of anal incontinence only few epidemiological studies have focused on the role of pregnancy and delivery. This study aimed to evaluate the prevalence of anal incontinence at 16 weeks of gestation and to identify possible maternal and obstetric risk factors.
Population and Methods
Cross-sectional study: All women attending routine antenatal care from 1993 to 1996 were asked to complete a questionnaire at 16 weeks of gestation; a total of 7557 (93%) women answered the questions about anal incontinence. This cross-sectional study was used to describe the prevalence of anal incontinence and to identify maternal risk factors.
Cohort study: A sub-group of 1726 pregnant women from the cross-sectional study with one previous delivery at our department between 1989 and the index pregnancy. From 1989, the obstetric records of all deliveries have been prospectively registered; thus we were able in this cohort to evaluate obstetric risk factors for developing anal incontinence at 16 weeks of gestation.
Anal incontinence was defined as involuntary loss of flatus, liquid or solid stools within the last year. The questions about anal incontinence had previously bee calibrated through in-depth interviews; and repeatability also had been assessed. We restricted the analyses about maternal and obstetric risk factors to women with isolated flatus incontinence at least once a week. Bivariate associations between possible risk factors and anal incontinence were tested by the χ^2-test. Multiple logistic regression analyses were performed to identify independent maternal as well as obstetric risk factors.
Results
Prevalence: The prevalence of anal incontinence within the preceding year was 8.6%. Isolated flatus incontinence was reported in 4.2% at least once a week; isolated incontinence of liquid and solid stools in 0.2% and 0.1%, respectively. Maternal factors: After adjusting for age, parity, BMI, smoking, previous abortions, and previous lower abdominal or urological surgery in a logistic regression model we found the risk of flatus incontinence at least once a week was increased with age > 35 years (OR=1.6; 95% CI 1.1-2.4) and with previous lower abdominal or urological surgery (OR=1.5; 95% CI 1.1-2-1). Increasing parity did not increase the risk. Obstetric factors: The risk of flatus incontinence was increased after anal sphincter tear (OR=6.2; 95% CI 1.1-35.8) and birth weight > 4000 g (OR=2.3; 95% CI 1.1-4.7). Mediolateral episiotomy was insignificantly associated (OR=1.7, 95% CI 0.8-3.7); spontaneous perineal tear > 3 cm and a number of other intrapartum factors were not associated.
Conclusions
Age > 35 and previous lower abdominal or urological surgery increased the risk of flatus incontinence by contrast with increasing parity. However, analysing obstetric variables separately, birth weight > 4000 g and anal sphincter tear were significant risk factors for flatus incontinence. Episiotomy may be a possible risk factor, by contrast with spontaneous perineal lacerations > 3 cm. Our data may suggest that childbirth has less influence on the prevalence of anal incontinence than previously believed.

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PREGNANCY AND NOT DELIVERY ASSOCIATED WITH POSTPARTUM INCONTINENCE IN PRMIGRAVID WOMEN

AIMS OF STUDY Incontinence is common, its prevalence increases with age and with parity (1). Vaginal delivery is thought to be a major factor in the development of genuine stress incontinence (2). There have been very few prospective studies through pregnancy and postpartum examining the relationship between pregnancy, delivery and stress incontinence. Previous studies have demonstrated that the results of urodynamic investigations correlate poorly with urinary tract symptomatology in pregnancy and the postpartum period (3). This study was designed to examine urinary symptoms prospectively, in particular frequency, urgency and incontinence, through pregnancy and for three months postpartum

METHODS This study was a prospective observational study. Ethical approval was obtained and all women gave written consent. 264 primiparous women were recruited from the antenatal clinic at their booking appointment, all women were less than 20 weeks pregnant at the time of their first visit. Patients were interviewed, using a standard questionnaire, at booking, 28 weeks, 34-36 weeks of pregnancy and at 6 and 12 weeks postpartum. At the initial interview patients were asked about incontinence prior to pregnancy. The patients completed a frequency – volume voiding chart prior to each visit, except the first, and at each interview they performed a standing stress test. Details about the delivery were collected at the first postpartum interview. The frequency of declaration of incontinence at the visits were summed and these results used to compare those with and without incontinence postpartum ,using caparison of medians by the Mann Whitney U Test.

RESULTS To date 176 women have completed the study and are reported. Mean maternal age at delivery was 29.4 (SD = 4.60). The incidence of incontinence, both stress and urge increased in pregnancy with the greatest incidence at the 28 week visit. The incidence of incontinence declined after delivery. 16.4% of women gave a positive answer to the question 'is there any urine loss with coughing or sneezing' at the booking visit, 31.7% at 28 weeks, 30.1% at the 34-36 week visit, 10.9% at the first postpartum visit and 8.8% at the second postpartum visit. 9 (5.1%) women had stress incontinence at both of the postpartum visits, all these women were incontinent at the 28 week visit. Only two women (1% of whole sample; 10% and 15% of incontinent) at each postpartum visit described incontinence dating from delivery. Postpartum incontinence, at six and twelve weeks, was strongly associated with more numerous prepartum declarations of incontinence ($W=11 \times 10^3$, $p<0.001$; $W=12 \times 10^3$, $p<0.001$). The very low incidence of delivery-associated new incontinence precluded a meaningful vaginal/caesarean comparison.

CONCLUSION The incidence of incontinence during pregnancy is much greater than in the first 12 weeks postpartum and incontinence dating solely from delivery is very unusual. This suggests that it is pregnancy rather than delivery which plays the important role in the development of stress incontinence. It may be that pregnancy causes tissue damage or reveals an underlying tendency to stress incontinence which in most women resolves after delivery but which may then lead to incontinence in later life.

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ASSESSMENT OF PUDENDAL NERVE FUNCTION IN POSTPARTUM FECAL INCONTINENCE

Aims of study: Fecal incontinence following childbirth arises secondary to direct disruption of the anal sphincter muscles and /or traction of the pudendal nerves. Identification of neurological injury is critical for the selection of appropriate therapeutic protocols. The aim of this study was therefore to determine the role of detailed electrodiagnostic assessment of pudendal nerve function by comparing needle EMG and combined distal nerve conduction latency assessment to conventional proximal nerve conduction velocity assessment alone in women with postpartum fecal incontinence following obstetric injury.

Methods: A consecutive cohort of 33 women with a history of impaired fecal continence following primary repair of recognised obstetric anal sphincter disruption, were recruited twelve weeks postpartum. All patients were assessed by continence questionnaire, trans-anal ultrasound and electrodiagnostic assessment of pudendal nerve function. Electrodiagnostic assessment was performed using two techniques, 1) Proximal nerve conduction velocity assessment (NCV) using the St. Mark's technique to determine the pudendal nerve terminal motor latency (PNTML) and 2) combined needle EMG and peripheral NCV assessment using the Pudendo-anal reflex .

The PNTML was determined using a St. Mark's electrode. A stimulus of 50 volts for 0.1 msecs was delivered at one pulse per second, and the shortest reproducible latency recorded on an EMG machine. A PNTML of greater than 2.4 m/s was considered prolonged . Needle EMG and distal nerve conduction latency assessment was performed using a laptop EMG system. Electrodes were attached to the perineal area and a conventional concentric needle inserted into the external anal sphincter (EAS). Needle localisation was achieved by analysis of the auditory and visual appearance of the motor unit action potentials (MUAPs) observed on the oscilloscope and EMG speaker. Insertional activity was observed by asking the patient to ' bear down' as if to inhibit defecation.

Abnormal insertional activity was identified as a reduction in insertional electrical activity or the presence of positive sharp waves or fibrillation potentials. Recruitment was assessed by asking the patient to voluntarily contract the EAS. The