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Childbirth and pelvic floor dysfunction: An epidemiologic approach to the assessment of prevention opportunities at delivery

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Abstract

Female pelvic floor dysfunction is integral to the woman's role in the reproductive process, largely because of the unique anatomic features that facilitate vaginal birth and also because of the trauma that can occur during that event. Interventions such as primary elective cesarean delivery have been discussed for the primary prevention of pelvic floor dysfunction; however, existing data about potentially causal factors limit our ability to evaluate such strategies critically. Here we consider the conceptual principles of epidemiologic function and the availability of data that are necessary to make informed recommendations about prevention opportunities for pelvic floor dysfunction at delivery. Available epidemiologic data on pelvic floor dysfunction suggest that there may be substantial opportunities for the primary prevention of pelvic organ prolapse at delivery. Although definitive recommendations await further epidemiologic studies of the potential risk and benefits of obstetric practice change, it is hoped that this discussion will provide a novel, quantitative framework for the assessment of pelvic floor dysfunction prevention opportunities.

Keywords

Childbirth; Epidemiology; Pelvic floor dysfunction; Prevention

A contentious debate regarding the role of primary elective cesarean delivery is being held in the Western world. An important aspect of this debate relates to the potential benefits of cesarean delivery in the prevention of pelvic floor dysfunction (PFD). However, the development of appropriate prevention strategies has been hindered by a lack of adequate data. Hippocrates, in his treatise *The Law*, warned: "There are in fact two things, science and opinion; the first begets knowledge, the second, ignorance." Regarding PFD, there is a need for a more quantitative framework on which to base discussions that potentially will affect 4 million women who are delivered annually in the United States.¹ In discussing these issues, we consider primary elective cesarean deliveries as those that are performed for reasons other than dystocia (ie, abnormalities of the expulsive forces; abnormalities of fetal presentation,

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position, or development; abnormalities of the maternal bony pelvis; or abnormalities of soft tissues of the reproductive tract that form an obstacle to fetal descent).

Female PFD encompasses a wide variety of clinical conditions that include pelvic organ prolapse (POP), urinary incontinence (UI), and fecal incontinence (FI), among others. PFD is integral to the woman's role in the reproductive process, largely because of the unique anatomic features that are required to allow vaginal birth and because of the trauma that can occur during that event. Because of its high prevalence, deleterious effects on quality of life and its impact on the health care system, PFD is an important public health issue and should be a focus of primary prevention.

The overall model for the development of PFD in women includes factors that predispose (eg, family history of PFD), incite (eg, vaginal delivery through its effect on nerve damage, muscle damage, and tissue disruption), and promote (eg, lifestyle) dysfunction and may be important in 1 PFD type, but not another. Strategies such as primary elective cesarean delivery or avoidance of episiotomy recently have been discussed as interventions for the primary prevention of all, or some, forms of PFD. Beliefs about the role of each element in causation, based primarily on expert opinion, are supported by sparse evidence, which limits our ability to develop and assess critically the effectiveness of PFD prevention strategies from an epidemiologic standpoint.

Further complicating the development of prevention strategies is the differing disease mechanisms for each type of PFD. Changing obstetric practices may not result in the same effects on different forms of PFD. For example, some recommendations suggest the avoidance of episiotomy to decrease anal sphincter injury (and associated long-term risks of FI) but do not consider the potential effect of this decision on the development of POP. If a lack of episiotomy increases the risk of POP, a small reduction in the incidence of anal sphincter injury because of episiotomy may be outweighed by a greater increase in POP incidence later in life.

The development of prevention recommendations would benefit from an approach that considers each element of PFD in a co-ordinated framework. Comprehensive reviews of the epidemiologic condition of PFD have been conducted,² with some focusing on POP,³ UI,⁴ and FI,⁵ and some specifically examining the role of obstetric management in the prevention of PFD.⁶⁻⁹ Rather than duplicate those efforts, our purpose here was to consider epidemiologic concepts that are well-suited to the evaluation of prevention opportunities. Although important risk factors that have been identified through other research (eg, obesity, lifestyle) are not addressed here, it is hoped that this discussion will shed light on concepts that are useful for the assessment of primary prevention opportunities at delivery for PFD.

Primary prevention and the science of epidemiology

Primary prevention denotes an action taken to prevent the development of a disease in a person who is well and does not have the disease in question. We focus here on primary prevention because this strategy involves the elimination or reduction of risk factors for PFD before the dysfunction ever develops; in contrast, secondary and tertiary preventions denote the identification, through screening and early intervention, of individuals who already have a disease at an early stage in the natural history of the disease and the prevention of further sequelae of the disease through treatment.

To understand the impact of specific preventive strategies for PFD, we must be able to determine the proportion of PFD incidence in women who are delivered vaginally that actually can be attributed to vaginal delivery. Epidemiologic studies typically are used to determine incidence and risks of disease in a population. As a prelude to considering these issues, we will provide a brief review of relevant epidemiologic concepts.

The science of epidemiology is focused on the determination of whether an association observed between an exposure and an outcome reflects a causal association. Relative differences, which estimate the magnitude of these associations, are used commonly in the assessment of causality. With respect to prevention, however, measures that are based on absolute differences are often preferred, because they provide an estimate of the excess risk that is associated with a given exposure. Attributable risk measures, which are based on absolute differences, indicate the potential for prevention if the exposure could be eliminated, given that the exposure and outcome are linked causally. The basic concept of attributable risk (Figure) is that it subtracts the incidence of the outcome in the “unexposed” (eg, incidence of PFD in women who did not undergo vaginal delivery) from that in the “exposed” (eg, incidence of PFD in women who have undergone vaginal delivery) to estimate the excess risk of the outcome that can be attributed to the specific exposure. For example, if the association between vaginal delivery and a particular type of PFD is causal, the calculation of attributable risk would indicate the amount of that type of PFD in women who had vaginal deliveries that can be attributed to vaginal delivery. In other words, the attributable risk expresses the most we can hope to accomplish in reducing the risk of a particular type of PFD, if the exposure (eg, vaginal delivery) were to be eliminated completely.

Epidemiologic evaluation of PFD

Measures of disease incidence are needed to determine the proportion of disease that can be attributed to particular exposures. However, much of the existing data on PFD risk factors have been derived from cross-sectional studies that do not provide measures of disease incidence. The use of cohort studies to clarify the epidemiologic function and cause of PFD is complicated by the long duration between the occurrence of the putative risk factor (in this case, childbirth) and the clinical examination of PFD, such as POP. Although not intended as a comprehensive review, the following sections present selected data that are available to estimate the excess risk of PFD attributed to childbirth.

POP

Approximately 200,000 women undergo inpatient surgery for POP each year in the United States.¹⁰ However, because many women with PFD are treated conservatively or never evaluated, surgically treated patients do not represent the full spectrum of disease in the population. Research on POP is also complicated by the lack of a standardized clinical definition. Some population-based estimates of POP prevalence exist. A cross-sectional analysis of women 50 to 79 years old who were enrolled in the Women’s Health Initiative indicated that 41% of women with a uterus had some form of POP at baseline.¹¹ In a cross-sectional study of 487 Swedish women 20 to 59 years old, Samuelsson et al¹² reported that 31% of women overall and 44% of parous women had some form of POP, although only 2% of all women in the study had a prolapse that reached the introitus when straining. The exact threshold that justified this diagnosis was not described. However, population-based incidence of POP has not been defined clearly, mainly because there are few prospective or longitudinal studies on which to base estimates and even fewer studies that focus on groups other than women who are admitted to hospitals with symptomatic prolapse. In 1 of the only prospective cohort studies of POP with long-term follow-up, Mant et al¹³ followed 17,032 women who attended family planning clinics in England and Scotland between 1968 and 1974 for 20 years and reported an incidence of hospital admission with prolapse of 2.04 per 1000 woman-years at risk.

Of all risk factors that were examined by Mant et al,¹³ parity showed the strongest association with risk of requiring surgery for POP; compared with nulliparous women, women with 1 child were 4 times more likely and women with 2 children were 8.4 times more likely to experience POP that required hospital admission. POP was not the primary outcome of the research, and

the authors did not distinguish between parity and mode of delivery. Even so, separating out cesarean delivery would most likely increase the risk of prolapse that occurs with each birth, because it is implausible that cesarean delivery would pose a greater risk than vaginal birth. Notably, <1% of prolapse occurred in nulliparous women in the study. Samuelsson et al¹² reported that the most prominent factors of etiologic importance for POP were parity, age, and pelvic floor muscle strength, with high birth weight also associated with increased prevalence of POP among parous women. Existing data on the incidence of POP and the role of childbirth, albeit limited, suggest important opportunities for prevention. Current data on POP only permit attributable risk calculations for parity, not for specific mode of delivery. Additional prospective studies that stratify mode of delivery will be useful in the clarification of the roles of these putative risk factors for POP and, subsequently, for the development of appropriately targeted prevention strategies.

UI

The consequences of this common condition can be debilitating, both physically and psychologically. One review of the literature suggested that the prevalence of UI varies from 2% to 55%, or even more widely,¹⁴ mainly because of differences in the definition and measurement of UI, study methods, and the populations under study. The prevalence of any UI varies by age, with a pattern of an early peak in mid life followed by a steady increase among the elderly population. Available prevalence estimates are based predominantly on older, white populations; thus, information on the burden of UI among other age or racial/ethnic groups is limited.

Several cross-sectional studies have reported an increased prevalence of UI among parous women compared with nulliparous women. The extensive literature on risk factors for UI was reviewed recently by Hunskaar et al,¹⁵ who found that vaginal birth was the most important etiologic factor for the development of stress UI. Rortveit et al¹⁶ found a strong and significant association between parity and stress UI, with relative risks of 1.9 (95% CI, 1.6–2.2) for primiparous women and 2.3 (95% CI, 2.0–2.6) for women with 2 deliveries. In a separate analysis, Rortveit et al¹⁷ examined 15,307 women who were enrolled in a community-based cohort study of the epidemiologic condition of incontinence and reported odds of stress UI of 3.0 (95% CI, 2.5–3.5) for women who had vaginal deliveries compared with nulliparous women, after an adjustment for age, parity, years since last delivery, and body mass index. Based on the data reported in this study, we were able to derive unadjusted odds ratios for any UI (including stress UI, urge UI, or mixed-type incontinence) for women with 1 vaginal delivery (odds ratio [OR], 2.3) or 2 vaginal deliveries (OR, 2.8), compared with nulliparous women. A large prospective cohort study of Swedish pregnant women indicated that, 1 year after childbirth, multiparous women who had elective cesarean deliveries were at a decreased risk of stress UI (relative risk, 0.5; 95% CI, 0.3–0.9), relative to multiparous women who had vaginal deliveries.¹⁸ Notably, none of the 43 primiparous women in the study reported symptoms of stress UI 1 year after elective cesarean delivery.

FI

A sizeable proportion of adults are affected by FI. A recent review of several cross-sectional studies of various populations worldwide suggested a prevalence of 11% to 15% of FI in the community-dwelling adult population.¹⁹ Patterns of incidence are less well characterized in the literature.

Despite the high prevalence and distressing nature of FI, the mechanism by which childbirth influences this condition is not understood completely. Laceration of the external anal sphincter during vaginal delivery is a risk factor for incontinence of flatus or feces. The coexistence of unrecognized injury to the internal anal sphincter may explain the reason that up to one half

of parturients subsequently experience FI even after repair of a recognized sphincter laceration.⁸ Vacuum extraction also increases risk of FI. Associations of vaginal delivery with FI have been documented. Ryhammer et al²⁰ reported that the odds of flatus incontinence were 6.6-fold higher (95% CI, 2.4–18.3) after the third vaginal delivery, compared with the first or second vaginal delivery. Pollack et al²¹ prospectively followed 309 nulliparous women for 5 years after vaginal delivery and found that, compared with women with only 1 vaginal delivery, women who had ≥ 1 subsequent childbirths during the follow-up period were at significantly increased risk of anal incontinence (OR, 2.4; 95% CI, 1.1–5.6). In this study, most of subsequent childbirths (95%) were vaginal deliveries.

Several studies, including a recent review article,⁷ have examined outcomes that were associated with cesarean delivery versus vaginal delivery for the primary prevention of FI. With the exception of a few studies, most do not provide evidence for cesarean delivery as a preventive strategy. Moreover, the impact of delivery type on FI appears to decline with age. Thirty years after delivery, comparable prevalence of flatus incontinence and FI was found among women whose index delivery was complicated by anal sphincter disruption, women who had vaginal deliveries with episiotomy, and women who had cesarean deliveries.²² Similarly, Bollard et al²³ did not find any significant difference in reported FI symptoms between women with a history of forceps delivery (14%), unassisted vaginal deliveries (10%), or elective cesarean deliveries (0%) 34 years after delivery but acknowledged the need for a larger sample size to detect a statistically significant difference.

Use of epidemiologic data to identify prevention opportunities at delivery

On the basis of these epidemiologic data, we easily can calculate the attributable risk percentage from a cohort study using the following formulas: $[(\text{incidence in exposed} - \text{incidence in unexposed}) / \text{incidence of exposed}] \times 100$ or $[(\text{relative risk} - 1) / \text{relative risk}] \times 100$. Although risk measures are preferable, the odds ratio may be substituted for the relative risk in the latter formula. Ultimately, our intent is to examine the risk of PFD that can be attributed to vaginal delivery; however, the data required for such estimates are sparse. With the use of published data from the EPINCONT study¹⁷ to derive odds ratios for incontinence (of any type) according to number of vaginal deliveries, 56.5% of UI among women with 1 vaginal delivery (OR, 2.3; $[(2.3 - 1) / 2.3] \times 100 = 56.5\%$) and 64.3% among women with 2 vaginal deliveries (OR, 2.8; $[(2.8 - 1) / 2.8] \times 100 = 64.3\%$) can be attributed to vaginal delivery. Although Rortveit et al¹⁷ reported an attributable risk of 35% for the proportion of any incontinence among women who were delivered vaginally that would be preventable by cesarean delivery, this estimate was not stratified by the number of vaginal deliveries. The finding by Pollack et al²¹ that women with subsequent childbirths had significantly elevated odds of anal incontinence suggests that, among women with 1 vaginal delivery, 58.3% of anal incontinence (OR, 2.4; $[(2.4 - 1) / 2.4] \times 100 = 58.3\%$) can be attributed to subsequent childbirths. (Only 5% of the subsequent childbirths among women in this study were cesarean deliveries.)

Data to calculate the excess risk of POP attributable to vaginal delivery, although needed, currently are unavailable. To our knowledge, there are very few population-based data on risk factors at delivery for POP and no such published reports of studies that have been conducted in the United States that stratify risk by mode of delivery. However, the strong associations of POP with parity imply that interventions at delivery to prevent the development of POP may have greater impact than those interventions that are aimed at preventing UI or FI. Mant et al¹³ found that, compared with nulliparous women, women with 1 child had 4.0 times higher odds and women with 2 children had 8.4 times higher odds of the development of POP that required hospital admission. This indicates that 75% of POP among women with 1 child $[(4.0 - 1) / 4.0] \times 100 = 75\%$ and 88.1% of POP among women with 2 children $[(8.4 - 1) / 8.4] \times 100 = 88.1\%$ can be attributable to parity. Although not differentiated by mode of delivery, these

strongly observed associations between parity and risk of POP allude to the importance of obstetric care at delivery in the reduction of PFD in later life. However, until long-term follow-up data on the risk of POP that is associated with each mode of delivery are available, we cannot make conclusive recommendations for prevention at delivery.

Most research to date has focused on the association between mode of delivery and UI and FI, yet similar data for POP are limited. The studies that have been conducted do not distinguish between parity and mode of delivery, which is crucial to the ongoing debate regarding the role of cesarean delivery in PFD prevention. Given the annual incidence of surgical treatment for POP¹⁰ and that this type of PFD is associated most strongly with parity,¹³ this represents a fruitful area of research that may lead to development of interventions that have the most impact on PFD prevention. Long-term prospective studies that differentiate parity from route of delivery will provide the data that are necessary to quantify the excess risk of PFD that can be attributed to vaginal delivery and, hence, to the development of targeted prevention strategies.

Future directions and emphasis for research

As our understanding of specific risk factors at delivery for POP improves, more thoughtful decision-making regarding prevention may be pursued. For example, research on factors that may be associated with increased risk of the development of POP, such as instrumental vaginal delivery, will enable us to compare the impact of these factors on the various types of PFD and to develop targeted interventions. In addition, the full spectrum of cesarean delivery versus vaginal delivery warrants considerable study. For example, specific details regarding the timing of cesarean delivery (eg, early in labor vs late in second stage, number of centimeters of cervical dilation) may influence the development of PFD. These data could be used to develop interventions that are aimed at women at high risk for PFD, thus reducing the number of women that would be subjected to unintended harm through a more global approach to intervention.

As an illustration, we can consider the current debate surrounding primary elective cesarean delivery as a primary preventive strategy to preserve the integrity of the maternal pelvic anatomy. Although primary elective cesarean delivery potentially may reduce the long-term risk of PFD, other potential sequelae that are associated with this intervention must be seriously considered. Some of the complications of cesarean delivery include increased risk of infection, thromboembolic events, blood loss and necessary transfusions, postpartum pain, maternal death, longer hospital stay, recovery time, time off work, increased risk for subsequent pregnancies, and increased risk of fetal neurologic injuries and death that result from attempting vaginal birth after a cesarean delivery.

Most women in the United States are delivered vaginally, yet the lifetime risk of undergoing a single operation for POP and UI is estimated to be only 11.1%,²⁴ which suggests that the development of PFD may be attributable to factors beyond vaginal versus cesarean deliveries. An intervention such as primary elective cesarean delivery that is targeted at all births potentially could cause harm to a sizeable proportion of women who otherwise would have been delivered vaginally and not have experienced PFD.

Thus, for every 1 woman (ie, 11.1%, approximated to 1/10) who would benefit from primary elective cesarean delivery, 9 women [ie, $100\% - 11.1\% = 88.9\%$, approximated 9/10] would gain no benefit yet would have to assume the potential risks that are related to the procedure.

Although urgently needed, the data to pursue targeted interventions presently are limited. Studies similar to the Norwegian EPINCONT study,¹⁶ which enabled attributable risk calculations for UI, are required to better estimate the excess risk of the other PFDs that are attributed to vaginal delivery. In the United States, the precedent for long-term prospective studies with adequate sample size and power to identify predictors of major chronic diseases

that affect women has been set by the Women's Health Initiative²⁵ and the Nurses' Health Study.²⁶ Similarly, mechanistic studies that indicate what specific injuries occur during vaginal birth that are associated with PFD are needed.

Studies that are designed to appropriately disentangle birth-associated factors that influence the development of various types of PFD could lead to more effective prevention strategies that could benefit the approximately 4 million women who are delivered each year in the United States.¹ Careful consideration of exposure and outcome measurement will strengthen the internal validity of such studies, and inclusion of participants from a broad distribution of ages, races, and ethnicities would enhance the generalizability of the findings. Definitive recommendations will have to wait for further epidemiologic studies that will define the potential risk and benefits of various obstetric practice changes as preventive strategies for PFD.

References

1. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Munson ML. Births: final data for 2002. *Natl Vital Stat Rep* 2003;52:1–113. [PubMed: 14717305]
2. Bump RC, Norton PA. Epidemiology and natural history of pelvic floor dysfunction. *Obstet Gynecol Clin North Am* 1998;25:723–46. [PubMed: 9921553]
3. Brubaker L, Bump R, Jacquetin B, Schuessler B, Weidner A, Zimmern P, et al. Pelvic organ prolapse. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. *Incontinence*. Plymouth, UK: Plymbridge Distributors Ltd; 2002. p. 243–65.
4. Hunskaar S, Burgio K, Diokno A, Herzog AR, Hjalmas K, Lapitan MC. Epidemiology and natural history of urinary incontinence in women. *Urology* 2003;62:16–23. [PubMed: 14550833]
5. Johanson JF, Lafferty J. Epidemiology of fecal incontinence: the silent affliction. *Am J Gastroenterol* 1996;91:33–6. [PubMed: 8561140]
6. Lal M. Prevention of urinary and anal incontinence: role of elective cesarean delivery. *Curr Opin Obstet Gynecol* 2003;15:439–48. [PubMed: 14501249]
7. Lal M, Mann CH, Callender R, Radley S. Does cesarean delivery prevent anal incontinence? *Obstet Gynecol* 2003;101:305–12. [PubMed: 12576254]
8. Handa VL, Harris TA, Ostergard DR. Protecting the pelvic floor: obstetric management to prevent incontinence and pelvic organ prolapse. *Obstet Gynecol* 1996;88:470–8. [PubMed: 8752261]
9. Gregory WT, Nygaard I. Childbirth and pelvic floor disorders. *Clin Obstet Gynecol* 2004;47:394–403. [PubMed: 15166863]
10. Boyles SH, Weber AM, Meyn L. Procedures for pelvic organ prolapse in the United States, 1979–1997. *Am J Obstet Gynecol* 2003;188:108–15. [PubMed: 12548203]
11. Hendrix SL, Clark A, Nygaard I, Aragaki A, Barnabei V, McTiernan A. Pelvic organ prolapse in the Women's Health Initiative: gravity and gravidity. *Am J Obstet Gynecol* 2002;186:1160–6. [PubMed: 12066091]
12. Samuelsson EC, Arne Victor FT, Tibblin G, Svardsudd KF. Signs of genital prolapse in a Swedish population of women 20 to 59 years of age and possible related factors. *Am J Obstet Gynecol* 1999;180:299–305. [PubMed: 9988790]
13. Mant J, Painter R, Vessey M. Epidemiology of genital prolapse: observations from the Oxford Family Planning Association Study. *BJOG* 1997;104:579–85.
14. Thom D. Variation in estimates of urinary incontinence prevalence in the community: effects of differences in definition, population characteristics, and study type. *J Am Geriatr Soc* 1998;46:473–80. [PubMed: 9560071]
15. Hunskaar S, Burgio K, Diokno AC, Herzog AR, Hjalmas K, Lapitan MC. Epidemiology and natural history of urinary incontinence (UI). In: Abrams P, Cardozo L, Khoury S, Wein A, editors. *Incontinence*. Plymouth, UK: Plymbridge Distributors Ltd; 2002. p. 165–201.
16. Rortveit G, Hannestad YS, Daltveit AK, Hunskaar S. Age- and type-dependent effects of parity on urinary incontinence: the Norwegian EPINCONT study. *Obstet Gynecol* 2001;98:1004–10. [PubMed: 11755545]

17. Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S. Urinary incontinence after vaginal delivery or cesarean section. *N Engl J Med* 2003;348:900–7. [PubMed: 12621134]
18. Schytt E, Lindmark G, Waldenstrom U. Symptoms of stress incontinence 1 year after childbirth: prevalence and predictors in a national Swedish sample. *Acta Obstet Gynecol Scand* 2004;83:928–36. [PubMed: 15453888]
19. 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;47:1341–9. [PubMed: 15484348]
20. Ryhammer AM, Bek KM, Laurberg S. Multiple vaginal deliveries increase the risk of permanent incontinence of flatus urine in normal premenopausal women. *Dis Colon Rectum* 1995;38:1206–9. [PubMed: 7587765]
21. 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;104:1397–402. [PubMed: 15572505]
22. Nygaard IE, Rao SS, Dawson JD. Anal incontinence after anal sphincter disruption: a 30-year retrospective cohort study. *Obstet Gynecol* 1997;89:896–901. [PubMed: 9170461]
23. Bollard RC, Gardiner A, Duthie GS, Lindow SW. Anal sphincter injury, fecal and urinary incontinence: a 34-year follow-up after forceps delivery. *Dis Colon Rectum* 2003;46:1083–8. [PubMed: 12907903]
24. Olsen AL, Smith VJ, Bergstrom JO, Colling JC, Clark AL. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. *Obstet Gynecol* 1997;89:501–6. [PubMed: 9083302]
25. Department of Health and Human Services, National Institutes of Health. National Heart, Lung, and Blood Institute. Women's Health Initiative. Last accessed: December 15, 2005. Available at: <http://www.nhlbi.nih.gov/whi/>
26. The Nurses' Health Study. Last accessed: December 15, 2005. Available at: <http://www.channing.harvard.edu/nhs/>

Figure.

Schematic of attributable risk concept. The *hatched area* represents the attributable risk or the excess incidence of PFD in women that can be attributed to vaginal delivery, assuming causality (Adapted from Gordis L. More on risk: estimating the potential for prevention. In: Epidemiology. 2nd ed. Philadelphia: Saunders; 2000. p. 172–3.)