INTRODUCTION

Postpartum hemorrhage (PPH) plays a major role in maternal mortality. It represents a risk that attends every delivery, and is an impending danger to every childbearing woman in the world. The chance of dying is not the same for everyone, as a number of factors may lead to a severe, life-threatening condition and to an adverse outcome. These conditions are not the same in every region of the world, but tend to worsen as do economic conditions. For many years, PPH was considered a public health issue, primarily in low resource countries. In recent years, however, often because of an improvement in the quality of patients’ clinical records, industrialized countries have witnessed what appears to be a slow but steady increase in the incidence of PPH, particularly due to postpartum uterine atony (PPUA). PPUA is the most common cause of PPH. However, all sources do not share the same definition or accept the same criteria of what is PPH; the very definition of PPH is not uniform in all jurisdictions; and the same may be said for the method of measuring the amount of blood loss. Remarkable differences also exist regarding what is accepted as immediate, early, or late PPH. Therefore, it is difficult to quantify and even to analyse to what extent it is true that this pathology is increasing. Possible causes of the variations cited here remain unclear, and no single cause has yet been identified as being responsible. However, as described later in this chapter, many different elements may be involved.

Notwithstanding these limitations, some indirect indicators point towards similar trends during recent years, for example, the number of transfusions administered within the first 12 hours postdelivery or the number of transfused blood derivatives (erythrocyte concentrates, platelets, etc.). In addition, population-based studies from Canada, Ireland, Australia, France, Norway, USA and other countries have demonstrated an increase in the incidence of PPH during the past decade. It is true that during this same period a number of changes in obstetric practice as well as maternal demographic characteristics may have contributed to an increased risk of PPH; these include an increase in the rate of cesarean delivery, a larger proportion of multiple births, and more pregnant women of advanced maternal age. However, there is insufficient evidence to support the proposition that these changes alone can be responsible for this increase. Furthermore, some well designed studies fail to find these reputed causative factors, but find other possible risk factors.

To date, there are surprisingly few population-based studies on PPH, particularly longitudinal studies. Most studies investigating risk factors for this condition have been small, case-controlled or hospital-based in design. These studies vary regarding the classification of PPH in terms of amount of blood loss, actual measurement of hemorrhage and the accompanying markers of hemodynamic compromise. Moreover, very few have considered the covariates in multivariate analysis. Despite the wide variation in design and results, the coinciding trends towards an increase is striking when the results are plotted (Figure 1).

Recently, several collaborative studies have searched for the relationship between specific risk factors and the increased incidence of PPH. Unfortunately most of the data are heterogeneous and reliable comparison of collected data remains a difficult task. In 2009, the International Collaborative Group, which included representatives from Australia, Belgium, Canada, France, UK and USA, presented the results of pooled data from studies carried out in their respective countries. Some of the issues raised at that meeting are summarized here.

Australia

Some of the sources employed by the International Collaborative Group were national registers, for example, in Australia, the data came from the Admitted Patient Data Collection (APDC), which carries the registers of every hospital discharge record in New South Wales (NSW). Diagnoses and procedures are coded according to the International Statistical Classification of Diseases and Related Health Problems, Australian Modification (ICD-9 to July 1998 and ICD-10 subsequently) and the affiliated Australian Classification of Health Interventions. PPH is defined as a hemorrhage of 500 ml or more following vaginal
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or 750 ml or more following a cesarean delivery resulting in a recorded clinical diagnosis of PPH and identified during the birth hospitalization from the hospital data. Data for Victoria were derived from the perinatal data collection form. The ICD-10AM definition of PPH was also used.

In the case of the Australian registers, in at least two regions, NSW and Victoria, a continuous increase in PPH was registered between 1991 and 2006 despite a stepped change between 1998/1999 which might be related to a change in definition of PPH from 600 to 500 ml)20. In NSW, Cameron et al.21 assessed trends and outcomes of PPH in a population-based descriptive study of 52,151 women who had PPH either at the time of delivery or requiring a re-admission to hospital for this condition in the years between 1994 and 2002. The outcome measures included maternal death, hysterectomy, admission to intensive care unit (ICU), transfusion and major maternal morbidity, including procedures to reduce blood supply to the uterus, acute renal failure and postpartum coagulation defects. The author found that, during that period, both the number and adjusted (for under-reporting) rate of PPH during the birth admission increased from 8.3% to 10.7% of deliveries. The rate of PPH adjusted for maternal age and mode of delivery was similar to the unadjusted rate. There was a six-fold increase in the rate of transfusions among women who hemorrhaged from 1.9% to 11.7%. At the same time, the hospital re-admissions for PPH declined from 1.2% of deliveries to 0.9%. These changes were statistically significant, although no significant changes occurred in the rates of hysterectomies, procedures to reduce blood supply to the uterus, admissions to ICU, acute renal failure or coagulation defects.

Canada

In Canada, information was based on all hospital deliveries as documented in the Discharge Abstract Database of the Canadian Institute for Health Information from 1991 to 20057,8,22. All medical diagnoses were coded according to the ICD, whereas specific procedures were coded using the Canadian Classification of Diagnostic, Therapeutic and Surgical Procedures (CCP) and the Canadian Classification of Interventions (CCI). PPH was coded if blood loss after childbirth exceeded 500 ml after a vaginal delivery, 1000 ml after a cesarean delivery or when the physician made a notation of PPH in the medical chart.

The Canadian authors used code the ICD-9, registering PPH and, lately, ICD-10 codes for PPH due to retained placenta (third stage hemorrhage), uterine atony (immediate PPH, within the first 24 hours following delivery of placenta), delayed and secondary PPH (after the first 24 hours following delivery) and PPH due to coagulation defects, respectively. It is important to emphasize that those women whose deliveries were complicated by PPH and who additionally had an abdominal hysterectomy were identified using the relevant CCP and CCI codes. Blood transfusions were identified using a specific code introduced in the database in 1994 (blood transfusion rates were unavailable before that year).

France

The information from France came through the published PPH data by Dupont et al.23. According to these data, PPH occurred in 1144 of 21,350 deliveries, an overall incidence of 5.4 ± 0.3%. Among these, 316 cases were coded as severe. Diagnosis was clinical in 82.5% of severe cases and 77.5% of non-severe cases; the remainder were detected by postpartum laboratory tests. Uterotonic agents were given prophylactically to 46.7% of the 896 patients following vaginal delivery. In cases in which PPH was due to uterine atony, 83.1% of women underwent examination of the uterine cavity and 96.3% received oxytocin, which proved to be therapeutic. Sulprostone was administered to 39.5% cases of persistent PPH. Regarding cesarean delivery, a uterotonic was given prophylactically to 85.4% of the 247 patients delivered. Oxytocin was therapeutic in 94.8% of cases of uterine atony. Sulprostone was administered in 84.4% of cases of persistent PPH. However, the median delay for second-line pharmacological treatment was significantly shortened (from 80 min, range 35–130, in 2002 to 32.5 min, range 20–75 in 2005). An increase was observed in the use of surgery for PPH (0.06% versus 0.12% of deliveries; p = 0.03) and in blood transfusions (0.18% versus 0.33%; p = 0.01). Nonetheless, the prevalence of major PPH did not change (0.80% versus 0.86% of deliveries; p = 0.62)24.

United Kingdom

In the UK, the information from Scottish sources was obtained from the Scottish Confidential Audit of Severe Maternal Morbidity25. UK data on PPH, including data from England, Scotland, Wales and Northern Ireland were obtained from the UK Obstetric Surveillance System (UKOSS) survey of hemorrhage-associated peripartum hysterectomy and severe obstetric hemorrhage in UK between 2003 and
In their registers, 318 women underwent peripartum hysterectomy. The most commonly reported causes of hemorrhage were uterine atony (53%) and morbidly adherent placenta (39%). Women were not universally managed with uterotonic therapies. Fifty women were unsuccessfully managed with B-Lynch or other brace surgery prior to hysterectomy, 28 with activated factor VII and nine with arterial embolization. Twenty-one per cent of women who suffered damage to other structures, 20% required a further operation and 19% were reported to have additional severe morbidity. Bladder damage was more likely in women with placenta accreta than in women with uterine atony. There were no significant differences in outcomes between women undergoing total or subtotal hysterectomy. Two women died resulting in a case fatality rate of 0.6% (95% CI 0–1.5%). Regarding changes in incidence, a mild increase in the numbers of PPH was documented during the study period, particularly when evaluating the past 6 years.

Belgium

In Belgium, data came from the hospital discharge data for the Flanders region, and to determine the proportion of women receiving a blood transfusion within 24 h of birth as a proxy for PPH. A population-based study reported an increase in the risk of severe PPH associated with induction of labor (OR 1.71). However, further analysis of data on peripartum hysterectomy to control PPH in the region did not show an association with induction of labor even after adjustment for previous cesarean delivery. According to authors’ findings, labor induction and other changes in obstetric practice could lead to an increased duration of labor, in both first and second stage, which might contribute to the already documented increase in the frequency of PPH.

United States

In the US data used by the International Collaborative Group were obtained from the Nationwide Inpatient Sample (NIS) for 1994–2006 (partially summarized in Figure 1). The NIS is a large public-use administrative dataset that includes approximately 20% of all of the discharges from non-Federal, acute-care hospitals in the US. The database is maintained by the Agency for Healthcare Research and Quality as part of the Healthcare Utilization Project. The database contains up to 15 diagnosis fields and 15 procedure fields; diagnoses and procedures are coded at the hospital at the time of discharge using the ICD-9-CM. Patients were subsequently stratified based on the presumed etiology of their PPH according to the categories defined in the ICD-9 classification: 666.0 for PPH from retained placenta (including placenta accreta), 666.1 for PPH from uterine atony, 666.2 for delayed and secondary PPH (after the first 24 hours after delivery), and 666.3 for PPH caused by coagulation defects. Patients who received transfusion of blood products or who underwent a peripartum hysterectomy were also identified using the ICD-9 codes. Deliveries in which maternal age was not recorded were excluded from analysis.

In summary, findings indicated that, during the period 1996–2004, the percentage of deliveries with a diagnostic code of PPH increased by 26% (from 2.3 to 2.9% of all deliveries). This change in incidence was mainly due to uterine atony. It is worth mentioning that the highest rate of uterine atony occurred among women whose labor was induced and who delivered vaginally. This was followed by women whose induction ended in cesarean delivery and women who had vaginal births without induction of labor. Women, who had cesarean deliveries and did not have induced labor, consistently had the lowest rates of PPH caused by atony.

According to this particular register, women who had vaginal deliveries were at increased risk for PPH. However, the data from the past 2 years show that, the incidence of PPH is similar for all groups (spontaneous, induced, failed induction and scheduled cesarean section). Thus, the percentage change in uterine atony was dramatically greater for cesarean deliveries compared with vaginal deliveries.

In Canada, Australia and the USA, rates of hysterectomy for peripartum hemorrhage increased from 2.3 to 4.0 per 100,000 deliveries in 1991 to 18.4 per 100,000 deliveries in 2004 (a 73% increase, 95% CI 27–137). On the other hand, maternal mortality from hemorrhage appeared to be relatively static (Australia between 1994–1996 and 2003–2005, France between 1997–1999 and 2000–2002, UK between 1985–1987 and 2003–2005, US between 1998 and 2004)7, although the rate of Sheehan’s syndrome increased in Canada from 3.7 per million deliveries in 1991–1993 to 12.6 in 2002–2004 (241% increase, 95% CI 8% decrease to 1,158% increase p = 0.10; p value or increasing annual linear trend = 0.008). Unfortunately, similar data were not available from other countries. In Canada no evidence of an increase in maternal mortality from PPH and/or in blood transfusion for PPH was seen.

In all populations examined, maternal age at childbirth was increasing, cesarean delivery was becoming more common, and multiple
pregnancy rates were also increasing\textsuperscript{15,37,38}. The proportion of induced labors increased over a similar time period as the observed increase in PPH\textsuperscript{7,16,27,39}.

The International Collaborative Group also investigated possible associations between environmental contaminants, toxins or environmental toxins, alternative/complementary medicine, antidepressants and PPH but failed to identify environmental factors that may have been responsible for these recent increases\textsuperscript{23}.

RELATED ISSUES

Variations in definition

It is unfortunate that no single definition of PPH is accepted. In some countries definitions are 500 ml for a vaginal delivery and 1000 ml for a cesarean section. In contrast, a blood loss of 500 ml for a vaginal delivery and 750 ml for a cesarean delivery is used in Australia\textsuperscript{20}, and in others the 500 ml blood loss is used to define PPH irrespective of the mode of delivery; further research is required to investigate how definitions are applied in practice to the coding of data. Regardless of the specific definitions used, routine visual estimates of blood loss are frequently inaccurate\textsuperscript{30,31} (see Chapters 9 and 11), and recent analyses using calculated blood losses demonstrate that many and perhaps most women lose sufficient blood at delivery to meet the diagnostic criteria for PPH\textsuperscript{31,32}.

Alternatively, PPH has been defined as a 10\% or more drop in hematocrit\textsuperscript{1}. The use of blood transfusions and procedures to control bleeding have both been used as markers of the severity of PPH and to identify women with severe pregnancy morbidity\textsuperscript{2,3,6,25,40}. How these definitions are used, their inherent inaccuracies, and the translation of definitions to administrative ICD coding may complicate the interpretation of trend data. In Australia, Scotland and the USA, for example, increases in the reported rates of severe complications of childbirth have been almost entirely due to increases in the use of blood transfusions and/or severe obstetric hemorrhage\textsuperscript{10,41–45}. In these countries, it appears that not only are PPH rates increasing, but so is the hemorrhage severity. In contrast, Canadian rates of severe maternal morbidity remained stable between 1991 and 2000 in the context of comparatively low and stable rates of transfusion\textsuperscript{4,7,15}. Such international differences may reflect differing attitudes among obstetricians about blood transfusions\textsuperscript{16,37,46}.

It has been suggested that restriction of a definition of PPH to 500 ml or 750 ml of blood loss is somewhat arbitrary and does not take into account other markers of hemodynamic compromise and the wide variation in maternal blood volume that can be lost without risking the patient’s life\textsuperscript{24}. As noted previously in this chapter and in many other chapters in this volume, the visual estimation of blood loss almost invariably results in underestimation\textsuperscript{47,48} (Chapter 9). Thus, while assessing severity of PPH was attempted in every cited study by including transfused cases only, different authors were unable to model risk factors due to the relatively few well documented cases of transfusion in the early years of the different surveys and the considerably broad variation in transfusion numbers over the periods the studies were carried out. Similarly, coexistence of coding indicating a hysterectomy had been used as a marker for severity, but here again too few studies attempting a multivariate analysis were found.

It is important to keep in mind that a diagnosis of PPH must be reported in the medical record by a clinician, obstetrician or midwife for a case to be coded as such by medical coders. Clinicians might be unwilling to record a diagnosis of PPH unless there are accompanying signs of compromise.

Regarding the issue of potential differences in the recording of PPH, the ICD-9 has a universal code for this diagnosis. Also, a single code is available for all types of retained, trapped and adherent placenta with hemorrhage. Considering that the necessity of separating codes for adherent placenta might be useful, given the increases in the frequency of cesarean delivery, such a code was added to the ICD-10. The Australian local modification introduced in 2002 (adding a code for morbidly adherent placenta, including placenta accreta, increta and percreta), enabled subsequent study on this population\textsuperscript{34}. One of the suggestions made by the International Collaborative Group was that future revisions of the ICD should include separate codes for atomic PPH and PPH immediately following childbirth due to other causes. Also, additional codes are required for placenta accreta/percreta/increta. This recommendation is supported by the fact that currently collected data do not allow adequate categorization of PPH according to severity; this deficiency inhibits the ability to determine outcomes for women with differing degrees of blood loss.

Despite the quite well described pathways for solution, for example by recording actual estimated blood loss using a simple blood collector bag\textsuperscript{48}, the different strategies to manage the third stage of labor, and the different guidelines pointing to the timing and route of prophylactic oxytocic administration, as well as surgical procedures and therapies undertaken to control PPH as fully described in the other chapters of this volume\textsuperscript{6–8,13,17,23,29,44}, the fact remains that PPH and uterine atony both are slightly and steadily increasing in their respective incidences.

Additional factors

[Editor’s note: The factors described below are also mentioned in greater or lesser detail in other chapters of this volume L.G.K.]

A number of factors appear to be acting in concert to influence the increased incidence of PPH in developed countries. In the populations cited above, maternal age at childbirth has been increasing significantly\textsuperscript{5,16,49}, and although, the vast majority of studies failed to demonstrate a clear impact of aging on PPH rates\textsuperscript{5,6,39}, increasing maternal age has been
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consistently described to be a substantial risk factor in all registers of obstetric hysterectomy for PPH. At the same time, in the UK, Australia and in the vast majority of the European countries, births of immigrant women are increasing, and the rates of severe maternal morbidity, although not specifically PPH, are higher in women from ethnic minority groups albeit not consistently. Also, the rising rates of obesity demonstrated in many countries may also impact on the incidence of PPH; an increased body mass index (BMI) is a reported risk factor for hemorrhage.

Cesarean delivery is not only more frequent globally, but also results in a higher blood loss when compared to normal delivery. Of interest, validation of data on PPH from NSW showed that there is significant under-recording of blood loss after cesarean delivery (60% of cesarean deliveries with recorded blood loss versus 96% of vaginal births). Additionally, post cesarean transfusion for low hematocrit or post cesarean section laparotomy for evacuation of hematoma are not captured as a part of the PPH code, which may explain the lack of increase in risk of PPH in women undergoing cesarean section in this population. Other studies have shown that the antecedent of a previous delivery by cesarean section is associated with increased risk of abnormal placenta, hemorrhage and peripartum hysterectomy. In Canada, where PPH following cesarean delivery by definition requires a blood loss over 1000 ml, cesarean delivery was shown to have a protective effect on atonic PPH (adjusted odds ratio 0.52).

Strikingly, the planned cesarean section seems to play a role in protection against PPH, and this has not been an isolated finding. While, modeling yielded similar results to models including all singleton deliveries and also tended to confirm the major contribution of vaginal/instrumental deliveries to PPH, it remains possible, however, that there is a considerable underestimation of blood loss at cesarean delivery and consequent under-enumeration of cesarean deliveries with PPH. Another possible explanation for the deceiving and apparently protective effect of cesarean section is that postsurgical patients may be under closer observation and are more likely to have early diagnosis of hemorrhage with earlier interventions than patients who deliver vaginally.

Another potential factor is the practice of labor induction, which is currently performed more often than in the past. In some areas, this practice was associated with significantly increased odds for PPH. In addition, after adjustment for mode of delivery, maternal age, birth weight and public/private admission status, the use of oxytocin infusion for augmentation also independently increased the odds for PPH (adjusted (a)OR 1.19). A population-based Norwegian study also reported an increase in the risk of severe PPH associated with induction of labor (aOR 1.71). Despite this, secondary analysis of data on peripartum hysterectomy to control PPH in the UK did not show an association with induction of labor even after adjustment for previous cesarean delivery.

Labor induction and other changes in obstetric practice may lead to an increased duration of labor, in both first and second stage, which may contribute to an increase in the frequency of PPH. An increasing duration of labor over time has been demonstrated in Victoria (Australia) as well as in Nova Scotia (Canada). The Nova Scotia study found an increase in the risk of PPH related to an increasing duration of the second stage of labor. A role for increased labor duration in PPH is also supported by the above mentioned UK study of hemorrhage-associated peripartum hysterectomy, which showed an independent association between peripartum hysterectomy and labor of 12 hours or greater duration. After adjusting for the effects of age, parity, previous cesarean section delivery, other uterine surgery and multiple pregnancy, the aOR remained at 3.04 (95% CI 1.52–6.08). This finding is also supported by data on atonic PPH in Canada, which show an increased risk after a prolonged first stage, prolonged second stage and prolonged labor (without specifying a definition).

Multiple pregnancy rates are also increasing; possible contributory factors include assisted reproductive techniques and, again, an aging population of women giving birth. Multiple pregnancy has been associated with an increased risk of PPH and associated complications in a number of studies, and the observed rise in the rate of multiple pregnancy may logically contribute to increasing PPH incidence. However, although there was a significant increase in multiple pregnancies in NSW between 1994 and 2002 (1.4–1.7% of all pregnancies), there was no significant change in the proportion of PPH among multiple pregnancies. The PPH rate among multiple pregnancies varied from 2.5% in 1994 to 3.1% in 1996 and 2002 (with an average rate of 2.9%), representing an increase of 183 pregnancies, which in the overall context of PPH risk could be considered as inconsequential.

A secondary analysis of Canadian PPH data showed that multiple pregnancy, even after adjustment for cesarean delivery, labor induction, maternal characteristics and obstetric practices, failed to explain the increase in PPH rates, although it did explain some of the increase in hysterectomy for PPH.

Finally, it is quite possible that the rise in PPH rates may be associated with risk factors not included in the different models cited previously, including chorioamnionitis, pyrexia in labor, overweight leading to peripartum obesity, duration of the third stage of labor, previous PPH and placenta accreta, although some studies have included retained placenta as a separate risk factor. The role it plays in PPH could not be investigated in the vast majority of publications, due to retained placenta being included in the definition of PPH in the ICD-9 coding.
Other potential risk factors that could have a possible role include, the increased use of oxytocin in the management of first stage of delivery\textsuperscript{24,27,100,101}, and increased rates of coagulation disorders in the puerperium when using dinoprostone for cervical ripening, although this risk seems to be low\textsuperscript{101}. However, when all these facts act together, even if only slightly, they can account for a change which has impacted on the overall risk of PPH.

Furthermore, changes in the management of the third stage of labor may have affected rates of PPH. There is clear evidence that active management of the third stage of labor reduces PPH when compared to physiological or expectant management. It also substantially, decreases bleeding and transfusion requirements as well as postpartum hysterectomies\textsuperscript{7,46,86,99,100,102,103}. Active management involves drug administration, early umbilical cord clamping and controlled cord traction, whereas expectant management involves waiting for signs of separation and allowing the placenta to deliver spontaneously or aided by gravity or nipple stimulation.

Over the period of these studies (1994–2002) and across the world, it is likely that variations in third stage management occurred. A Cochrane systematic review was not published until 2000 and state and national policies/recommendations regarding third stage management not introduced until 2002–2003. A more recent report indicates there has been considerable variation in active or expectant management, midwifery practices such as fundal massage and in the choice and dose of prophylactic drug used (i.e. ergometrine–oxytocin or oxytocin)\textsuperscript{30}. Oxytocin is now more commonly used than ergometrine–oxytocin, having fewer maternal side-effects; however, it has been shown to be slightly less effective in reducing the risk of PPH of 500–1000 ml\textsuperscript{12}. Misoprostol has been shown to play a role in controlling PPUA with higher success rates than oxytocin\textsuperscript{7,93,104,105}. Competing interests of mothers and babies have also complicated third stage management with delayed cord clamping recommended for preterm babies. Completed studies on management or length of the third stage of labor were not available at the time of this review\textsuperscript{35,91,106,107}.

Matters of controversy

Studies findings are not always consistent with the commonly held belief that increasing rates of PPH are due to changes in maternal characteristics and obstetric procedures. In particular, increased PPH rates were not consistently explained by increases in maternal age, cesarean section, multiple pregnancy, induction/ augmentation of labor or epidural use.

It is also noteworthy that, the ‘classic’ risk factors for PPH reflected in the recent literature corroborate those found in older series: large babies\textsuperscript{108,109}, placental abnormalities\textsuperscript{72}, induction\textsuperscript{36} and instrumental delivery\textsuperscript{38,110}, which were the strongest predictors of PPH after adjustment for other factors\textsuperscript{8,28,111,112}. Yet, those risk factors are claimed to be under control in the developed world.

So, if the increase in PPH rates is not related to increases in the proportion of cesarean deliveries, or trends in other risk factors, what is it due to? One possible explanation may be changes in reporting or ascertainment. Changes in reporting, such as the change from ICD-9 to ICD-10 in 1998 (which did not alter PPH coding), and the introduction of a statewide policy for management and reporting of PPH in 2002 do not coincide with observed increases in the PPH rate, which occurred primarily between 1994 and 1999. It is possible that hospitals may have been directed by state health authorities or their own administrators to vary their coding practice from the national/state standard for local reasons, thus introducing inconsistent practices between hospitals\textsuperscript{20,23,26,41,102}, although there is no reason to assume this is widespread or would have resulted in a false increase in reported cases.

Accounting for changes in risk factors

As we noted earlier in this chapter, attempts to explain the increase in PPH rates by taking into account changes in the observed risk factors for PPH over time cannot convincingly explain the rise in these rates. Ford \textit{et al.} investigated risk factors for any PPH among singleton deliveries in Australia over the period 1994–2002\textsuperscript{85}, while Joseph \textit{et al.} investigated risk factors for atonic PPH among deliveries in Canada over the period 1991–2004\textsuperscript{48}. Using different methods, the two studies took into account maternal age, parity, year of birth, country of birth, onset of labor, mode of delivery, epidual analgesia, abnormal labor (precipitate labor, incoordinate contractions, etc.), prolonged or obstructed labor, hypertensive disorders, placental abnormalities (placenta or vasa previa), placental abruption, gestational age, birth weight, perineal trauma, cervical laceration, previous cesarean, multiple pregnancy, polyhydramnios and amniotic cavity infection. Both studies concluded that although the frequency of many risk and protective factors for PPH changed during the studies period, controlling for these factors did not seem to alter temporal trends, suggesting factors other than those considered were responsible for the rising PPH rates.

The authors postulated that other factors such as a more liberal approach to duration of labor, which allows women to labor for longer (information which was not collected with sufficient detail in any of the series), increases in obesity (not recorded in all hospital records) or changes in the management of third stage of labor (not always recorded in hospital data) may play a part in rising PPH rates. Other possible risk factors merit further investigation and should include the effect of induction of labor – taking into account agents used – or the infinitely more complex interactions of risk factors such as the interplay between body mass index, oxytocin agents or misoprostol
CONCLUSION AND KEY RECOMMENDATIONS

The incidence of PPH seems to be rising in the industrialized countries. In spite of multiple possible confounding factors, this appears as a real and concerning problem. An important number of risk factors could be considered as directly involved; however, none accounts substantially for the situation. The most likely scenario is a gathering of all these factors, which leads to this increase as the final outcome.

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