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Obstetric vesico-vaginal fistula is preventable by timely recognition of prolonged labour and identification and management of post-partum urinary retention

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Abstract

Obstetric vesico-vaginal fistula is still prevalent in developing countries at an incidence of about 150,000 cases annually. This is mainly the result of a poorly developed health care delivery system where a majority of women labour away from the care of trained birth attendants. When ineffective uterine contractions occur and it is not corrected, prolonged labour results in the fetal head impacting deeply in the pelvis causing field injury due to pressure ischaemia-necrosis. Bladder care is least attended to in the settings where obstetric fistula is prevalent and this is likely to result in post-partum urinary retention. Overt or covert urinary retention when not corrected in a timely manner causes the ischaemic tissues not to heal resulting in fistula formation. Prevention of obstetric fistula should include universal access to maternity care, recognition and timely correction of abnormal progress of labour and punctilious attention to bladder care to avoid post-partum urinary retention.

Key words: Obstetric fistula, Risk factors, Pathophysiology, Post-partum urinary retention

Introduction

Obstetric vesico-vaginal fistula is an acquired defect resulting in abnormal communication between the lower genital and urinary systems causing continuous or intermittent urine incontinence that has far reaching implications for the woman (1). Descriptions of this agonising condition are found in medical literature dating back several centuries with the first successful treatment attributed to the work of James Marion Simms in the 19th century. The aetio-pathogenesis of obstetric vesico-vaginal fistula is related to complex socio-economic matrix resulting in neglected labour.

The burden of obstetric fistula is borne by developing countries of Africa and Asia with prevalence estimates in the region of 3 million women having untreated fistulae and about 30,000 – 150,000 developing new cases each year (2). The WHO in Global Burden of Disease study shows that 6.5 million of seven million women affected by obstructed labour annually live in least developed countries. In sub-Africa, this study estimated that of these women with neglected obstructed labour 2.15% would develop a fistula yielding a prevalence of 1.88 cases per 1000 women aged 15 – 44 years (3). In a more recent meta-analysis of national household survey data, Maheu-Giroux and colleagues (4) estimate in 19 sub-Saharan African countries that the lifetime prevalence of vaginal fistula is about 3 per 1000 women aged 15 - 44 years (95% CrI 1.3 – 5.5) and a point prevalence of 1.0 cases per 1000 women of reproductive age (95% CrI 0.3 – 2.4). Ninety percent reported the fistula to be pregnancy related.

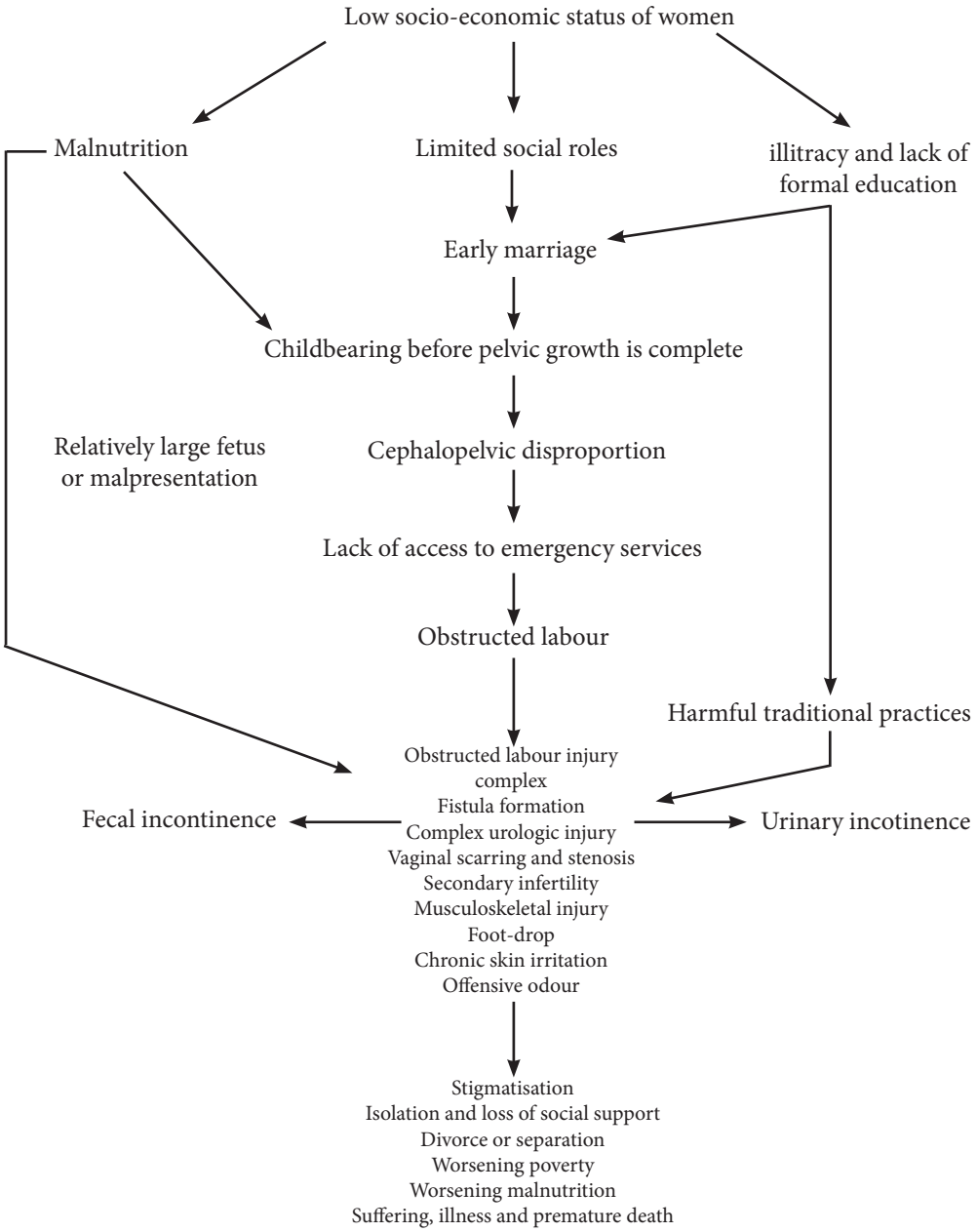
In the developed world obstetric fistula is virtually unheard of following its successful elimination between 1935 – 1950 in Europe and USA. This is largely due to universal access to safe maternity care, which is reflected in the lower obstetric related mortality and morbidity. For instance in Sweden the lifetime risk of dying from a pregnancy related cause is one in 29,800 but disproportionately one in six in under privileged Africa and Asia (5).

Pathophysiology

It is accepted that obstetric fistula results from obstructed labour where there is cephalo-pelvic disproportion and inability of the fetus to safely navigate through the birth canal. As uterine contractions impact the fetal head deeply into the pelvic cavity, there is resultant pressure necrosis of the soft tissues of the vagina and bladder trapped between the bony pelvis and fetal head. Arrowsmith *et al* (6) describe this as a 'field injury' to a broad area. Unlike bladder injury resulting from surgical misadventure for instance a stitch placed as a ligature inadvertently travelling through the bladder causes a localised trauma with a narrow area of fistula formation. However, this injury is much broader affecting many tissues in the field. It should be

borne in mind, however, that obstruction must mean failure to progress due to mechanical problems (7). As Arrowsmith *et al* (6) detail in their description, the field injury is not only limited to causing fistula formation but also results in more devastating complications including renal failure, neuromuscular disability, vaginal stenosis and infections of the pelvic pubic bone and the upper genital tract. Some women also experience amenorrhoea with secondary infertility. Substantially, a higher risk of maternal mortality from uterine rupture and sepsis as well as fetal death due to asphyxia are reported in these patients (8,9). It is believed that obstructed labour and its sequelae result from the panoply of biological and socioeconomic factors as shown in Figure 1.

Figure 1: Biological and socio-economic factors that interplay resulting in obstetric fistula formation and its sequelae: Adapted from Worldwide Fistula Fund with permission (2).



Epidemiological studies have shown that primiparity, young age at first pregnancy, lower educational attainment in addition to cultural factors and a poorly developed health care system are at the core of this complication. When prolonged labour results from a lack recognition or delay in intervention, this greatly predisposes the woman to the risk of developing obstetric fistula (10-12). Tebeu *et al*. (10) in a systematic review, show that 57.5% to 94.8% of women with obstetric fistula labour at home and are then transferred to hospital. They have a mean duration of labour lasting about 2.5 to 4 days. Ninety five percent of patients laboured more than 24 hours and operative delivery was performed in 11% to 60% of the cases (10). The majority of studies on obstetric fistula are from Africa and show that the greater proportion of women having been younger than 25 years when they suffered prolonged neglected labour. Malnutrition

in childhood with rickets as a consequence distorts the bony architecture of the pelvis which does not bode well that labour and delivery will be successful vaginally. In addition a majority of teenage girls have the bony pelvis immature and should they be pregnant and went into labour, it is unlikely that the labour will lead to a successful vaginal birth (13).

In a large series reported from Nigeria, Wall *et al* (12) noted that obstetric fistula occurred in women with no formal education, were more likely to be teenagers at the index pregnancy resulting in fistula and lived in rural areas where they had no or poor access to hospital care. This has been replicated in other studies from across Africa (14-15).

The nature of labour in obstetric fistula

Normal labour is characterised by the onset of regular and frequent uterine contractions together with changes to the cervix of effacement and progressive dilatation culminating in successful delivery of the fetus as well as expulsion of the placenta. It requires that the character of uterine activity and changes to the cervix are assessed by a trained birth attendant to assess progress and timely detect an abnormal pattern. The efficiency of uterine activity is maintained through positive feed-forward mechanism between the posterior pituitary gland secretion of oxytocin and relay from the uterus through the autonomic nervous system (7). Labour is an energy intensive endeavour for the smooth muscle of the uterus that has to be sustained for hours before the fetus is born (16,17). This process necessitates a complex interplay of molecular interactions between endocrine and cytokine systems and may be affected by acidosis, ketosis, dehydration and catecholamine concentration leading to slowing down of uterine activity manifesting itself as prolonged labour (7).

The definition of prolonged labour was the subject of controversy until the advent of the cervicograph as described by Friedman in 1954. Friedman in his work on labour demonstrated that normal cervical dilatation occurred at the rate of 1 cm/hour in the active phase of labour, and represented this as a sigmoid curve. This was however modified by Philpott and Castle and presented pictorially as a straight line, representing the slowest 10th percentile of women in labour. This also served as the alert line (20). Recently, it has been agreed that a rate of 0.5 cm/hour represents the normal rate of cervical dilatation (18). There have been several modifications to the partograph, as previously it had the latent phase of labour included more recently the WHO partograph has only the active phase (19). At present, it is the consensus that labour lasting longer than 12 hours be considered prolonged (18).

On the partograph a parallel line drawn 4 hours to the right of the alert line serves as the action line. When labour slows down, its curve starts to falter and may cross the action line. In Philpott's initial model the partogram was designed to be used in peripheral midwifery led units with an alert line 2 hours to the right and parallel to the normal line where arrangements were made to transfer the woman to the regional hospital if her labour curve crossed this line. On the partogram, a further parallel line four hours to the right of the alert line was added as the action line. When this line was crossed, a diagnosis of dysfunctional labour was made prompting appropriate management (20). The commonest reason for dysfunctional labour especially in primiparous women is inefficient uterine activity. However, careful consideration should be given to other causes like cephalo-pelvic disproportion or abnormalities of presentation and lie (21). Once dysfunctional labour is diagnosed, interventions such as amniotomy and/or oxytocin may be administered to correct the dystocia. O'Driscoll and his group at the National Maternity Hospital in Dublin, Ireland had shown that their regime of active management of labour resulted in a reduction in the caesarean section rates. This was neither confirmed in a subsequent study with a similar methodology nor in a recent Cochrane review where oxytocin was compared to placebo in the treatment of dysfunctional labour (21-23). However, Bugg *et al*. showed in their meta-analysis that oxytocin did shorten the duration of labour by a mean of -2.2 hours (95% CI -3.29 to -1.10) (21).

The nature of uterine activity in labour dystocia tends to be dissimilar between primiparous and multiparous women with the former showing a diminishing pattern of contractions that is not characteristic of the latter (7). It is hypothesised to be due to myometrial acidification that results from anaerobic metabolism, dehydration and ketosis. These conditions stop contractions in primiparous and not in multiparous women who may be resistant to their effects due to unknown mechanisms (7). As we have seen from the characteristics of women who develop obstetric fistula, they are more likely to be in their first pregnancy, labouring at home miles away from medical assistance with no monitoring. They are likely to be disallowed food or drink on socio-cultural grounds resulting in prolonged neglected labour (23). When the course of labour halts due to diminished uterine activity, the fetal head remains fixed within the pelvis exerting prolonged pressure to neighbouring maternal soft tissues against the bony pelvis. The bladder is more vulnerable to injury as it continues to fill with urine resulting in elevation of the bladder neck and inability to void. Intrapartum bladder over-distension was demonstrated to correlate with the incidence of postpartum urine retention, a risk that increases with the duration of both first and second stages of labour (24). Soft tissue pressure ischaemia-necrosis of the bladder is not likely to heal spontaneously in these women with undiagnosed or untreated urinary retention and it will result in fistula formation. This is a significant last step

in fistula formation, though it is hardly mentioned in the literature.

Prevention of obstetric vesico-vaginal fistula

Prevention of obstetric vesico-vaginal fistula may be approached in four different ways as follows. Firstly, by identification of women at risk. Secondly educating and encouraging women to labour and deliver under the care of trained birth attendants. Thirdly, by recognition of abnormal progress of labour and timely intervention to correct dystocia. Lastly, meticulous bladder care during labour and the immediate post-partum period.

Many epidemiological studies consistently show that young, illiterate women who have none or minimal access to family planning, prenatal care or skilled birth attendant are mostly at risk of obstetric fistula formation. They are likely to labour at home with labour lasting on average 2.5 to 4 days without recourse to emergency obstetric care. Where they eventually get transferred to hospital perinatal mortality is almost invariable and severe obstetric morbidity including fistula development is common sequelae (12,14,25). Tebeu e*t al* (10), have comprehensively reviewed these risk factors and conclude that they are entirely preventable. As Wall (2) points out, obstetric fistula is a complex interplay between biological, social and economic factors. To overcome these, advocacy and multisector collaboration between government and international organisations like Engender Health and Worldwide Fistula Fund Initiative may provide a long term solution. Eventually, access to universal emergency obstetric care as was characterised by the disappearance of obstetric fistula in the developed world will be the ultimate solution.

It is also clear from many observational studies that 10% to 85% of women with obstetric fistula will have laboured at home with an average duration of labour between 2.5 to 4 days (14,26-28). Thaddeus and Maine (29) discuss the classic three phases of delay in seeking emergency obstetric care and summarise these as delay in deciding to seek care; delay in arriving; and delay in receiving appropriate care. As discussed extensively by Wall (2), overcoming phase one delay will involve a complex understanding of the socio-cultural context of individual women often as community 'norms' where the biomedical option of care is in competition with alternative pluralistic choices. Community education, antenatal care and an efficient health care delivery system should incentivise women to seek help when a problem is suspected. Governments may borrow from the case of El-Salvador to construct health facilities, provide ambulances and link them with radio-communication to allow faster access to emergency obstetric services (30).

Recognition of abnormal progress of labour and taking timely intervention is crucial in the prevention of obstetric fistula and other morbidity. Involvement of well-informed community players and raising their awareness for the recognition of labour and referral to a trained birth attendant will deliver in safe environments. Once in a health facility trained birth attendants should be able to recognise dysfunctional labour by use of the partograph and judiciously use oxytocin to correct dystocia. In a large WHO study involving 35,484 women in Asia the partograph was able to reduce prolonged labour, need for augmentation and reduced risk of sepsis (31). The health care practitioner should be consciously aware that use of oxytocin in *obstructed* labour would be counterintuitive. In a recent Cochrane review, partograph use in low resource setting resulted in lower caesarean section rates (RR 0.38, 95% CI, 0.24 - 0.61) (19). A related Cochrane review concluded that use of early oxytocin resulted in a statistically significant difference in the mean duration of labour of about 2 hours but did not increase normal delivery rates (21). It has to be remembered that a majority of studies included in these meta-analyses are not from low resource countries, settings where healthcare dynamics is different. These interventions are part and parcel of the package of basic obstetric care that also encompasses instrumental vaginal delivery. Certainly caesarean section would not be available at basic level, but use of a partogram with a transfer line comes in handy as demonstrated by Philpott and Castle (7) for timely transfer to a comprehensive obstetric care facility.

Meticulous bladder care intrapartum and in the immediate post-partum period should ensure that postpartum voiding dysfunction is avoided. Post-partum urinary retention is not uncommon and may affect up to 14% of women (32). This is categorised into overt retention where the woman is unable to void spontaneously and covert where there is an elevated post-void residual volume of 150ml or greater. A number of well-known risk factors predispose to postpartum urinary retention including prolonged labour, use of regional anaesthesia, instrumental delivery, caesarean section, perineal trauma and primigravidity (33). Nekre *et al* (34) were able to demonstrate that labour lasting longer than 11 hours 40 minutes was significantly associated with post-partum urinary retention. The mechanisms involved in the development of postpartum urinary retention are multiple. These could be related to inattention to bladder care in labour leading to over-distension, physiological changes of pregnancy that predispose to detrusor hypotonia, mechanical trauma to the pudendal nerve that disrupts bladder and pelvic floor innervation and perineal oedema (35). Prompt recognition and treatment of postpartum urinary retention prevents long term sequelae on bladder function (33). At risk women should be identified and evaluated to exclude the diagnosis. If a woman

has not passed urine after 6 hours following vaginal delivery or from removal of catheter following caesarean section, she should be suggested measures to encourage spontaneous voiding and if unable to, then catheterisation should be offered. Where there has been successful spontaneous void, post void residual should be measured. Standard intrapartum care now involves greater attention to bladder care which aims to reduce the risk of postpartum bladder dysfunction.

Unfortunately, data on post-partum urinary retention in women with obstetric fistula is lacking. It is however possible to extrapolate that the incidence of urinary retention in women with prolonged labour that ends in fistula is far greater than14% reported in other settings (32). All the characteristics that predispose to fistula formation are indeed risk factors for urinary retention. The global field injury that describes damage causing fistula is likely to be accentuated by urinary retention. Where ischaemic injury to the bladder would have healed with immediate recognition and relief of urinary retention, this goes unrecognised resulting in the distended bladder encouraging necrosis and fistula formation.

Conclusion

Obstetric fistula is prevalent in developing countries with an incidence of about 150,000 cases each year. Obstetric fistula has been eliminated from the developed world due to universal maternity care. In developing countries however, poor health care infrastructure, young age at first pregnancy, lack of education and a failure to recognise prolonged labour and/ or delayed medical intervention often end in obstetric fistula formation. It is likely that intrapartum and post-partum urinary retention is the final event in the pathophysiology of obstetric fistula resulting from the field injury inflicted on pelvic soft tissues. Obstetric fistula is totally preventable. Effective prevention strategies should focus on strengthening health system infrastructure through multi-sectoral collaboration, extensive public education and continued education of healthcare providers. These should address the three phases of delay in seeking emergency maternity care. Emphasis must be laid on recognition of dysfunctional labour, its correction and particularly punctilious attention to bladder care with proactive seeking, diagnosis and treatment of post-partum urinary retention.

Conflict of interest

The authors have no conflict of interest to declare

References

1. Ahmed, S. and Holtz, S.A. Social and economic consequences of obstetric fistula: life changed forever? *Int J Gynaecol Obstet.* 2007; **99** (suppl 1)**:** S10–15.
2. Wall, L.L. Obstetric vesicovaginal fistula as an international public-health problem. *Lancet.* 2006; **368:** 1201–1209.
3. Abou-Zahr, C. Prolonged and obstructed labour. In: Murray, C., Lopez, A., eds. Health dimensions of sex and reproduction: the global burden of sexually transmitted diseases, HIV, maternal conditions, perinatal disorders and congenital anomalies. Cambridge: Harvard University Press for WHO, 1998: 243–66.
4. Maheu-Giroux, M., Fillipi, V., Samadoulougou, S., *et al.* Prevalence of vaginal fistula symptoms in 19 sub-Saharan African countries: a meta-analysis of national household survey data. *Lancet Glob Health.* 2015; **3:** e271–278.
5. WHO. Maternal mortality in 2000: estimates developed by WHO, UNICEF and UNFPA. Geneva: World Health Organization, 2004.
6. Arrowsmith, S., Hamlin, E.C. and Wall, L.L. Obstructed labor injury complex: obstetric fistula formation and the multifaceted morbidity of maternal birth trauma in the developing world. *Obstet Gynecol Surv*. 1996; **51**(9):568-74.
7. Neilson, J.P., Lavender, T., Quenby, S. and Wray, S. Obstructed labour. *Br Med Bull.* 2003; **67:** 191–204.
8. Orach, C.G. Maternal mortality estimated using the sisterhood method in the Gulu district, Uganda. *Trop Doct.* 2000; **30**: 72–74.
9. Prual, A., Bouvier-Colle, M.H., de Bernis, L. and Breart, G. Severe maternal morbidity from direct obstetric causes in West Africa: incidence case fatality rates. *Bull World Health Organ.* 2000; **78:** 593–602.
10. Tebeu, P.M., Fomulu, J.N., Khaddaj, S., de Bernis, L., Delvaux, T. and Rochat, C.H. Risk factors for obstetric fistula: a clinical review. *Int Urogynecol J.* 2012; **23**(4):387–394.
11. Blair, B.W., Raker C.A., Kabeja G.A., Kay, A. and Brittany, S. Hampton demographic and delivery characteristics associated with obstetric fistula in Kigali, Rwanda. *Int J Gynecol Obstet.* 2015; **129** 34–37.
12. Wall, L.L., Karshima, J.A., Kirschner, C. and Arrowsmith, S.D. The obstetric vesicovaginal fistula: characteristics of 899 patients from Jos, Nigeria. *Am J Obstet Gynecol.* 2004; **190**(4):1011–9.

13. Chang, S.C., O'Brien, K.O., Nathanson, M.S., Mancini, J. and Witter, F.R. Characteristics and risk factors for adverse birth outcomes in pregnant black adolescents. *J Pediatr.* 2003; **143**(2): 250–257).
14. Hilton, P. and Ward, A. Epidemiological and surgical aspects of urogenital fistulae: a review of 25 years' experience in southeast Nigeria. *Int Urogynecol J Pelvic Floor Dysfunct.* 1998; **9**(4):189–194.
15. Rijken, Y. and Chilopora, G.C. Urogenital and recto-vaginal fistulas in southern Malawi: a report on 407 patients. *Int J Gynaecol Obstet.* 2007; **99**(Suppl 1):S85–S89.
16. Sanborn, B.M. Hormones and calcium: mechanisms controlling uterine smooth muscle contractile activity. The Litchfield Lecture. *Exp Physiol.* 2001; **86**: 223–237.
17. Gimpl, G. and Fahrenholz, F. The oxytocin receptor system: structure, function, and regulation. *Physiol Rev.* 2001; **81**: 629–683.
18. NICE 2008 National Collaborating Centre for Women's and Children's Health. Intrapartum care: management and delivery of care to women in labour. National Institute for Clinical Excellence (NICE). http://guidance.nice.org.uk/CG55 Issued: September 2008. *Accessed August 10, 2015.*
19. Lavender, T., Hart, A. and Smyth, R.M.D. Effect of partogram use on outcomes for women in spontaneous labour at term. Cochrane Database of Systematic Reviews 2013, Issue 7. Art. No.: CD005461. DOI: 10.1002/14651858.CD005461.pub4.
20. Philpott, R.H. and Castle, W.M. Cervicographs in the management of labour in primigravidae. *J Obstet Gynaecol Brit Commonwealth.* 1972; **79**:599–602.
21. Bugg, G.J., Siddiqui, F. and Thornton, J.G. Oxytocin versus no treatment or delayed treatment for slow progress in the first stage of spontaneous labour. *Cochrane Database of Systematic Reviews* 2013, Issue 6. Art. No.: CD007123. DOI:10.1002/14651858.CD007123.pub3.
22. O'Discoll, K., Jackson, R.J. and Gallagher, J.T. Prevention of prolonged labour. *BMJ* 1969; **2**(5655):477–480.
23. Frigoletto, F.D. Jr, Lieberman, E., Lang, J.M., Cohen, A., Barss, V., Ringer, S. and Datta, S. A clinical trial of active management of labor. *New Engl J Med*. 1995; **333**(12):745–750.
24. Yip, S.K., Hin, L.Y. and Chung, T.K. Effect of the duration of labor on postpartum post void residual bladder volume. *Gynecol Obstet Invest*. 1998; **45**(3):177-180.
25. Chang, S.C., O'Brien, K.O., Nathanson, M.S., Mancini, J. and Witter, F.R. Characteristics and risk factors for adverse birth outcomes in pregnant black adolescents. *J Pediatr.* 2003; **143**(2):250–257.
26. Arrowsmith, S.D. Genitourinary reconstruction in obstetric fistulas. *J Urol*. 1994; **152**(21):403–406.
27. Nafiou, I., Idrissa, A., Ghaichatou, A.K., Roenneburg, M.L., Wheeless, C.R. and Genadry, R.R. Obstetric vesico-vaginal fistulas at the National Hospital of Niamey, Niger. *Int J Gynaecol Obstet.* 2007; **99** (Suppl 1):S71–S74.
28. Holme, A., Breen, M. and MacArthur, C. Obstetric fistulae: a study of women managed at the Monze Mission Hospital, Zambia. *BJOG.* 2007; **114**(8):1010–1017.
29. Thaddeus, S. and Maine, D. Too far to walk: Maternal mortality in context. *Soc Sci Med.* 1994, **38**:1091–1110.
30. Wall, L.L. Overcoming phase 1 delays: the critical component of obstetric fistula prevention programs in resource-poor countries. *BMC Pregnancy and Childbirth.* 2012; **12**:68.
31. World Health Organization Maternal Health and Safe Motherhood Programme. World Health Organization partograph in management of labour. *Lancet.* 1994; **343**: 1399–404.
32. Lee, S.N.S., Lee, C.P., Tang, O.S.F. and Wong, W.M. Postpartum urinary retention. *Int J Urogynecol Obstet.* 1999; **66**:287–288.
33. Chaliha, C. Postpartum bladder dysfunction. *Reviews Gynaecol Perin Practice.* 2006; **6**: 133–139.
34. Aruna, N., Vijayanand K.S., Dasgupta, R. and Kekre N. Postpartum urinary retention after vaginal delivery. *Intern J Gynecol Obstet*. 2011; **112:** 112–115.
35. Sultan, A.H., Kamm, M.A. and Hudson, C.N. Pudendal nerve damage during labour: prospective study before and after childbirth. *Br J Obstet Gynaecol.* 1994; **101**:22–28.