Third degree obstetric anal sphincter tears: risk factors and outcome of primary repair

A H Sultan, M A Kamm, C N Hudson, C I Bartram

Abstract

Objectives—To determine (i) risk factors in the development of third degree obstetric tears and (ii) the success of primary sphincter repair.

Design—(i) Retrospective analysis of obstetric variables in 50 women who had sustained a third degree tear, compared with the remaining 8553 vaginal deliveries during the same period. (ii) Women who had sustained a third degree tear and had primary sphincter repair and control subjects were interviewed and investigated with anal endosonography, anal manometry, and pudendal nerve terminal motor latency measurements.

Setting—Antenatal clinic in teaching hospital in inner London.

Subjects—(i) All women (n=8603) who delivered vaginally over a 31 month period. (ii) 34 women who sustained a third degree tear and 88 matched controls.

Main outcome measures—Obstetric risk factors, defaecatory symptoms, sonographic sphincter defects, and pudendal nerve damage.

Results—(i) Factors significantly associated with development of a third degree tear were: forceps delivery (50% v 7% in controls; P=0.00001), primiparous delivery (85% v 43%; P=0.00001), birth weight >4 kg (P=0.00002), and occipitoposterior position at delivery (P=0.003). No third degree tear occurred during 351 vacuum extractions. Eleven of 25 (44%) women who were delivered without instruments and had a third degree tear did so despite a posterolateral episiotomy. (ii) Anal incontinence or faecal urgency was present in 16 women with tears and 11 controls (47% v 13%; P=0.00001). Sonographic sphincter defects were identified in 29 with tears and 29 controls (85% v 33%; P=0.00001). Every symptomatic patient had persistent combined internal and external sphincter defects, and these were associated with significantly lower anal pressures. Pudendal nerve terminal motor latency measurements were not significantly different.

Conclusions—Vacuum extraction is associated with fewer third degree tears than forceps delivery. An episiotomy does not always prevent a third degree tear. Primary repair is inadequate in most women who sustain third degree tears, most having residual sphincter defects and about half experiencing anal incontinence, which is caused by persistent mechanical sphincter disruption rather than pudendal nerve damage. Attention should be directed towards preventive obstetric practice and surgical techniques of repair.

Introduction

A tear involving the anal sphincter during vaginal delivery has great bearing on a woman's future continence. Primary sphincter repair, performed by obstetricians immediately after delivery, has traditionally been regarded as providing a good outcome.¹⁻⁵ However, recent studies in a total of 70 patients have reported subsequent anal incontinence

in 29-48% of women three months to three years after primary sphincter repair.⁶⁴

This study aimed to determine the risk factors associated with the development of third degree tears and the success of primary sphincter repair with respect to defaecatory symptoms and anal sphincter function. All women who had experienced a third degree tear over a 31 month period in one obstetric unit of a teaching hospital were included in this study.

Methods

A tear was classified as third degree if the anal sphincter was torn, with or without a breach of the anal epithelium.

RISK FACTORS

In a 31 month period between 1989 and 1992 there were 8603 vaginal deliveries, during which 50 (0.6%)women sustained a third degree tear (as documented in the labour ward delivery book and computer records). All 8603 deliveries were analysed retrospectively with respect to parity, induction of labour, use of epidural analgesia, fetal presentation and position, instrumental delivery, shoulder dystocia, and birth weight.

OUTCOME OF PRIMARY SPHINCTER REPAIR

Thirty four of the 50 women who had sustained a third degree tear agreed to be interviewed and investigated. Two of the remaining 16 women were pregnant at the time of the study and declined participation; 14 women could not be traced. Obstetric factors in these 16 women were similar to those of the 34 women who participated in the study. The 34 women comprised 30 primiparas and four multiparas who had each had two previous vaginal deliveries. Eighteen women were white, 14 were black, and two were of Asian origin; they had a mean age of 26 years (range 18-37 years).

The women were assessed at a median 49 days (range 42-651 days) after delivery. Six women who were investigated less than two months after delivery were re-examined six months after delivery. Each woman was interviewed by one investigator (AHS) and a questionnaire was completed. The frequency of bowel motions, the presence of straining of more than a quarter of the time at stool, faecal urgency (inability to defer a bowel action for more than five minutes), and incontinence to flatus, liquids, or solids were recorded. The type of anaesthesia and suture material used for the repair and the use of postoperative antibiotics, were also noted.

Seventy seven consecutive consenting primiparous women and 11 multiparas who had had two previous vaginal deliveries formed the control group. None of these women had sustained a third degree tear. Women in the study group and control group were matched for parity, age, and ethnic origin and were not significantly different with respect to age or time from delivery to assessment (two sample t test or χ^2 test). They were studied at a median 49 days (range 36-630 days) after their first vaginal delivery.

St Bartholomew's (Homerton) Hospital, London E9 6SR A H Sultan, research fellow C N Hudson, professor of obstetrics and gynaecology

St Mark's Hospital,

London ECIV 2PS M A Kamm, director, medical physiology unit C I Bartram, consultant radiologist

Correspondence to: Mr A H Sultan, Department of Obstetrics and Gynaecology, Whipps Cross Hospital, London E11 1NR.

BMJ 1994;308:887-91

INVESTIGATIONS Anal manometry

Anal manometry was carried out with an air filled microballoon system (Stryker 295-1, Kalamazoo, Michigan) according to previously described methods.⁹ The manometric anal length, the maximum resting pressure (a reflection of predominantly internal anal sphincter function),¹⁰ and the maximum voluntary squeeze pressure (increment above resting pressure, a reflection of external anal sphincter function)¹⁰ were measured.

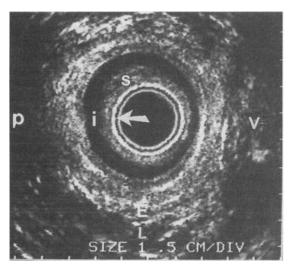
Pudendal nerve terminal motor latency

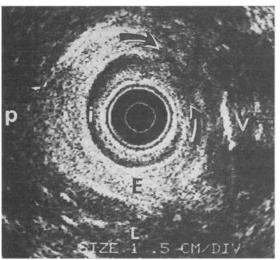
The latency between pudendal nerve stimulation at the ischial spines and contraction of the external anal sphincter was measured on both sides with the St Mark's pudendal electrode¹¹ (Dantec Electronics, Bristol) according to previously described methods.¹²

Anal endosonography

To image the internal and external anal sphincters, anal endosonography was carried out with the Bruel and Kjaer (Naerum, Denmark) type 1850 rotating endoprobe.¹³¹⁴ The 7 MHz transducer (focal range 2-4-5 cm) was covered with a hard sonolucent plastic cone with an outside diameter of 17 mm. Serial radial images of the anal canal were obtained and recorded on to video tape.

The internal anal sphincter appears as a well defined homogeneous hypoechoic ring (fig 1)¹³⁻¹⁵; defects of the internal sphincter are clearly seen as a disruption in this ring (fig 2).^{15 16} The external anal sphincter is lateral to the internal sphincter and has a heterogeneous hyperechoic appearance.^{14 15} An external sphincter defect is recognised as an amorphous, usually hypo-





echoic, break in the continuity of the normal sonographic texture of the muscle^{15 17}; it usually appears hypoechoic but can be of mixed echogenicity (fig 2).

All investigations were performed by one operator (AHS) and the stored images independently reported by a consultant radiologist (CIB) who was unaware of the women's obstetric history or symptoms.

ETHICAL APPROVAL

This study was approved by the City and Hackney District Research Ethics Committee. All subjects gave written informed consent.

STATISTICAL ANALYSIS

Data were analysed with Confidence Interval Analysis (British Medical Association) and Minitab Statistical Software (University of Pennsylvania). Continuous variables in the study and control group were compared by using the two sample t test. Categorical data were compared by using Fisher's exact test. Relative risk estimates and 95% confidence intervals of differences are presented where appropriate.

Results

RISK FACTORS FOR THIRD DEGREE TEAR

All 50 women with a third degree tear had delivered beyond 36 weeks' gestation and all had had a cephalic presentation. None of the 95 vaginal breech deliveries during the same period had sustained a third degree tear. Forceps delivery (relative risk, 13.3), primiparity (7), birth weight >4 kg (2.9), and occipitoposterior position at delivery (4.4) were all significantly more common in women who sustained a third degree tear than in those women who did not (table I).

It is possible to sustain a third degree tear without any of the mentioned risk factors. Of the 50 women who sustained a third degree tear, three had none of the risk factors, 17 had one risk factor, 24 had two risk factors, and six had three risk factors: 94% of women with a third degree tear had at least one risk factor. However, it may still not be possible to predict who will sustain a tear, as third degree tears occur in less than 1% of all vaginal deliveries.

Although 36 of the 50 (72%) women who developed a third degree tear had had a posterolateral episiotomy, most of these were associated with forceps delivery. Delivery was achieved with forceps (Simpson's, 23; Kielland's, two) in 25 women. Sixteen of these women were delivered by a registrar and nine by a senior house officer under supervision. All women had had a posterolateral episiotomy before forceps delivery. The anorectal mucosa was affected more frequently as a result of forceps delivery (12/25), than in noninstrumental delivery (8/25), but this difference was not statistically significant. No third degree tear occurred during 351 vacuum extractions (4% of all vaginal deliveries).

Sixteen of the 25 women in the non-instrumental delivery group were delivered by qualified midwives and nine by student midwives under supervision. In 11 of these 25 (44%) women an episiotomy had been performed, and the remaining 14 sustained a spontaneous third degree tear; the obstetric risk factors did not differ in frequency between these two groups.

OUTCOME OF PRIMARY SPHINCTER REPAIR Details of sphincter repair

The primary sphincter repair was performed by a registrar or senior registrar in all cases. Twenty two of the 34 women had a sphincter repair under regional (spinal, epidural, or caudal) or general anaesthesia. The remaining 12 were repaired under local anaesthesia (pudendal block).

FIG 1—Cross sectional image of the mid anal canal in a 25 year old nulliparous woman. P=posterior; L = left; V = vagina; the bright ring (arrow) represents the reflections off the cone; s=submucosa; i=internal anal sphincter; E=external anal sphincter. Both sphincter rings are normal

FIG 2—Image of the mid anal canal (same orientation as fig 1) from a 26 year old primiparous woman six months after a primary sphincter repair for a third degree tear. Arrows indicate an external sphincter defect. The hypoechoic internal anal sphincter (i) has also been damaged and is incomplete anteriorly. This is the typical site of obstetric sphincter damage TABLE I-Relation between obstetric factors and third degree tears in 8603 deliveries

Variable	No (%) of women with third degree tears (n=50)	P value (Fisher's exact test)	Relative risk estimates (95% confidence interval)	
Delivery:				
Forceps (n=600)	25 (4.2)		13·3 (7·7 to 23)	
Non-forceps (n=8003)	25 (0.3)	0.00001*		
Parity:	()			
Primipara (n=3698)	42 (1.1)		7·0 (3·3 to 14·8)	
Multipara (n=4905)	8 (0.2)	0.00001*		
Baby's weight:	/			
$\geq 4 \text{ kg} (n=681)$	11 (1.6)]		2·9 (1·5 to 5·8)	
< 4 kg (n = 7922)	39 (0-5)	0.00002*		
Persistent occipitoposterior position:				
Yes (n=170)	5 (2.9)			
No (n=8443)	45 (0.5)	0.003*	4·4 (1·6 to 12·2)	
Labour:				
Induced (n=1157)	11 (1.0)]		1.8 (0.9 to 3.5)	
Spontaneous (n=7446)	39 (0.5)	0.07		
Shoulder dystocia:				
Yes (n=162)	2 (1.2)			
No (n=8441)	48 (0.6)	0.24	2·2 (0·5 to 8·6)	
Epidural analgesia:	\> ->			
Yes (n=1871)	11 (0.6)]			
No (n=6732)	39 (0.6)	1.00	1.0 (0.5 to 2.0)	

*Denotes statistically significant difference between those who sustained a third degree tear and those that did not (Fisher's exact test).

TABLE II—Mean (SD) measurements on anal manometry and pudential nerve terminal motor latency in control women having a vaginal delivery without a third degree tear and women who sustained a third degree tear

	Controls (n=88)	Women with third degree tears (n=34)	P value	95% Confidence interval of difference
Anal manometry:				
Anal length (cm)	3.8 (0.5)	3.3 (0.5)	0.0001*	0·2 to 0·6
Resting pressure (mm Hg)	58 (13)	44 (15)	0.0001*	8 to 20
Squeeze pressure (mm Hg)	63 (35)	34 (15)	0.0001*	20 to 38
Pudendal nerve terminal motor latency* (ms):				
Right	2 (0.2)	1.9 ([0.2)	0.17	0 to 0-2
Left	2.1 (0.2)	2 (0.2)	0.06	0 to 0-2

*Measured in 79 controls and 31 women with third degree tears.

Repair usually consisted of inserting two or three "figure of eight" sutures to approximate the torn ends of the sphincter. Chromic catgut was used in 23 women and polyglycolic acid (Vicryl) or polyglactin sutures (Dexon) in 11 women.

Repair of torn anal epithelium was done separately by means of interrupted sutures, with the knots in the anal canal. All women who sustained a third degree tear affecting the anal epithelium were prescribed a one week course of a broad spectrum antibiotic after repair. A stool softener (lactulose) was also prescribed for 7-14 days.

Wound infection requiring antibiotics occurred in six women, three of whom had already taken a course of prophylactic antibiotics. Two of these women developed fistulas (one anovaginal and one rectovaginal).

There was no significant association between the use of antibiotics, occurrence of wound infection, form of anaesthesia for repair, or the type of suture material used and the outcome in terms of the later development of symptoms, anal manometry measurements, or the development of sphincter defects.

Defaecatory symptoms

Sixteen (47%) women with a repaired third degree tear had defaecatory symptoms at the time of examination: 14 (41%) anal incontinence (11 to flatus only and three to flatus and liquid) and nine (26%) faecal urgency (seven of these nine women also suffered from anal incontinence). One of these women with incontinence also had a rectovaginal fistula, and another had an anovaginal fistula. A further three women had had temporary symptoms lasting for a few weeks after delivery.

Among the controls 11 (13%) women had defaecatory symptoms: anal incontinence in five (flatus, three; flatus and liquid stool, two; faecal urgency, eight, of whom two also had anal incontinence).

Anal endosonography

Sonography showed sphincter defects in 29 women (85%) with a third degree tear (one affecting the internal sphincter alone, five affecting the external sphincter, and 23 affecting both anal sphincter muscles). All 19 women with symptoms, in addition to the three with temporary symptoms, had combined internal sphincter and external sphincter defects. Incontinence was significantly associated with internal sphincter defects (P < 0.01) and external sphincter defects (P < 0.025).

Twenty nine (33%) of the 88 controls were found to have sphincter defects (14 internal sphincter alone, five external sphincter alone, and 10 both).

In the women who had experienced a third degree tear the sphincter defects were usually along the full length of the sphincter; in the control women the defect usually involved only a part of the sphincter length.

Anal manometry

In comparison with the control group, on anal manometry the women who had had a third degree tear had a significantly lower maximum resting pressure, maximum squeeze pressure, and a shorter anal canal length (table II).

The maximum resting pressure was significantly lower in the 14 women with faecal incontinence than in the 20 who were continent (mean 35 (SD 10) v50 (15) mm Hg, P=0.002; 95% confidence interval of difference 6 to 24). No significant difference was observed in relation to the maximum squeeze pressure.

The 24 women with an internal sphincter defect had a lower maximum resting pressure than the 10 in whom the internal sphincter was intact (40 (12) v 53 (18), P=0.05; 0.3 to 27). Anal pressures were not significantly related to the presence of an external sphincter defect.

Pudendal nerve terminal motor latency

Pudendal nerve terminal motor latency was measured in 31 of the 34 women who sustained a third degree tear and 79 of the control group. No significant differences were found between the groups (table II).

Six month follow up

The six women who were studied less than two months after their delivery all had residual sphincter defects. These were unchanged when the women were scanned again six months after delivery.

Discussion

Third degree tears are an uncommon complication of childbirth, occurring in 0.6% of vaginal deliveries in this study, a similar incidence to that reported previously.⁶⁷ Although these tears are uncommon, we have shown that primary sphincter repair in these women is often unsatisfactory and associated with morbidity.

RISK FACTORS FOR THIRD DEGREE TEARS

In keeping with other studies,^{67,18,19} we found that nulliparous women were at greater risk of sustaining a third degree tear than women who had already had a vaginal delivery. This probably relates to relative inelasticity of the perineum.^{20,21} If other risk factors are also present the attending obstetrician should anticipate the possibility of a major tear.

Half the women who sustained a third degree tear were delivered by forceps, although this complication occurred in only 4% of all forceps deliveries. In contrast, during the same period no third degree tear occurred with a vacuum extraction. We have shown by anal endosonography that 80% of primiparous women delivered by forceps develop subclinical sphincter defects.²² In that prospective study no defects were identified after a vacuum extraction. In another study of 43 women who had an instrumental delivery we found that 81% of forceps deliveries were associated with sonographic anal sphincter damage compared with 24% of vacuum deliveries.23 Johanson et al in their randomised study of 600 women also found a significantly higher incidence of maternal injuries after forceps delivery than vacuum delivery.24 The use of forceps therefore seems to be a major determinant of sphincter damage and supports the opinion that the vacuum extractor should be the instrument of choice.25

Forty two percent of the women who sustained a third degree tear without an instrumental delivery did so despite a posterolateral episiotomy. Other studies have also questioned the benefits of an episiotomy,^{18 19 26-30} although factors such as the timing and extent of episiotomy have not been evaluated.

OUTCOME OF PRIMARY SPHINCTER REPAIR

Third degree tears have not been regarded as a major complication of childbirth.¹⁵ We have shown, however, that about half the women with such a tear continue to experience some impairment of anal continence, despite a primary sphincter repair. The cause of anal incontinence is persistent mechanical sphincter disruption rather than pudendal nerve damage.

A poor functional result from primary repair may relate to failure of identification of the components of the sphincter and hence incomplete union along the full length of the sphincter. The shorter anal canal in women who had had a sphincter repair would support this explanation. Alternatively the inherent tone in the sphincter mucles may cause the approximated torn ends of the muscle to retract. Technical differences in surgical technique may also be important; it has not been determined whether the most effective repair involves simple approximation³¹ or overlap of the muscle ends,³² nor whether separate repair of the internal anal sphincter should be undertaken. In addition, some have attempted to unite the puborectalis muscle at the apex of the perineal body.^{33 4}

No study has ascertained whether outcome could be improved if primary repair were undertaken by an experienced obstetrician or surgeon experienced in sphincter surgery, or if the repair was delayed. These factors and other aspects of postoperative management need to be studied prospectively.

In the present study all the women with impaired continence had sonographic defects in both sphincter muscles, an appearance which has been previously validated to accurately reflect the presence of defects.^{16 17} Functional sphincter impairment, as shown by significantly lower anal pressures, was also evident. The pudendal nerve motor latencies were normal in most of these women, confirming that incontinence in these women is related to mechanical disruption rather than nerve damage.

Sonographic defects were identified in some asymptomatic women with a third degree tear and also some women in the control group. The occurrence of occult sphincter damage in about a third of women having their first vaginal delivery has been documented in a prospective study.²² In women without a third degree tear such lesions could be due to extrinsic blunt trauma during crowning of the fetus's head or to an unrecognised extension of a second degree tear or episiotomy. Long term studies are required to determine if these asymptomatic women with sphincter defects are more likely to develop late faecal incontinence, although the almost universal finding of

Clinical implications

• Third degree obstetric tears are an uncommon but serious complication of vaginal delivery

• Forceps delivery, first vaginal delivery, a large baby (>4 kg), and persistent fetal occipitoposterior position are the main risk factors

• Almost half the affected women have persistent defaecatory symptoms despite a primary sphincter repair

• The cause of anal incontinence is persistent anatomical sphincter disruption rather than pudendal nerve damage

sphincter defects in women presenting later in life with faecal incontinence³⁹ would suggest that this is the case.

Although 47% of women with a third degree tear said they had defaecatory symptoms, none had sought medical attention. This highlights the need to ask women directly about such symptoms at their postnatal visit. Even temporary anal incontinence after a third degree tear, which occurred in three women in the present study, has been shown to be a predictive factor for anal incontinence after subsequent vaginal delivery.³⁶

The ideal management in subsequent deliveries of women who have sustained a third degree tear has not been prospectively established. However, we believe that these women should be assessed by anal endosonographic and physiological tests before delivery. Any woman who has symptoms or major sphincter defects should be offered a caesarean section.³⁷ In the presence of minor defects, a potentially traumatic vaginal delivery should be avoided.

CONCLUSION

In summary, third degree tears are an uncommon but serious complication of vaginal delivery. When multiple risk factors are present, special attention should be directed to preventing tears. Primary sphincter repair seems to be inadequate in at least half the women, often resulting in persistent symptoms. Because incontinence can be such a devastating social disability, the nature of sphincter repair deserves serious further attention.

We are grateful to the obstetric consultants of St Bartholomew's (Homerton) Hospital for allowing us to study their patients and to Janice Thomas, medical statistician, St Bartholomew's Hospital, for her advice.

AHS was supported by the Joint Research Board and the Clinical Directorate of Obstetrics and Gynaecology, St Bartholomew's Hospital, and MAK by the St Mark's Research Foundation.

This paper was read at the British Congress of Obstetrics and Gynaecology (Manchester) in July 1992 and the British Society of Gastroenterology (Warwick) in September 1992. An abstract has been published in *Gut* 1992;33:S29.

- 1 Ingraham HA, Gardner MM, Heus GE. A report on 159 third degree tears. Am J Obstet Gynecol 1949;57:730-5.
- 2 Flemming AR. Complete perineotomy. Obstet Gynecol 1960;16:172-4.
- Barter RH, Parks J, Tyndal C. Median episiotomies and complete perineal lacerations. Am J Obstet Gynecol 1960;80:654-62.
 Sieber EH, Kroon JD. Morbidity in the third degree laceration. Obstet Gynecol
- 1962;19:677-80.
 5 O'Leary JL, O'Leary JA. The complete episiotomy. Analysis of 1224 complete lacerations, sphincterotomies, and episiproctotomies. Obstet Gynecol 1965;25:235-40.
- 6 Haadem K, Dahlstrom JA, Lennart L. Anal sphincter competence in healthy women: clinical implications of age and other factors. Obstet Gynecol 1991;78:823-7.
- 7 Sorensen SM, Bondesen H, Istre O, Vilmann P. Perineal rupture following vaginal delivery. Acta Obstet Gynecol Scand 1988;67:315-8.
- 8 Neilsen MB, Hauge C, Rasmussen OO, Pedersen JF, Christiansen J. Anal endosonographic findings in the follow-up of primarily sutured sphincteric ruptures. Br J Surg 1992;79:104-6.

- 9 Orrom WJ, Williams JG, Rothenberger DA, Wong WD. Portable anorectal manometry. Br J Surg 1990;77:876-7.
- 10 Frenckner B, Euler CV. Influence of pudendal block on the function of the anal sphincters. Gut 1975;16:482-9. 11 Rogers J, Henry MM, Misiewicz JJ. Disposble pudendal nerve stimulator:
- evaluation of the standard instrument and new device. Gut 1988;29:1131-3.
- 12 Swash M, Snooks SJ. Motor nerve conduction studies of the pelvic floor innervation. In: Henry MM, Swash M, eds. Coloprotology and the pelvic floor. London: Butterworth-Heinemann, 1992:196-206.
- 13 Law PJ, Bartram CI. Anal endosonography: technique and normal anatomy. Gastrointest Radiol 1989;14:349-53 14 Sultan AH, Nicholls RJ, Kamm MA, Hudson CN, Beynon J, Bartram CI.
- Anal endosonography and correlation with in vitro and in vivo anatomy. Br J Surg 1933;80:508-11.
- 15 Sultan AH, Kamm MA. Ultrasound of the anal sphincter. In: Schuster MM, ed. Atlas of gastrointestinal motility in health and disease. Baltimore: Williams and Wilkins, 1993:115-21.
- 16 Sultan AH, Kamm MA, Nicholls RJ, Bartram CI. Prospective study of the extent of internal sphincter division during lateral sphincterotomy. Dis Colon Rectum (in press).
- 17 Sultan AH, Kamm MA, Talbot IC, Nicholls RJ, Bartram CI. Anal endosonography: precision of identifying sphincter defects confirmed histologically. Br 7 Surg (in press). 18 Gass MS, Dunn C, Stys SJ. Effect of episiotomy on the frequency of vaginal
- outlet lacerations. 7 Reprod Med 1986;31:240-4. 19 Green JR, Soohoo SL. Factors associated with rectal injury in spontaneous
- deliveries. Obstet Gynecol 1989:73:732-8. 20 Combs CA, Robertson PA, Laros PK Jr. Risk factors for third-degree perineal
- lacerations in forceps and vacuum deliveries. Am J Obstet Gynecol 1990;163: 100-4
- 21 Fischer SR. Factors associated with the occurrence of perineal lacerations. 3 Nurse Midwifery 1979;24:18-26. 22 Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. A prospective
- study of anal sphincter disruption during vaginal delivery. N Engl J Med 1993:329:1905-11
- 23 Sultan AH, Kamm MA, Bartram CI, Hudson CN. Anal sphincter trauma during instrumental delivery. A comparison between forceps and vacuum extraction. Int J Gymaecol Obstet 1993;43:263-70.

- 24 Johanson RB, Rice C, Doyle M, Arthur M, Anyanwu L, Ibrahim J, et al. A randomised prospective study comparing the new vacuum extractor policy with forceps delivery. Br J Obstet Gynaecol 1993;100:524-30.
- 25 Chalmers IA. Chalmers I. The obstetric vacuum extractor is the instrument of Gynaecol 1989;96:505-6.
- 26 Thacker SB, Banta DH. Benefits and risks of episiotomy: an interpre review of the English language literature. 1860-1980. Obstet Gynecol Surv 1983;38:322-38
- 27 Sleep J, Grant A, Garcia J, Elbourne D, Spencer J, Chalmers J, West Berkshire perineal management trial. BMJ 1984;289:587-90. 28 Thorp JM Jr, Bowes WA Jr. Episiotomy: can its routine use be defended?
- Am J Obstet Gynecol 1989;160:1027-30. 29 Larsson P-G, Platz-Christensen J-J, Bergman B, Wallstersson G. Advantage
- or disadvantage of episiotomy or disadvantage of episiotomy compared with spontaneous perineal lacera-tion. Gynecol Obstet Invest 1991;31:213-6. 30 Henriksen TB, Bek KM, Hedegaard M, Secher NJ. Episiotomy and perineal
- lesions in spontaneous vaginal deliveries. Br 7 Obstet Gynaecol 1992;99: 950-4 31 Blaisdell PC. Repair of the incontinent sphincter ani. Surg Gynecol Obstet
- 1940;70:692-7 32 Browning GGP, Motson RW. Results of Parks operation for faecal incontinence
- after anal sphincter injury. BMJ 1983;286:1873-5. Corman ML. Anal sphincter reconstruction. Surg Clin N Am 1980;60:457-63.
- 34 Pezim MF, Spencer RJ, Stanhope CR, Beart RW, Ready RL, Ilstrup DM. Sphincter repair for fecal incontinence after obstetrical and iactrogenic injury. Dis Colon Rectum 1987;30:521-5.
- 35 Burnett SJD, Spence-Jones C, Speakman CTM, Kamm MA, Hudson CN, Bartram CI. Unsuspected sphincter damage following childbirth revealed by anal endosonography. Br J Radiol 1991;64:225-7.
- 36 Bek KM, Laurberg S. Risks of anal incontinence from subsequent vaginal delivery after a complete obstetric anal sphincter tear. Br J Obstet Gynaecol 1992:99:724-6.
- 37 Sultan AH, Kamm MA, Bartram CI, Hudson CN. Perineal damage at delivery. Contemp Rev Obstet Gynaecol 1994;6(1):18-24.

(Accepted 30 November 1993)

Deep venous thrombosis and occult malignancy: an epidemiological study

M Nordström, B Lindblad, H Anderson, D Bergqvist, T Kjellström

Abstract

Objective-To determine the risk of subsequent cancer in patients with deep venous thrombosis confirmed by venography.

Design-Follow up of all patients who had venography for suspected deep venous thrombosis during 1984-88. Patients were traced through a cancer registry up to 1 January 1991.

Subjects—4399 patients who had phlebography in one hospital.

Setting-General hospital in Malmö, Sweden, serving a population of 230 000.

Main outcome measure-Number of cancers recorded.

Results-4399 patients had venography for suspected deep venous thrombosis; 604 were known to have a malignancy at the time of venography and were excluded from further analysis. 1383 had deep venous thrombosis, 150 of whom subsequently developed cancer. 182 of the 2412 patients without thrombosis developed cancer.

During the first six months after venography 66 patients with thrombosis developed malignancy compared with 37 patients without thrombosis (P < 0.0001). 38 of the cancers in the deep venous thrombosis group were detected by history, physical examination, and laboratory tests. Three patients had postoperative or post-traumatic deep venous thromboses. Only two of the remaining patients would have benefited from early detection by extensive screening. After six months the incidence of cancer was identical in patients with and without thrombosis.

Conclusion-Deep venous thrombosis is associated with a significantly higher frequency of malignancy during the first six months after diagnosis. Malignancies can be found with simple clinical and

diagnostic methods and extensive screening is not required.

Introduction

Although a large number of studies have investigated venous thromboembolism, information about its epidemiology is scarce. A prospective study of 366 patients in Malmö, Sweden, who had treatment after positive results on venography reported an overall incidence of deep venous thrombosis of 159 per 100 000 inhabitants per year.¹ At the time of diagnosis of deep venous thrombosis 71 patients (19%) had a known cancer and a further 19 (5%) developed cancer within the following year. Eight of the cancers were obvious at the time of diagnosis of the deep venous thrombosis and 11 were occult. In 1865 Trousseau described an association between deep venous thrombosis and malignancy,² but the relation remains controversial. It is generally accepted that cancer can cause deep venous thrombosis by compressing the veins as well as prothrombotic haematological changes. It is still unclear, however, whether deep venous thrombosis that is not associated with any obvious risk factor, so-called kryptogenic thrombosis, may be an early sign of occult cancer.

Only a few studies have looked at the frequency of occult malignancy in patients with deep venous thrombosis. We analysed the relation between deep venous thrombosis and subsequent malignancy in all patients who had venography for suspected deep venous thrombosis during 1984 to 1988 in Malmö. We wanted to know if patients with deep venous thrombosis had a higher risk of cancer and if so to determine the time elapsing between venography and diagnosis of cancer. We also studied the diagnostic methods used to detect the cancers.

Department of Medicine and Surgery, Malmö General Hospital, University of Lund. S-214 01 Malmö, Sweden M Nordström, fellow B Lindblad, associate professor

Department of Surgery, Academic Hospital, Uppsala University, Uppsala, Sweden D Bergqvist, professor

Regional Tumour Registry, University Hospital, Lund, Sweden H Anderson, biostatistician

Department of Medicine, Helsingborg Hospital, Sweden

T Kiellström, associate professor

Correspondence to: Dr Nordström.

BM71994:308:891-4