

COMMENTARY

Routine induction of labour at 41 weeks gestation: nonsensus consensus

*Falsehood flies and the truth comes limping after; so that when men come to be undeceived it
is too late: the jest is over and the tale has had its effect.*

Jonathan Swift, *The Examiner*, No. 15, November 9, 1710

Introduction

Traditionally pregnancy has been considered ‘post-term’ at 42 completed weeks of gestation. At this gestation, if the cervix is unfavourable, debate over best practice has been between routine induction of labour and expectant management with some form of serial fetal monitoring. Popular wisdom seems to be that meta-analysis of the available randomised controlled trials has settled the question in favour of routine induction¹. The largest included trial, containing over half the cases ($n = 3407$), was carried out in Canada and published in 1992². The results of the meta-analysis led the Society of Obstetricians and Gynaecologists of Canada (SOGC) to issue *Clinical Practice Guidelines* in 1997³. The guidelines recommended that: 1. after 41 completed weeks of gestation, if the dates are certain, women should be offered elective delivery; 2. if the cervix is unfavourable, ripening should be undertaken; and 3. if expectant management is chosen, assessment of fetal health should be initiated.

It is presumed that randomised controlled trials or, even better, meta-analyses of randomised trials, provide the best evidence to determine appropriate care. However, once information has been declared ‘the best available evidence’, particularly if that assertion is used to justify clinical practice guidelines or ‘consensus’, further inquiry may be inhibited⁴. Since it is implied that ‘the answers are all in’, mutation from clinical practice guideline to standard of care is prompt and uncomplicated, particularly if the labels ‘consensus’ or ‘policy statement’ are used between the two as conceptual mutagens. The standard of care in Canada now is assumed to be routine induction at 41 weeks. This commentary is intended to give pause to those who have accepted and adopted this standard.

What are the true fetal and neonatal risks of reaching 41 weeks of gestation?

The SOGC *Clinical Practice Guidelines* assert that “women who reach 41 weeks should be counselled appropriately regarding the higher risk...to their babies if they

should pursue a policy of expectant management”³. How large is this purported higher risk, and what is the strength of evidence used to support this assertion?

The following information comes from studies conducted before 1992 when ‘41 weeks undelivered’ had not been categorised as vigorously as a time of higher risk. In that era, intervention by induction for gestation only was not routine practice, and fetal surveillance because the pregnancy was undelivered at 41 weeks of gestation was not used.

Such evidence does not describe the natural history of all pregnancies that would have reached 41 weeks without intervention. Women with identified maternal or fetal complications such as pregnancy-induced hypertension, other medical problems, or suspected fetal growth restriction likely would have been delivered sooner, and women with favourable cervixes might have been induced, but the situation of the remainder would have been similar to that of women eligible for the Canadian study.

Based on data from New York City (1987–1989)⁵, Japan (1989–1992)⁶, Sweden (1982–1991)⁷, and London (1989–1991)⁸, the risk of stillbirth in the subsequent week (41 weeks zero days) is about 0.1% (1.04–1.27 per 1000) (Table 1). Similar estimates were presented in reports from New Zealand (1983–1986)⁹ and England (1978–1985)¹⁰. The stillbirth rate in the expectant arm of the Canadian study was similar at two fetal deaths in 1700 women (1.18 per 1000)².

These estimates are contemporary with the Canadian trial and are consistent, as they are with the situation contemporary to the Canadian study at one Winnipeg tertiary obstetric hospital (Table 2). In the latter setting, from 1982–1991, induction for gestation only was not routine and fetal surveillance for post-term pregnancy was not begun until 42 weeks. Of 7725 pregnancies that reached 41 weeks undelivered, eight stillbirths occurred in the next week, and there were three neonatal deaths—two from disseminated herpes, one from birth asphyxia—in babies born between 41 weeks zero days and 41 weeks six days.

The authors of the Canadian study suggested that, were it not for their fetal monitoring, perinatal mortality would

Table 1. Stillbirth risk in following week per 1000 undelivered women at beginning of week.

Week	New York City ⁵ 1987–9	Japan ⁶ 1989–92	Sweden ⁷ 1982–91	NE Thames London ⁸ 1989–91
36	0.39	0.27		0.45
37	0.51	0.29		0.35
38	0.59	0.32	0.39	0.56
39	0.64	0.42	0.45	0.57
40	0.88	0.68	0.68	0.86
41	1.17	1.04	1.11	1.27
42 (or ≥42)	1.63	3.10	1.87	1.55
43	2.72			2.12

have been higher than two babies in 1700 (1.18 per 1000). However, evidence from six countries (one being Canada) suggests that, as of a decade ago, when such monitoring was not done for gestation alone, the stillbirth rate in the subsequent week was about one in 1000 and approximately 1000 inductions would have been necessary at 41 weeks to prevent one stillbirth in the ensuing week, presuming cause and effect relationship between that death and gestational age. Since serial fetal assessment is now common in our setting, in part in defence against implications of the SOGC *Clinical Practice Guidelines*, most significant perinatal complications have been identified and dealt with by delivery before 41 weeks, and the odds of stillbirth in the following week likely have been further reduced.

What of the meta-analysis¹ which claims to demonstrate that “routine induction of labour *after* 41 weeks reduces perinatal death”? Meta-analysis attempts to describe what happened, but not why. As retrospective assessment, inevitably such efforts are subject not only to what happened in the past, but also to the accuracy and completeness with which those events have been described and analysed. According to the aggregate data, seven nonanomalous perinatal deaths occurred in 3002 women randomised to expectant management, compared with 1 from 3071 women who were induced. Of the seven perinatal deaths in the expectant arms, two deaths occurred in the 1960s¹¹, before the availability of modern fetal testing. Of those, one was a stillbirth in a mother with an abnormal glucose

tolerance test; such a situation would not likely be allowed to reach 41 weeks now, and was a specific exclusion from the Canadian study. The other was a neonatal death, from meconium aspiration, following refusal of induction by the mother after positive amniocentesis. One perinatal death¹², in China, was caused by pneumonia in the newborn period, a cause unrelated to the duration of pregnancy. One perinatal death¹³ was caused by meconium aspiration in a baby born at 43⁺³ weeks, which is irrelevant to whether induction should be carried out at 41 weeks. Of the two deaths in the Canadian trial², one was a stillbirth at 41⁺⁵ weeks, but the mother had not received any fetal testing. The second was an intrapartum death of a 2600 gm infant at 42 weeks, ascribed to fetal distress, which presumably could have occurred and resulted in similar management difficulties during earlier induction. This death was plausibly preventable by induction at 41 weeks, as was a stillbirth in another study¹⁴ from massive abruption at 41⁺⁵ weeks. However, 2600 grammes is an abnormal birthweight in Canada for 42 weeks of gestation, so the hypothesis that death occurred as a result of gestation alone is dubious. Thus of seven perinatal deaths in 3002 women randomised to expectant management, only two occurred in women who received contemporary fetal testing, before 43 weeks, from a cause possibly related to pregnancy duration. This is against one death in the induction group. The higher risk that routine induction at 41 weeks aims to reduce is dubious, if it exists at all.

Table 2. Women’s Hospital, Winnipeg 1982–1991. Exclusions: 1) Transferred from other institution with fetus already dead *in utero*; 2) No prenatal care before gravida arriving with dead fetus; 3) Fetus known to have died before 36 weeks; 4) Principal cause of death related to congenital anomaly.

Gestational age	No. undelivered at beginning of week	No. stillbirths that week	Stillbirth rate per 1000 undelivered women at beginning of week	Neonatal death(s)	Total perinatal death rate that week
36	33,740	10	0.30	1	0.33
37	32,658	8	0.25	2	0.31
38	30,717	13	0.42	4	0.55
39	25,631	11	0.43	8	0.74
40	17,639	8	0.45	6	0.79
41	7725	8	1.04	3 [†]	1.42
42+	2455	4	1.63	1 [‡]	2.04

[†] 2 deaths from disseminated herpes infection; 1 death from birth asphyxia.

[‡] 1 death from meconium aspiration syndrome.

What are the maternal risks of reaching 41 weeks of gestation?

The SOGC *Clinical Practice Guidelines* asserts that “women who reach 41 weeks of gestation should be counselled appropriately regarding the higher risks to themselves...if they should pursue a policy of expectant management”³.

Bias arising from improperly executed randomised designs threatens, and potentially invalidates, the conclusions of such efforts. Avoiding such errors requires not only eliminating bias from entry allocation, but also differing treatment. The Canadian study found that “the rate of cesarean section was significantly higher among women in the monitoring group (24.5%) than among those in the induction group (21.2%)”². Its authors admit a bias that might have accounted for part of the induction cohort’s lower rate of caesarean section, that prostaglandin gel was used for cervical ripening only in that arm of the trial. Although the authors acknowledged later that “use of prostaglandin gels appears to be the best method for inducing labour, particularly when the cervix is unfavourable”¹⁵, prostaglandin was proscribed in the expectant cohort “because we thought that most of the women in that group who would require induction of labour would have evidence of fetal compromise”².

In fact, a third (34%) of the women in the monitoring group were induced, but only half of them (17% overall) for ‘fetal compromise’, with the nature and validity of that generalisation undefined. The above-mentioned rationalisation for withholding prostaglandin from the monitoring group implies that the method was believed to be too dangerous given possible fetal compromise. It is more logical that suspected fetal compromise would make reduction in the number of contractions needed to accomplish vaginal delivery desirable. Thus, prostaglandin cervical ripening would be indicated, not contraindicated, and rationalisation in the Canadian trial’s is a *non sequitur*.

A second bias that could have contributed to the higher rate of caesarean section in the expectant cohort is that the trial was not blinded. Both *accoucheurs* and patients knew that what was being assessed was whether it was safe to let pregnancy continue past 41 weeks. It is likely that as the duration of pregnancy extended, both groups would have felt increasing pressure to intervene, possibly with caesarean section, in this so-called and so-conceived high risk situation. This assertion is not hypothetical, rather probable, given the revelations of Tversky and Kahneman about how humans make decisions in the presence of uncertainty¹⁶.

There is a third important bias which could lead to greater use of caesarean section in the monitoring cohort. Within that group, 17% of women were believed to have sufficient evidence of fetal compromise that “the fetus was to be delivered immediately”². Envision a woman randomised to the expectant, possibly conceptually high risk group. The clinician is told that monitoring indicates that

the fetus is ‘compromised’ or in distress and should “be delivered immediately”. In such enhanced alarm, tolerance of typically benign intrapartum fetal heart rate changes or the passage of meconium would be reduced, and caesarean section for such imprecise signals would be more likely¹⁷.

There is considerable evidence for such behavioural bias in obstetric settings, and that obstetric thinking confirms *in vivo* what Tversky and Kahneman described as the availability error¹⁶. The Toronto Tri-hospital trial revealed that labelling a woman gestational diabetic conferred a doubled rate of caesarean section, regardless of the fetal or maternal condition, and with no relationship to birth-weight¹⁸. Elsewhere, false positive prediction by ultrasound of macrosomia provoked a 50% increase in caesarean delivery of same weight babies¹⁹. In a German study, the label growth retardation biased interpretation of, and action taken for, fetal cardiotocography and led to twice as many caesarean sections as occurred in undetected cases of growth restriction²⁰. In a Swedish study, older nulliparae had dramatically increased odds of caesarean delivery, regardless of maternal or fetal condition²¹. A study from Iceland and Scotland of 522 twin pregnancies in 1990–1993 revealed no difference in management or outcomes of natural ($n = 453$) versus assisted ($n = 69$) conceptions, except that elective caesarean delivery was twice as likely in the assisted conception group²². A Canadian study of the definition and management of dystocia found that among the strongest determinants of a decision for caesarean section were acquisition of a dystocia perception and label, or its equivalent in the mind of the attending physician and the hospital in which the decision was made, although a significant proportion of such decisions were made before active labour²³.

The higher rate of caesarean sections in the Canadian study’s expectant group was almost completely accounted for by more operations for fetal distress [8.3 versus 5.7%]². The authors suggested this occurred because fetuses become progressively compromised and more prone to intrapartum fetal distress as pregnancy becomes more post-term. An alternative, better substantiated explanation is that monitoring created and reinforced bias toward inference of fetal distress and made it more likely that caesarean delivery would be the response to that inference. Imprecision of the term fetal distress in obstetric care, despite its liberal use, promotes the availability error in decision making, given uncertainty¹⁶. As the true risk, in contrast to the perceived risk, of a fetus dying between 41 and 42 weeks, *in the absence of monitoring*, is only 0.1%, it is extremely unlikely that the 17% of fetuses in the expectant group believed to be compromised were actually in trouble. Over 99% of the supposedly compromised fetuses detected by monitoring most likely were not, but were rescued from normalcy by operative delivery for enhanced provider and patient anxiety.

The assertion that induction at 41 weeks results in fewer caesarean sections than expectant management is doubtful

at best. It is particularly difficult to reconcile with considerable and consistent evidence that induction, especially in nulliparae with unfavourable cervixes, markedly increases the rate of caesarean sections²⁴⁻³². In a four-year period in southern Alberta, the caesarean rate for women induced in their 41st week was 23%, compared with 14% in those who laboured spontaneously in the 41st week³². The SOGC cautioned against induction before 41 weeks, in that “particularly in nulligravida...the likelihood of caesarean section may be twice as great when labour is induced as compared with spontaneous”³. Why this should not be the case for induction at 41 weeks is unexplained, and unlikely. Given the odds of stillbirth of 0.1% in the 41st week without induction for dates alone or special fetal surveillance, the influence of fetal risk is more likely that of perception than reality.

One of the most influential biases in the acquisition of evidence is choice of the question, and the best evidence in answer to the wrong question is useless. The rate of caesarean section were reported in the Canadian study by intention-to-treat, but they should be analysed also as *actually* treated. In the intended-to-induce group, 31% of women were *not* induced, and in the intended-not-to-induce cohort, 34% of women *were* induced. In essence, one-third

of each cohort were treated by the opposite method to that intended. In that true fetal compromise is rare at 41 weeks, the Canadian study was comparing elective induction compared with expectant management at 41 weeks. Comparing the rates of caesarean section in all women induced *versus* all women who laboured spontaneously at 41 weeks would be a more valid test of whether induction at 41 weeks alters the caesarean rate, or conveys any other advantage or disadvantage.

One can estimate the results presented as ‘treatment actually received’ using the Canadian study’s reported percentage of women in each group treated by the method intended (Fig. 1a and b). Assuming a rate of caesarean section of 16% in women starting spontaneous labour, regardless of intention-to-treat allocation, one would obtain a caesarean rate of 16% in women who laboured spontaneously compared with 29% in those who were induced. If one recalculates using the 14% caesarean rate for spontaneous labour at 41 weeks in Alberta in the early 1990s³², this difference becomes even more striking.

The appropriate counselling “regarding the higher risks to themselves”, that the SOGC *Clinical Practice Guidelines* assert must be provided to women who reach 41 weeks of gestation, should be that the higher risk is of caesarean

		INTENTION TO TREAT GROUP			
		TO INDUCE		NOT TO INDUCE (MONITORING)	
ACTUAL TREATMENT RECEIVED	INDUCTION	1174	580	1754	
	SPONT LABOR	527	1126	1653	
		1701	1706		
		360 CS (21.2%)	418 CS (24.5%)		

		INTENTION TO TREAT GROUP			
		TO INDUCE		NOT TO INDUCE (MONITORING)	
ACTUAL TREATMENT RECEIVED	INDUCTION	$\frac{276}{1174}$ (24)	$\frac{238}{580}$ (41)	$\frac{514}{1754}$ (29)	
	SPONT LABOR	$\frac{84}{527}$ (16)	$\frac{180}{1126}$ (16)	$\frac{264}{1653}$ (16)	
		$\frac{360}{1701}$ (21.2)	$\frac{418}{1706}$ (24.5)	$\frac{778}{3407}$ (22.8)	

Fig. 1. (A) The numbers of women and caesareans at the bottom of the vertical columns from Hannah¹⁵. The numbers in each cell are estimates derived from ref. 2; (B) the results if one assumes a 16% caesarean rate in women who start labour spontaneously. The numerators are numbers of caesareans, the denominators are numbers of women, and in parentheses is the percentage of caesarean sections.

delivery for dubious reasons, and that to avoid it they should labour and deliver where induction for dates alone is not the ritual at 41 weeks of gestation. Despite excluding women with medical or fetal problems, an urgent need for delivery or contraindications to vaginal delivery, 31% of nulliparous women in the Canadian study were delivered by caesarean section¹⁵. Rather than establishing the case for routine induction at 41 weeks, the results of the Canadian trial reflect the high intervention rates of obstetric practice in Canada, which has the second highest rate of caesarean section in the developed world³³. Given this specific intervention epidemic, it may be appropriate to note as well that since previous caesarean section was an exclusion criterion, the conclusions of the Canadian study even if valid, would be inapplicable to women in such circumstances.

Resource consequences of a policy of routine induction at 41 weeks of gestation

Left alone, a significant proportion of pregnancies are undelivered by 41 weeks of gestation. In one study using ultrasound dating, 19% of women were undelivered at 41 weeks, whereas only 3.5% were undelivered at 42 weeks³⁴. In the aforementioned Swedish study, 30% of nulligravidae reached 287 days undelivered whereas 10% reached 294 days⁷. In our setting, 23% of women undelivered by 36 weeks remained as such at 41 weeks *versus* 7.5% at 42 weeks (Table 2).

Although proportions of pregnancies undelivered by 41 *versus* 42 weeks vary between populations, depending in part on the use of ultrasound dating³⁵, about 15%–20% more women will be induced given routine induction at 41 as opposed to 42 weeks. Using an annual delivery volume of 4000 births per year, about 1000 inductions would be done solely because gestation had reached 41 weeks, *versus* 140–400 per year (3.5%–10%) if induction for gestation only was deferred to 42 weeks. Presuming that hospitals would reserve such induction for otherwise untroubled mothers and fetuses to five weekdays in each of 52 weeks, a hospital with 4000 births per year would have to provide for three added inductions per day, given a policy of such interference at 41 weeks. These would be in addition to those indicated for legitimate and significant maternal or fetal threat. This is a staggering imposition, given that at least 500 and more likely over 1000 inductions must be done to prevent one perinatal death from unspecified relationships to gestation.

We anticipate at least two objections to this analysis. One is that the SOGC *Clinical Practice Guidelines* do not state explicitly that induction must occur at 41 weeks zero days. The document is written vaguely enough to be interpreted that induction any time between 41 weeks zero days and 41 weeks six days is acceptable. However, in response to the *Clinical Practice Guidelines*, Canadian obstetricians, at least the ones in the authors' hospitals,

now book induction by, if not before, one week past the supposed due date, ignoring the modifier 'estimated', as well as biologic norms and realities. They fear medico-legal implications should the fetus die at seven or more days past the due date, with no regard for the true odds and likely causation of such outcomes. The adverse consequences of *Clinical Practice Guidelines* have been described in various situations^{36–38}. Lest we be perceived as criticising the best intentions of our competent and caring colleagues, the nonsensical consensus about management of uncomplicated undelivered pregnancy at 41 weeks is simply a *Clinical Practice Guidelines*—reinforced example of the availability error. Thereby, adversity odds are significantly overestimated, normally odds are even more significantly underestimated, and both logic and behaviour are warped as a result¹⁶.

A second anticipated objection is 'Those to be induced at 41 weeks must labour and deliver sooner or later, so what is the difference?' The difference is between arriving in labour and delivering 5–10 hours later compared with induction with an unfavourable cervix, requiring ripening with its variable success, then labour for 10 hours or more. The workload increment for nursing, midwifery and medical staff is significant given the need to induce 15%–20% more of the pregnant population, and in that improved outcomes are dubious, indefensible.

Inevitable, unintended and undesirable consequences of routine induction at 41 weeks

Greatly increased obstetric workload may be argued to be an acceptable imposition because, otherwise, one baby in 1000 reaching 41 weeks *might* die. We concede that, rarely, one such fetus might be saved. No test of fetal wellbeing is or likely ever will be perfect. But it is uncertain that routine induction at 41 weeks will reduce the number of fetuses who die, and it is arguable that such practice could increase perinatal mortality and morbidity. Attention is a limited resource³⁹. The extra attention needed for such added induction and its consequences will draw attention away from women labouring spontaneously or who are being induced for more compelling reasons. A mother, or a fetus of less than 41 weeks who needed help, harmed because people were busy with somebody else who did not need help, will not be counted in morbidity and mortality analysis of intervention by induction of labour at 41 weeks of gestation.

In one of the authors' hospitals, a pregnant woman admitted because of hypertension complained of headaches while her blood pressure rose to 170/110 mmHg. Intravenous antihypertensive drugs were allowed to be given only on the labour floor. Transfer to the labour floor was delayed because there were no beds available, several being filled with 41-week inductions. The woman died from intracranial haemorrhage before transfer. Anecdotes are not the singular

Table 3. Number of stillbirths at each week of gestation.

Week	New York City ⁵ 1987–9	Japan ⁶ 1989–92	Sweden ⁷ 1982–91	NE Thames London ⁸ 1989–91
36	132	1284		
37	169	1345		56
38	181	1381	359	83
39	173	1431	349	72
40	175	1328	366	81
41	130	624	284	50
≥42	114	245	144	20

source of evidence. But we wonder, whenever near-misses, near-catastrophes or true disasters occur on labour wards, whether they could have been anticipated and prevented had the staff not been so busy. As was stated in another context “preoccupation with the potential benefit to the numerator may make doctors less sensitive to the adverse effects on the population”⁴⁰.

The Canadian trial² resulted in a grave morbidity which we discovered during research into cervical cord injury⁴¹. A mother randomised to induction was induced, with prostaglandin. Precipitate labour ensued, with rapid progress to full dilation, severe decelerations, forceps rotation and extraction. The baby sustained high cervical cord injury and quadriplegia. This complication was not identified in the publication², a subsequent reinterpretation¹⁵, nor in the SOGC *Clinical Practice Guidelines*³ and there was no such incident in the study’s expectant cohort.

Conclusion

The median and mode for uncomplicated singleton pregnancy are 40 weeks two days and 40 weeks three days, respectively^{42,43}, not ‘40 weeks’, and two standard deviations beyond that is approximately 13 days. Approximately one-quarter of pregnant women have not laboured by 41 weeks. Their stillbirth rate in the subsequent week without fetal surveillance is approximately 1 in 1000. Routine induction at 41 weeks is ritual induction at term, unsupported by rational evidence of benefit. It is unacceptable, illogical and unsupportable interference with a normal physiologic situation.

Two decades ago it was argued “that any infant born at term should survive, provided the infant has no lethal malformation”⁴⁴. If only a fragment of such hyperbole is used to rationalise ritual induction at 41 weeks, to be logically consistent, we should induce everybody at 40, or perhaps 39, or 38, or even 37 weeks. Although the stillbirth rate at those earlier gestations is less than at 41 weeks, the absolute number of fetuses who die is greater. Since more babies die at those gestations than die in week 41 (Tables 2 and 3), more lives *could*—we have not written *would*—be saved.

Almost a quarter of a century ago, the prescient authors of an article entitled *Intervention and Causal Inferences in*

Obstetric Practice cautioned that “as . . . interventions are applied to an increasingly large proportion of the obstetric and fetal population, a threshold will inevitably be reached beyond which the marginal risks of the procedure will outweigh the marginal benefits”⁴⁵. The ‘evidence’ on which current practice and popularity of routine or as we prefer to think of it, ritual induction at 41 weeks, is based is seriously flawed and an abuse of biological norms. Such interference has the potential to do more harm than good, and its resource implications are staggering. It is time for this nonsensical consensus to be withdrawn.

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References

1. Crowley P. Interventions to prevent or improve outcome from labour at or beyond term. The Cochrane Library, Oxford: Update Software Issue 1:2001.
2. Hannah ME, Hannah WJ, Hellmann J, Hewson S, Milner R, Willan A, and the Canadian Multicenter Post-term Pregnancy Trial Group. Induction of labor as compared with serial antenatal monitoring in post-term pregnancy. A randomized controlled trial. *N Engl J Med* 1992;**326**:1587–1592.
3. Maternal–Fetal Medicine Committee of the Society of Obstetricians and Gynaecologists of Canada. Post-term pregnancy (Committee Opinion). *SOGC Clinical Practice Guidelines*, No. 15, 1997.
4. Hall PF. The consensus cult. *J Soc Obstet Gynaecol Can* 2000;**22**:8.
5. Feldman GB. Prospective risk of stillbirth. *Obstet Gynecol* 1992;**79**:547–553.
6. Minakami H, Kimura H, Honma Y, Tamada T, Sato I. When is the optimal time for delivery?—purely from the fetuses’ perspective. *Gynecol Obstet Invest* 1995;**40**:174–178.
7. Ingemarsson I, Kallen K. Stillbirths and rate of neonatal deaths in 76,761 post term pregnancies in Sweden, 1982–1991: a register study. *Acta Obstet Gynecol Scand* 1997;**76**:658–662.
8. Hilder L, Costeloe K, Thilaganathan B. Prolonged pregnancy: evaluating gestation-specific risks of fetal and infant mortality. *Br J Obstet Gynaecol* 1998;**105**:169–173.
9. Widjaja AH, Mantell CD. Stillbirths in Auckland 1983–1986. *NZ Med J* 1988;**101**:768–770.
10. Yudkin PL, Wood L, Redman CWG. Risk of unexplained stillbirth at different gestational ages. *Lancet* 1987;**1**:1192–1194.
11. Henry GR. A controlled trial of surgical induction of labour and amniocentesis in the management of prolonged pregnancy. *J Obstet Gynaecol Br Commonw* 1969;**76**:795–798.

12. Bergsjø P, Gui-dan H, Su-qin Y, Zhi-zeng G, Bakketeig L. Comparison of induced versus non-induced labour in post-term pregnancy: a randomized prospective study. *Acta Obstet Gynecol Scand* 1989;**68**: 683–687.
13. Dyson DC, Miller PD, Armstrong MA. Management of prolonged pregnancy: induction of labor versus antepartum fetal testing. *Am J Obstet Gynecol* 1987;**156**:928–934.
14. Cardozo L, Fysh J, Pearce JM. Prolonged pregnancy: the management debate. *BMJ* 1986;**293**:1059–1063.
15. Hannah ME. Postterm pregnancy: should all women have labour induced? A review of the literature. *Fet Mat Med Rev* 1993;**5**:3–17.
16. Tversky A, Kahneman D. *Judgment Under Uncertainty: Heuristics and Biases*. Cambridge: Cambridge University Press, 1982.
17. Alfirevic Z, Walkinshaw SA. A randomized controlled trial of simple compared with complex antenatal fetal monitoring after 42 weeks of gestation. *Br J Obstet Gynaecol* 1995;**102**:638–643.
18. Naylor CD, Sermer M, Chen E, Sykora K. Cesarean delivery in relation to birth weight and gestational glucose tolerance: pathophysiology or practice style? *JAMA* 1996;**275**:1199–1200.
19. Levine AB, Lockwood CJ, Brown B, Lapinski R, Berkowitz RL. Sonographic diagnosis of the large for gestational age fetus at term: does it make a difference? *Obstet Gynecol* 1992;**79**:55–58.
20. Jahn A, Razum O, Berle P. Routine screening for intrauterine growth retardation in Germany: low sensitivity and questionable benefit for diagnosed cases. *Acta Obstet Gynecol Scand* 1998;**77**:643–648.
21. Cnattingius R, Cnattingius S, Notzon FC. Obstacles to reducing cesarean rates in a low-cesarean setting: the effect of maternal age, height, and weight. *Obstet Gynecol* 1998;**92**:501–506.
22. Agustsson T, Geirsson RT, Mires G. Obstetric outcome of natural and assisted conception twin pregnancies is similar. *Acta Obstet Gynecol Scand* 1997;**76**:45–49.
23. Stewart PJ, Duhllberg C, Arnett AC, Elmslie T, Hall PF. Diagnosis of dystocia and management with caesarean section among primiparous women in Ottawa Carleton. *CMAJ* 1990;**142**:459–463.
24. Xenakis EM-J, Piper JM, Conway DL, Langer O. Induction of labor in the nineties: conquering the unfavourable cervix. *Obstet Gynecol* 1997;**90**:235–239.
25. Smith LP, Nagourney BA, McLean FH, et al. Hazards and benefits of elective induction of labor. *Am J Obstet Gynecol* 1984;**148**:579–585.
26. Macer J, Macer CL, Chan LS. Elective induction versus spontaneous labor: a retrospective study of complications and outcome. *Am J Obstet Gynecol* 1992;**166**:1690–1697.
27. Buist R, Ranchhod N. Induction of labour: indications and obstetric outcomes in a tertiary referral hospital. *N Z Med J* 1999;**112**:151–153.
28. Maslow AS, Sweeny AL. Elective induction of labor as a risk factor for cesarean delivery among low risk women at term. *Obstet Gynecol* 2000;**95**:917–922.
29. Coonrod DV, Bay RC, Kishi GY. The epidemiology of labor induction: Arizona, 1997. *Am J Obstet Gynecol* 2000;**182**:1355–1362.
30. Seyb ST, Berca RJ, Socol ML, Dooley SL. Risk of cesarean delivery with elective induction of labor at term in nulliparous women. *Obstet Gynecol* 1999;**94**:600–607.
31. Yeast JD, Jones A, Poskin M. Induction of labor and the relationship to cesarean delivery: a review of 7001 consecutive inductions. *Am J Obstet Gynecol* 1999;**180**:628–633.
32. Lange I, Page R, Mah A, Yee J. Comparison of LSCS rates for induced vs spontaneous labour in low risk primigravids [poster presentation SOGC Conference, Calgary, Alberta]. *Southern Alberta Perinatal Audit Program Bulletin* 1995;**5**:2.
33. Stephenson PA, Bakoula C, Hemminki E, et al. Patterns of use of obstetrical interventions in 12 countries. *Paediatr Perinat Epidemiol* 1993;**7**:45–54.
34. Mongelli M, Wilcox M, Gardosi J. Estimating the date of confinement: ultrasonographic biometry versus certain menstrual dates. *Am J Obstet Gynecol* 1996;**174**:278–281.
35. Goldenberg RL, Davis RO, Cutter GR, Hoffman HJ, Brumfield CG, Foster JM. Prematurity, postdates, and growth retardation: the influence of use of ultrasonography on reported gestational age. *Am J Obstet Gynecol* 1989;**160**:462–470.
36. McIntyre KM. Medicolegal implications of consensus statements. *Chest* 1995;**108**(Suppl):502S–505S.
37. Hyams AL, Brandenburg JA, Lipsitz SR, Shapiro DW, Brennen TA. Practice guidelines and malpractice litigation: a two-way street. *Ann Intern Med* 1995;**122**:450–455.
38. Hirshfeld EB. Should practice parameters be the standard of care in malpractice litigation? *JAMA* 1991;**266**:2886–2891.
39. Cook RI, Woods DD. Operating at the sharp end: the complexity of human error. In: Bogner MS, editor. *Human Error in Medicine*. Hillsdale, NJ: Lawrence Erlbaum Associates, 1994:255–310.
40. Woolf SH, Kamerow DB. Testing for uncommon conditions: the heroic search for positive results. *Arch Int Med* 1990;**150**:2451–2458.
41. Menticoglou SM, Perlman M, Manning FA. High cervical spinal cord injury in neonates delivered with forceps: report of 15 cases. *Obstet Gynecol* 1995;**86**:589–594.
42. Bergsjø P, Denman DW, Hoffman HJ, Meirik O. Duration of human singleton pregnancy: a population based study. *Acta Obstet Gynecol Scand* 1990;**69**:197–207.
43. Baskett TF, Nagele F. Naegele's rule: a reappraisal. *Br J Obstet Gynaecol* 2000;**107**:1433–1435.
44. Stubblefield PG, Berek JS. Perinatal mortality in term and post-term births. *Obstet Gynecol* 1980;**56**:676–682.
45. Chalmers I, Richards M. Intervention and causal inferences in obstetric practice. In: Chard T, editor. *Benefits and hazards of the new obstetrics*. *Clin Devel Med* 1977;**64**:34–61.