We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Challenges Facing during Pregnancy and Measures to Overcome

*Gayatri Devi Ramalingam, Saravana Kumar Sampath
and Jothi Priya Amirtham*

Abstract

Pregnancy is a time of transformation for both the mother and the baby, with significant physical and emotional changes. There are many discomforts that occur during pregnancy. Morning sickness, headache and backache, bladder and bowel changes, changes in hair and skin colour, indigestion and heartburn, leg cramps and swelling, vaginal thrush and discharge are the few common complications facing during pregnancy. As a result, the aim of this study was to describe the difficulties in obtaining health information and the measures to overcome the discomfort during pregnancy. Research articles for this review were searched by using the keywords “pregnancy”, “health issues”, “measures to overcome”, “challenges”. There were studies that looked at the health problems that women face during pregnancy were included in this review article. Pregnancy issues such as gestational diabetes mellitus, hypertension, preeclampsia, caesarean birth, and postpartum weight retention are all more likely in overweight and obese women. More research into the link between nutritional advancements and the rising prevalence of GDM in the developing world is needed. Iron supplementation has been linked to glucose dysregulation and hypertension in mid-pregnancy; its effectiveness and potential risks should be carefully considered. As a result, legislators and health planners should remove barriers, promote self-care, and improve the quality of life for pregnant women, ultimately improving their health.

Keywords: Pregnancy, management, gestational diabetes, Preeclampsia, Anaemia

1. Introduction

Pregnancy is a time of transformation for both the mother and the baby, with significant physical and emotional changes. Even in uncomplicated pregnancies, these improvements can impact pregnant women’s quality of life as well as maternal and child health. Women’s wellbeing, as well as their current level of understanding and knowledge, would undoubtedly have a significant impact on society [1]. Pregnant women need health information to improve their self-care skills and increase their empowerment when following preventive health habits. The cardiovascular system undergoes various changes as a result of pregnancy. A normal, healthy pregnant woman’s blood volume rises by nearly 50% over that of a non-pregnant woman. In addition, due to the vascular permeability associated

with extreme preeclampsia, efforts to increase blood volume in these patients have been unsuccessful [1, 2]. Pregnancy (critical care scenario) is the reduction in venous return to the heart and decrease in cardiac output associated with the supine position. This effect is obviously more pronounced in the third trimester, when the uterus is largest. The so-called supine hypotensive syndrome.

When treating a critically ill pregnant woman, various hematologic changes must be taken into account. During pregnancy, the thrombocyte count remains largely constant, it leads to thrombocytosis. These improvements, when combined with a decrease in fibrinolysis, lead to the hypercoagulable state of pregnancy. Deep vein thrombosis and pulmonary embolism are five times more common during and immediately after pregnancy [3]. Both the residual volume and the expiratory reserve volume decrease, resulting in an obligate reduction in functional residual capacity. During pregnancy, the vital ability remains the same. Because of the reduction in residual volume, total lung capacity is only slightly reduced. The reduction in residual volume has a minor impact on total lung capacity. The tidal volume is raised, resulting in an increase in minute volume [4]. Early in pregnancy, both renal plasma flow and glomerular filtration rate rise. The increase in glomerular filtration rate reaches 50%, lowering serum creatinine to 0.8 mg/dL, the upper limit of average. Only a few changes in the gastrointestinal tract during pregnancy are essential in terms of critical care. The time it takes for the stomach to clear and the chance of aspiration that comes with general anaesthesia are both increased during labour. Placental development may cause a significant increase in alkaline phosphatase, but this does not mean hepatic obstruction. Gallbladder stasis can lead to increased stone formation.

The liver is the primary source of net endogenous glucose development while not pregnant. Fasting glucose levels in pregnant women decline as the pregnancy progresses [5]. GDM is characterised as the presence of glucose concentrations in pregnant women that are at the upper end of the population range for glucose and are first observed during pregnancy [6]. Insulin sensitivity decreases overall during pregnancy. Maternal insulin sensitivity, characterised as a decrease in the glucose infusion rate during the euglycemic hyperinsulinemic clamp to maintain 90 mg/dL, decreases in lean women during early pregnancy [7]. Since lean women are more likely to begin their pregnancies with greater insulin sensitivity than obese women, the increases in insulin concentration are more pronounced in lean women [8]. During pregnancy, healthy pregnant women's adipose tissue stores increase significantly. The mother and foetus can easily obtain calories from the subcutaneous stores, particularly during late pregnancy and lactation. Increases in visceral fat can be linked to decreased insulin sensitivity during late pregnancy [9].

In their research, Das and Sarka found that pregnant women faced a variety of difficulties when seeking health information, including inadequate hospital treatment, long wait times, anxiety and shame about discussing pregnancy with a physician, and a lack of time [10]. There are many discomforts that occur during pregnancy. Morning sickness, headache and backache, bladder and bowel changes, changes in hair and skin colour, indigestion and heartburn, leg cramps and swelling, vaginal thrush and discharge are the few common complications facing during pregnancy. As a result, the aim of this study was to describe the difficulties in obtaining health information and the measures to overcome the discomfort during pregnancy.

2. Materials and methods

Research articles for this review were searched by using the keywords “pregnancy”, “health issues”, “measures to overcome”, “challenges”. The following

were used as exclusion criteria: 1) the subject was unrelated to the study's goal; 2) there was no abstract available; 3) The research was limited to a single medical issue involving pregnancy in older women. 4) The report dealt with postpartum and maternity issues; and 5) the full research paper was not easily accessible. We looked for original research papers that were written in English and reported on studies that were performed using qualitative or quantitative methods. The current research did not include any other review papers. There were studies that looked at the health problems that women face during pregnancy were included in this review article.

3. Dental problems

According to previous literature, pregnant women's dental health care demands differ dramatically from those of the general population. The most frequent oral health concerns during pregnancy include periodontal disease, Xerostomia, halitosis, and tooth movement. During pregnancy, the hormonal balance of pregnant women alters. Because the placenta produces increased levels of oestrogen and progesterone during pregnancy, several tissues endure modifications. Increased sensitivity to irritations arises in the gingiva during this time [11]. Low vitamin C levels are thought to be another cause of this condition. When compared to mothers with healthy periodontium, mothers with attachment loss have an increased risk of giving birth to babies with low birth weight [12]. Tooth decay is more common among pregnant women for a variety of reasons, including increased acidity in the mouth, sweet food demands, and a lack of attention to oral health. Vomiting can have a severe impact on oral hygiene and induce degradation of the mother enamel layer [13]. Due to the effect of pregnancy hormones, pregnant women bleed more easily and may postpone brushing their teeth and it leads to an increase in bacterial plaque [14]. Due to diminished flow of saliva, caries are more likely to develop at this time. Pregnancy oral tumour is indistinguishable from pyogenic granuloma and occurs in up to 5% of pregnancies. Increased progesterone, in combination with local irritants and microorganisms, causes this vascular lesion [15]. With a prevalence of 60 to 75 percent, gingivitis is the most frequent dental illness among pregnant women. A severe aggravation of preexisting gingivitis occurs in around half of all pregnant women [16]. Researchers discovered very few oral bacteria in the amniotic fluid and placenta of women who had preterm labour with periodontitis in one investigation [17]. PGE2 production reduces placental blood flow, resulting in placental necrosis and intrauterine growth restriction [18]. Salivary oestrogen levels are greater in women who are expecting preterm babies than in women who are expecting full-term babies. Salivary oestrogen promotes oral mucosa proliferation and desquamation, as well as a rise in subgingival crevicular fluid levels. Desquamating cells offer a favourable environment for bacterial growth by supplying nutrients, hence preventing infection [19].

3.1 Management

Oral acid exposure is reduced through dietary and lifestyle changes, as well as the use of antiemetics, antacids, or both. Acid can be neutralised by rinsing the mouth with a teaspoon of baking soda in a cup of water after vomiting [20]. To lessen the risk of enamel damage, pregnant women should be encouraged to avoid brushing their teeth shortly after vomiting and to brush with a toothbrush with soft bristles when they do. Fluoride mouthwash can protect teeth that have been eroded or are sensitive. Proper dental hygiene can help women with previous periodontal

disease lower the risk of recurrence or worsening disease during pregnancy. Education, clear communication, and the creation of continuing collaborative relationships can help physicians and dentists solve this dilemma. Physicians and dental colleagues can communicate information about the safety of dental treatment during pregnancy [21]. There is a link between plaque accumulation and caries prevalence during pregnancy and preventive maintenance methods. Mouthwashes or warm salty water should be gargled. Gums are relaxed and gum sensitivity is reduced by drinking warm salty water [22]. During this time, women can maintain their oral health by taking the required precautions, preventing potentially irreversible tooth disorders.

4. Hypertension

The mother's cardiovascular physiology adapts significantly as a result of the hormonal changes that occur during pregnancy [23]. Oestrogen, progesterone, and relaxin levels rise early in the first trimester, resulting in systemic vasodilation [24]. The RAAS is activated to promote salt and water retention, resulting in an increase in plasma volume. When this is paired with an increase in ventricular wall mass, it results in greater stroke volume. During pregnancy, the combination of increased stroke volume and tachycardia causes an increase in cardiac output, which compensates for the decrease in vascular resistance in order to keep blood pressure high enough for mother and placental perfusion [25]. The increased volume load in the heart causes left ventricular hypertrophy, which is proportional to the increased cardiac labour necessary to accomplish the increased cardiac output [26]. Some changes in the systemic hemodynamics of pregnant women who are predisposed to hypertension may occur before the condition manifests itself clinically. A systolic blood pressure of 160 mmHg or a diastolic blood pressure of 110 mmHg, or both, indicates severe preeclampsia in pregnancy. Eclampsia is a severe form of pregnancy-induced hypertension that affects one in every 1,600 pregnancies and appears at the end of the pregnancy [27]. When compared to singleton pregnancies, twin pregnancies had more than three times the risk of developing hypertension during pregnancy [28]. Preeclamptic patients have lower renin levels than non-pregnant women, although they are still significantly higher than non-pregnant women. Because most preeclamptic individuals have a somewhat reduced plasma volume, maintaining relatively high levels of these hormones may be necessary [29].

Preeclampsia is the pathophysiology of de novo hypertension and proteinuria in pregnancy. The delivery of the placenta frequently triggers the remission of preeclampsia's acute clinical symptoms, implying that the placenta plays a key role in the disease's pathophysiology. The placenta undergoes substantial blood supply throughout normal pregnancy to allow circulation between the foetus and the mother [30]. The pathogenesis of preeclampsia has long been focused on altered uteroplacental blood flow. Relaxin is a hormone secreted more during pregnancy which acts as vasodilation. According to Jeyabalan et al., low first trimester relaxin concentrations were linked to an increased risk of preeclampsia [31].

4.1 Management

For non-severe hypertension in pregnancy, oral labetalol is a first-line treatment [32]. Other beta-blockers, such as oxprenolol, are less thoroughly studied than labetalol, and it is used as a first-line treatment for non-severe hypertension in pregnancy [33]. In contrast, when oxprenolol was compared to methyldopa, the results and safety were found to be equal [34]. When mothers are exposed to

calcium channel blockers during the first trimester, there is low teratogenicity [35]. Elsewhere in pregnancy, ACE inhibitors are still the first-line treatment for hypertension [36]. For non-severe hypertension, thiazide diuretics are considered second-line therapy.

5. Gestational diabetes (GDM)

The prevalence of gestational diabetes mellitus (GDM) is rising in lockstep with the rise in overweight and obesity among women of childbearing age. GDM-affected pregnancies increase the risk of caesarean and surgical vaginal delivery, macrosomia, neonatal hypoglycemia, and hyperbilirubinemia for both mother and child [37]. The onset of GDM is linked to a number of risk factors. Obesity, advanced maternal age, a significant family history of diabetes and belonging to an ethnic group are with a high prevalence of T2DM, polycystic ovarian syndrome, and chronic glucosuria. Because GDM usually starts in the late second trimester, when development is complete, congenital abnormalities do not occur at a higher rate in people with gestational diabetes. Because of the physiological, endocrine, and metabolic changes that occur throughout pregnancy in order to meet the fetus's constant nutritional and oxygen needs, a diabetogenic condition comparable to type 2 diabetes (T2D) develops, increasing insulin resistance, lowering insulin sensitivity, and thus increasing the demand for insulin [38]. As a result of the increased placental glucose transport, maternal hyperglycemia causes foetal hyperinsulinemia. Foetal macrosomia is caused by a high insulin level in the foetus, which accelerates growth [39]. Although glucose metabolism changes during pregnancy, tolerance occurs and there is no effect on the mother or the foetus when insulin production rises. There is a higher risk of foetal when there is an inappropriate response. When the output of the pancreatic β -cells does not match the insulin requirement of the tissues as a result of alterations in insulin resistance, abnormal glucose tolerance ensues [40]. Early in pregnancy, fasting blood glucose levels drop, and this trend continues throughout the pregnancy. Insulin sensitivity decreases as pregnancy progresses, reaching pre-gravid levels about 34–36 weeks of pregnancy [40, 41]. Increases in hepatic glucose production during pregnancy show that the insulin action deficiency also affects the liver. Placental loss of anti-insulin hormones such as human placental lactogen, cortisol, oestrogen, and progesterone causes insulin resistance after mid-pregnancy. Low FBS, a low kidney glucose threshold, and enhanced insulin production are all effects of these hormones [42]. Maternal tissues become increasingly insulin resistant during pregnancy. This is thought to be produced in part by placental hormones and in part by unknown obesity and pregnancy-related variables. The main locations for glucose disposal throughout the body are skeletal muscle and adipose tissue. Insulin-mediated whole-body glucose elimination declines by 50% during pregnancy, and the woman must raise her insulin output by 200–250 percent to maintain a euglycemic condition [43]. Even while most women revert to a euglycaemic state immediately after delivery, women who have had GDM have a significantly higher chance of developing T2DM [44]. The biochemical relationship between GDM and T2DM is still unknown. Insulin resistance and/or aberrant insulin production define both illnesses [45]. Multiple potential protein indicators for later GDM have been discovered through proteomic screening in early pregnancy, including a cluster linked with insulin production, binding, resistance, and signalling [46]. An oral glucose tolerance test (OGTT) is usually used to identify GDM during 24–28 weeks of pregnancy. Because most of the physiologic insulin resistance of pregnancy will be firmly established, this timeframe has traditionally been favoured for routine GDM diagnosis. Another important difference in GDM testing techniques around

the world is the ongoing debate over whether testing should be universal (for all pregnant women) or targeted solely for women with risk factors linked to a higher probability of a positive result.

5.1 Management

Preventive interventions are needed to avoid the undesired consequences of obesity and hyperglycemia during pregnancy, given the global rise in obesity and the resulting increase in GDM [38, 47]. In roughly 70 to 85 percent of women with diagnosed GDM, lifestyle changes are enough to meet glycemic objectives [48]. Dietary counselling, in combination with physical activity and blood glucose self-monitoring, is the major intervention indicated for GDM [49]. A lower-carbohydrate diet with more animal protein and fat enhanced the risk of type 2 diabetes. As a result, it's possible that the diet that's best for treating GDM in women is not the best long-term diet [50]. Other nutritional treatments, such as probiotics and vitamin supplements, have gained popularity, but there is not enough data to suggest their widespread usage [51]. Insulin therapy is the preferred treatment because it does not cross the placenta and is thus deemed safe for the foetus. Metformin therapy was deemed safe and effective, and the women preferred it for insulin treatment [52]. Another study states that Metformin and sulfonylurea have been increasingly and safely used in the treatment of GDM [53]. In diabetic pregnant women with vitamin D deficiency/insufficiency, vitamin D administration can lower the chance of developing GDM and/or improve glycemic control [54]. Vitamin D regulates intracellular calcium to promote insulin production and attenuates insulin resistance by acting directly on pancreatic beta cells via the development of vitamin D receptors and the enzyme 25(OH)D-1- α -hydroxylase [55]. Furthermore, recent evidence from a large prospective trial suggests that increased physical activity may help reduce the risk of T2DM progression [56]. Exercise activities did not have a significant influence on the overall incidence of GDM in obese or overweight pregnant women, but when the effect measure was taken into account, the incidence of GDM was 24 percent lower in that group [57]. The following five components of guideline content were examined: GDM diagnosis, prenatal care, intrapartum care, neonatal care, and postpartum care. The majority of the suggestions in the guidelines were on prenatal care, particularly all types of therapy that could lower the risk of bad pregnancy outcomes due to uncontrolled blood sugar prior to conception [58]. The usage of information technology and digital platforms by diabetic pregnant women is fast rising around the world [59]. Telemedicine has been linked to high patient satisfaction since it allows for quick management of care across distances with fewer face-to-face physician appointments [60]. As a result, the use of e-platforms in the management of gestational diabetes shows encouraging results in terms of patient satisfaction and has no negative impact on pregnancy outcomes. Adequately powered RCTs are needed to assess whether such healthcare technologies are cost-effective or can help enhance care in urban or distant settings [61].

6. Gestational thrombocytopenia and anaemia

During pregnancy, several biological markers, particularly haematological, are physiologically altered. Biologists and doctors who are aware of these changes in the maternal body can screen for potential abnormalities. The haematological parameters must adjust in several ways, including providing vitamins and minerals for foetal haematopoiesis (iron, vitamin B12, folic acid), which can increase maternal anaemia, and preparing for birth bleeding, which is necessary to improve

homeostasis [62]. The total blood volume increases by roughly 1.5 litres during pregnancy, primarily to meet the demands of the new vascular bed and to compensate for blood loss that occurs during birth [63]. At 6–12 weeks of pregnancy, the plasma volume expands by 10–15 percent. When maternal erythropoietin production rises, RBC mass rises as well, albeit at a slower rate than plasma volume, resulting in a drop in haemoglobin concentration. Dilutional anaemia is the result [64]. Haemodilution also contributes to a decrease in the rate of haematocrit (HCT) and haemoglobin (HGB), resulting in a false anaemia. Such a change is natural for pregnant women and demonstrates the adoption of a different threshold for the definition of pregnancy anaemia. The WHO defines anaemia in pregnancy as having a total circulating HGB concentration of less than 11 g/dl or an HCT of less than 33% at any point during the pregnancy. During pregnancy, RBC indices do not vary much. However, in an iron-replete woman, there is a slight rise in mean corpuscular volume (MCV) of around 4 fl, which peaks around 30–35 weeks gestation and does not indicate a vitamin B12 or folate shortage. The increased MCV can be explained by increased RBC production to fulfil the demands of pregnancy [65]. The haemoglobin concentration does not change until the 16th week of pregnancy, after which it falls steadily to the second trimester due to the expansion of plasma volume [66]. Haemodilution, or an increase in plasma volume greater than an increase in red cell mass, is the underlying cause of anaemia during pregnancy. This condition is also known as ‘physiological anaemia of pregnancy’ [67]. Because length is more stable than weight, haemoglobin demonstrated a positive connection with infant length but not with weight [68]. Haemoglobin and haematocrit increased on the first day after birth, decreased on the third and fifth days, and then began to rise again by day 42, achieving normal haemoglobin in non-pregnant women [69]. Because of the greater metabolic oxygen requirement, erythropoietin levels are 50 percent greater, which explains the mild bone marrow erythroid hyperplasia and enhanced reticulocyte count. A combination of a lowered maternal RBCs oxygen affinity from an enhanced 2,3 Diphosphoglycerate and a low maternal pCO₂ results in enhanced oxygen transfer throughout the placenta [70].

Pregnancy causes an increase in white blood cell count, with the lowest limit of the reference range typically being 6,000/cumm. Leucocytosis occurs during pregnancy as a result of the physiologic stress that comes with being pregnant [71]. Throughout the first and second trimesters of pregnancy, lymphocyte count drops, then rises during the third trimester. Total leukocyte count levels rise significantly in the II and III trimesters, but there is no difference between pregnant and non-pregnant women in the I trimester. In the first trimester of pregnancy, non-anaemic women have a higher TLC count than anaemic women, but in the second and third trimesters, anaemic women have a higher TLC count than non-anaemic women [62]. During normal pregnancy, leukocytosis is caused by an enhanced inflammatory response, which can be caused by selective immunological tolerance, immunosuppression, and immunomodulation of the foetus [72]. During pregnancy, the ratio of monocytes to lymphocytes rises dramatically. During pregnancy, however, eosinophil and basophil numbers do not alter appreciably [73]. The neutrophil count starts to rise in the second month of pregnancy and reaches a plateau in the second or third trimester, when total white blood cell counts range from 9,000 to 15,000 cells per microliter.

In 7–8 percent of all pregnancies, gestational thrombocytopenia occurs. Due to rapid degradation, platelet counts are slightly lower which results in younger, bigger platelets present in pregnancy. The majority of thrombocytopenia in pregnancy is caused by increased blood loss [74]. Although the average platelet count falls monotonically during pregnancy, platelet aggregation increases, notably during the last 8 weeks of pregnancy [75]. The decline in the quantity of circulating platelets during pregnancy has been attributed to increased platelet consumption as well as a

shorter life span in the uteroplacental circulation [76]. As the pregnancy progresses, the platelet volume distribution width widens dramatically and continually. As a result, as pregnancy progresses, the mean platelet volume becomes an insensitive indicator of platelet size.

Primary immune thrombocytopenia (ITP) affects about 3% of women who are thrombocytopenic at delivery. It occurs in 1/1000–1/10 000 pregnancies [77]. Two-thirds of women with ITP have pre-existing disease, according to most studies, and one-third are diagnosed for the first time during pregnancy [78]. The pathophysiological mechanism of thrombotic thrombocytopenic purpura (TTP) is thrombotic microangiopathy. Microangiopathic hemolytic anaemia, thrombocytopenia, fever, neurological signs, and renal impairment are all symptoms of TTP. Pregnancy is thought to be the trigger event in between 5 to 25% of TTP cases [79]. TTP occurs in the second trimester of pregnancy and occasionally in the postpartum period, although it is uncommon in the first trimester [80]. If TTP appears during the first trimester, regular plasma exchange may be able to maintain pregnancy.

6.1 Management

Preventing anaemia in pregnancy requires effective communication about diet and nutrition to all pregnant women. Most experts recommend regular iron supplementation during pregnancy since the extra demand for iron is typically unmet by a typical diet. Although iron supplementation recommendations vary by location, the CDC recommends that all pregnant women begin a 30 mg/day iron supplementation [81]. The average iron density in a typical Indian diet is 8.5 mg/1000 Kcal, with 13.3 and 5.3 percent iron absorption in pregnancy from a rice-based and wheat-based Indian diet, respectively [82]. Women can use smartphone applications to learn about their daily iron needs, the iron content of various foods, and how to track their dietary iron intake. We support the creation and use of such applications. For improved absorption, all pregnant women should be told to take oral iron on an empty stomach or 1 hour after meals, preferably with a vitamin C-rich product like orange juice or guava. Supplement 2 outlines the oral iron treatments that can be used during pregnancy [82, 83].

The choice of therapy is based on the urgency of the platelet increase, the duration of the increase, and any potential side effects, and should be determined on an individual basis for each patient. Platelets should be available on standby if the mother's platelet count remains low (50–109/l) around the time of delivery, but they are likely to be destroyed rapidly after infusion if due to an immune reaction, so they should be given in well-established rather than early labour if there are increased bleeding complications [84]. Given that there is no evidence that Caesarean delivery is safer for the foetus with thrombocytopenia than a simple vaginal delivery, which is usually safer than caesarean for the mother, the mode of delivery should be decided on obstetric concerns. Treatment may be required just during the later part of the third trimester to boost the platelet level before epidural anaesthesia or C section if the individual is asymptomatic and the platelet count is more than $20 \times 10^9/L$ [85]. Depending on the platelet level and stability, general measures such as avoiding aspirin, nonsteroidal anti-inflammatory medications, and intramuscular injections might be explored. Because low-dose aspirin is now commonly administered in pregnancy for a variety of reasons, it should not be avoided unless the risk of bleeding is significant. Prednisone at a low dose or intravenous immunoglobulin, or both, are viable alternatives in these circumstances. In symptomatic pregnant ITP patients or if the platelet count is less than standard level, other therapeutic options are available. When combined with intravenous immunoglobulin, a large dose of steroids can be employed [86]. Corticosteroids and intravenous IVIG are the most common treatments for maternal ITP [87].

7. Conclusion

When it came to getting health information, pregnant women faced personal, societal, and structural challenges. As a result, legislators and health planners should remove barriers, promote self-care, and improve the quality of life for pregnant women, ultimately improving their health. Pregnancy issues such as gestational diabetes mellitus, hypertension, preeclampsia, caesarean birth, and post-partum weight retention are all more likely in overweight and obese women. More research into the link between nutritional advancements and the rising prevalence of GDM in the developing world is needed. Iron supplementation has been linked to glucose dysregulation and hypertension in mid-pregnancy; its effectiveness and potential risks should be carefully considered.

Acknowledgements

I would like to thank Saveetha Institute of Medical and Technical Sciences for giving me this opportunity to carry out the research work.

Funding Support

This study received no specific support from public, private, or non-profit funding bodies.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

Author details

Gayatri Devi Ramalingam^{1*}, Saravana Kumar Sampath² and Jothi Priya Amirtham¹

¹ Department of Physiology, Saveetha Dental College, SIMATS, Chennai, Tamil Nadu, India

² Department of Anatomy, SEGI University, Malaysia

*Address all correspondence to: gayatri.physio88@gmail.com; dr.sharan_anatomist@yahoo.com

IntechOpen

© 2021 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

References

- [1] Jung J, Horta H, Yonezawa A. Researching Higher Education in Asia: History, Development and Future. Springer; 2017. 366 p.
- [2] Pritchard JA. Changes in the Blood Volume During Pregnancy and Delivery [Internet]. Vol. 26, Anesthesiology. 1965. p. 393-9. Available from: <http://dx.doi.org/10.1097/00000542-196507000-00004>
- [3] Toglia MR, Weg JG. Venous Thromboembolism during Pregnancy [Internet]. Vol. 335, New England Journal of Medicine. 1996. p. 108-14. Available from: <http://dx.doi.org/10.1056/nejm199607113350207>
- [4] Website.
- [5] Website.
- [6] Catalano PM, Huston L, Amini SB, Kalhan SC. Longitudinal changes in glucose metabolism during pregnancy in obese women with normal glucose tolerance and gestational diabetes mellitus [Internet]. Vol. 180, American Journal of Obstetrics and Gynecology. 1999. p. 903-16. Available from: [http://dx.doi.org/10.1016/s0002-9378\(99\)70662-9](http://dx.doi.org/10.1016/s0002-9378(99)70662-9)
- [7] Catalano PM, Tyzbir ED, Wolfe RR, Calles J, Roman NM, Amini SB, et al. Carbohydrate metabolism during pregnancy in control subjects and women with gestational diabetes [Internet]. Vol. 264, American Journal of Physiology-Endocrinology and Metabolism. 1993. p. E60-7. Available from: <http://dx.doi.org/10.1152/ajpendo.1993.264.1.e60>
- [8] Lain KY, Catalano PM. Metabolic changes in pregnancy. Clin Obstet Gynecol. 2007 Dec;50(4):938-948.
- [9] Kitzmiller J, Jovanovic L, Brown F, Coustan D. Managing Preexisting Diabetes and Pregnancy: Technical Reviews and Consensus Recommendations for Care. American Diabetes Association; 2008. 852 p.
- [10] Das A, Sarkar M. Pregnancy-related health information-seeking behaviors among rural pregnant women in India: validating the Wilson model in the Indian context. Yale J Biol Med. 2014 Sep;87(3):251-262.
- [11] Dörtbudak O, Eberhardt R, Ulm M, Persson GR. Periodontitis, a marker of risk in pregnancy for preterm birth. J Clin Periodontol. 2005 Jan;32(1):45-52.
- [12] Offenbacher S. Maternal Periodontal Infections, Prematurity, and Growth Restriction [Internet]. Vol. 47, Clinical Obstetrics and Gynecology. 2004. p. 808-21. Available from: <http://dx.doi.org/10.1097/01.grf.0000141894.85221.f7>
- [13] Silk H, Douglass AB, Maier R, Clark M, Deutchman M, Douglass J, et al. Smiles for Life National Oral Health Curriculum: Module 5. Oral Health in Pregnancy [Internet]. Vol. 8, MedEdPORTAL. 2012. Available from: http://dx.doi.org/10.15766/mep_2374-8265.9259
- [14] Website [Internet]. [cited 2021 May 26]. Available from: <https://doi.org/10.4103/0976-9668.166124>
- [15] Sills ES, Zegarelli DJ, Hoschander MM, Strider WE. Clinical diagnosis and management of hormonally responsive oral pregnancy tumor (pyogenic granuloma). J Reprod Med. 1996 Jul;41(7):467-470.
- [16] Website [Internet]. [cited 2021 May 26]. Available from: American Dental Association Council on Access, Prevention and Interprofessional Relations. Women's oral health issues. American Dental Association, 2006. <http://www.ada.org/prof/resources/>

topics/healthcare_womens.pdf.
 Accessed August 1, 2007.

[17] Goepfert AR, Jeffcoat MK, Andrews WW, Faye-Petersen O, Cliver SP, Goldenberg RL, et al. Periodontal Disease and Upper Genital Tract Inflammation in Early Spontaneous Preterm Birth [Internet]. Vol. 104, Obstetrics & Gynecology. 2004. p. 777-83. Available from: <http://dx.doi.org/10.1097/01.aog.0000139836.47777.6d>

[18] Offenbacher S, Lieff S, Boggess KA, Murtha AP, Madianos PN, Champagne CME, et al. Maternal Periodontitis and Prematurity. Part I: Obstetric Outcome of Prematurity and Growth Restriction [Internet]. Vol. 6, Annals of Periodontology. 2001. p. 164-74. Available from: <http://dx.doi.org/10.1902/annals.2001.6.1.164>

[19] Lopez BC, Chaveli Lopez B, Perez MGS, Jimenez Soriano Y. Dental considerations in pregnancy and menopause [Internet]. Journal of Clinical and Experimental Dentistry. 2011. p. e135-44. Available from: <http://dx.doi.org/10.4317/jced.3.e135>

[20] Wright GZ, Kupietzky A. Behavior Management in Dentistry for Children. John Wiley & Sons; 2014. 264 p.

[21] Livingston HM, Mark Livingston H, Dellinger TM, Holder R. Considerations in the management of the pregnant patient [Internet]. Vol. 18, Special Care in Dentistry. 1998. p. 183-8. Available from: <http://dx.doi.org/10.1111/j.1754-4505.1998.tb01737.x>

[22] Zanata RL, Navarro MF de L, Pereira JC, Franco EB, Lauris JRP, Barbosa SH. Effect of caries preventive measures directed to expectant mothers on caries experience in their children. Braz Dent J. 2003 Oct 3;14(2):75-81.

[23] Sanghavi M, Rutherford JD. Cardiovascular Physiology of Pregnancy [Internet]. Vol. 130, Circulation. 2014.

p. 1003-8. Available from: <http://dx.doi.org/10.1161/circulationaha.114.009029>

[24] Kodogo V, Azibani F, Sliwa K. Role of pregnancy hormones and hormonal interaction on the maternal cardiovascular system: a literature review. Clin Res Cardiol. 2019 Aug;108(8):831-846.

[25] Ngene NC, Moodley J. Physiology of blood pressure relevant to managing hypertension in pregnancy. J Matern Fetal Neonatal Med. 2019 Apr;32(8):1368-1377.

[26] Simmons LA, Gillin AG, Jeremy RW. Structural and functional changes in left ventricle during normotensive and preeclamptic pregnancy [Internet]. Vol. 283, American Journal of Physiology-Heart and Circulatory Physiology. 2002. p. H1627-33. Available from: <http://dx.doi.org/10.1152/ajpheart.00966.2001>

[27] L. PK, Professor A, Permi HS, V. MS, Guruprasad Y, Professor A, et al. Study of Biochemical Parameters in Pregnancy Induced Hypertension (PIH) [Internet]. Vol. 5, Indian Journal of Pathology: Research and Practice. 2016. p. 191-4. Available from: <http://dx.doi.org/10.21088/ijprp.2278.148x.5216.21>

[28] Tessema GA, Tekeste A, Ayele TA. Preeclampsia and associated factors among pregnant women attending antenatal care in Dessie referral hospital, Northeast Ethiopia: a hospital-based study [Internet]. Vol. 15, BMC Pregnancy and Childbirth. 2015. Available from: <http://dx.doi.org/10.1186/s12884-015-0502-7>

[29] Luft FC, Eileen D. M. Gallery, Lindheimer MD. Normal and Abnormal Volume Homeostasis [Internet]. Chesley's Hypertensive Disorders in Pregnancy. 2009. p. 269-85. Available from: <http://dx.doi.org/10.1016/b978-0-12-374213-1.00015-x>

- [30] Agarwal I, Ananth Karumanchi S. Preeclampsia and the anti-angiogenic state [Internet]. Vol. 1, Pregnancy Hypertension: An International Journal of Women's Cardiovascular Health. 2011. p. 17-21. Available from: <http://dx.doi.org/10.1016/j.preghy.2010.10.007>
- [31] Tregear GW, Ivell R, Bathgate RA, Wade JD. Relaxin 2000. Springer Science & Business Media; 2001. 460 p.
- [32] Tranquilli AL, Dekker G, Magee L, Roberts J, Sibai BM, Steyn W, et al. The classification, diagnosis and management of the hypertensive disorders of pregnancy: A revised statement from the ISSHP [Internet]. Vol. 4, Pregnancy Hypertension: An International Journal of Women's Cardiovascular Health. 2014. p. 97-104. Available from: <http://dx.doi.org/10.1016/j.preghy.2014.02.001>
- [33] Redman CWG. Hypertension in pregnancy: the NICE guidelines [Internet]. Vol. 97, Heart. 2011. p. 1967-9. Available from: <http://dx.doi.org/10.1136/heartjnl-2011-300949>
- [34] Fidler J, Smith V, Fayers P, De Swiet M. Randomised controlled comparative study of methyldopa and oxprenolol in treatment of hypertension in pregnancy [Internet]. Vol. 286, BMJ. 1983. p. 1927-30. Available from: <http://dx.doi.org/10.1136/bmj.286.6382.1927>
- [35] Magee LA, Schick B, Donnerfeld AE, Sage SR, Conover B, Cook L, et al. The safety of calcium channel blockers in human pregnancy: a prospective, multicenter cohort study. *Am J Obstet Gynecol*. 1996 Mar;174(3):823-828.
- [36] Guideline for the Diagnosis and Management of Hypertension in Adults. 2016. 74 p.
- [37] Catalano PM, Ehrenberg HM. Review article: The short- and long-term implications of maternal obesity on the mother and her offspring [Internet]. Vol. 113, BJOG: An International Journal of Obstetrics & Gynaecology. 2006. p. 1126-33. Available from: <http://dx.doi.org/10.1111/j.1471-0528.2006.00989.x>
- [38] Mottola MF, Artal R. Role of Exercise in Reducing Gestational Diabetes Mellitus [Internet]. Vol. 59, Clinical Obstetrics & Gynecology. 2016. p. 620-8. Available from: <http://dx.doi.org/10.1097/grf.0000000000000211>
- [39] The Pregnant Diabetic and Her Newborn. Problems and Management [Internet]. Vol. 43, Archives of Disease in Childhood. 1968. p. 391-391. Available from: <http://dx.doi.org/10.1136/adc.43.229.391-a>
- [40] Kim C, Ferrara A. Gestational Diabetes During and After Pregnancy. Springer Science & Business Media; 2010. 394 p.
- [41] Catalano PM, Tyzbir ED, Roman NM, Amini SB, Sims EAH. Longitudinal changes in insulin release and insulin resistance in nonobese pregnant women [Internet]. Vol. 165, American Journal of Obstetrics and Gynecology. 1991. p. 1667-72. Available from: [http://dx.doi.org/10.1016/0002-9378\(91\)90012-g](http://dx.doi.org/10.1016/0002-9378(91)90012-g)
- [42] A review article- gestational diabetes mellitus. *Int J Endocrinol Metab*. 2019 Feb 7;7(1):62-5.
- [43] Barbour LA, McCurdy CE, Hernandez TL, Kirwan JP, Catalano PM, Friedman JE. Cellular Mechanisms for Insulin Resistance in Normal Pregnancy and Gestational Diabetes [Internet]. Vol. 30, Diabetes Care. 2007. p. S112-9. Available from: <http://dx.doi.org/10.2337/dc07-s202>
- [44] Bellamy L, Casas J-P, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis [Internet].

Vol. 373, The Lancet. 2009. p. 1773-9.
 Available from: [http://dx.doi.org/10.1016/s0140-6736\(09\)60731-5](http://dx.doi.org/10.1016/s0140-6736(09)60731-5)

[45] Lauenborg J, Grarup N, Damm P, Borch-Johnsen K, Jørgensen T, Pedersen O, et al. Common type 2 diabetes risk gene variants associate with gestational diabetes. *J Clin Endocrinol Metab.* 2009 Jan;94(1):145-150.

[46] Zhou T, Huang L, Wang M, Chen D, Chen Z, Jiang S-W. A Critical Review of Proteomic Studies in Gestational Diabetes Mellitus. *J Diabetes Res.* 2020 Jul 14;2020:6450352.

[47] Bain E, Crane M, Tieu J, Han S, Crowther CA, Middleton P. Diet and exercise interventions for preventing gestational diabetes mellitus [Internet]. *Cochrane Database of Systematic Reviews.* 2015. Available from: <http://dx.doi.org/10.1002/14651858.cd010443.pub2>

[48] Association AD, American Diabetes Association. 13. Management of Diabetes in Pregnancy: Standards of Medical Care in Diabetes—2018 [Internet]. Vol. 41, *Diabetes Care.* 2018. p. S137-43. Available from: <http://dx.doi.org/10.2337/dc18-s013>

[49] Lapolla A, Fedele D, Dalfra. Management of gestational diabetes mellitus [Internet]. Vol. 2, *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy.* 2009. p. 73-82. Available from: <http://dx.doi.org/10.2147/dmso.s3407>

[50] Bao W, Li S, Chavarro JE, Tobias DK, Zhu Y, Hu FB, et al. Low Carbohydrate–Diet Scores and Long-term Risk of Type 2 Diabetes Among Women With a History of Gestational Diabetes Mellitus: A Prospective Cohort Study [Internet]. Vol. 39, *Diabetes Care.* 2016. p. 43-9. Available from: <http://dx.doi.org/10.2337/dc15-1642>

[51] Dolatkhah N, Hajifaraji M, Abbasalizadeh F, Aghamohammadzadeh N, Mehrabi Y, Abbasi MM. Is there a value for probiotic supplements in gestational diabetes mellitus? A randomized clinical trial [Internet]. Vol. 33, *Journal of Health, Population and Nutrition.* 2015. Available from: <http://dx.doi.org/10.1186/s41043-015-0034-9>

[52] Rowan JA, Hague WM, Gao W, Battin MR, Peter Moore M. Metformin versus Insulin for the Treatment of Gestational Diabetes [Internet]. Vol. 358, *New England Journal of Medicine.* 2008. p. 2003-15. Available from: <http://dx.doi.org/10.1056/nejmoa0707193>

[53] Dhulkotia JS, Ola B, Fraser R, Farrell T. Oral hypoglycemic agents vs insulin in management of gestational diabetes: a systematic review and metaanalysis. *Am J Obstet Gynecol.* 2010 Nov;203(5):457.e1-9.

[54] Poel YHM, Hummel P, Lips P, Stam F, van der Ploeg T, Simsek S. Vitamin D and gestational diabetes: a systematic review and meta-analysis. *Eur J Intern Med.* 2012 Jul;23(5):465-469.

[55] Alvarez JA, Ashraf A. Role of vitamin d in insulin secretion and insulin sensitivity for glucose homeostasis. *Int J Endocrinol.* 2010;2010:351385.

[56] Bao W, Tobias DK, Bowers K, Chavarro J, Vaag A, Grunnet LG, et al. Physical activity and sedentary behaviors associated with risk of progression from gestational diabetes mellitus to type 2 diabetes mellitus: a prospective cohort study. *JAMA Intern Med.* 2014 Jul;174(7):1047-1055.

[57] Nasiri-Amiri F, Sepidarkish M, Shirvani MA, Habibipour P, Tabari NSM. The effect of exercise on the prevention of gestational diabetes in obese and overweight pregnant women:

a systematic review and meta-analysis. *Diabetol Metab Syndr*. 2019 Aug 27;11:72.

[58] Mahmud M, Mazza D. Preconception care of women with diabetes: a review of current guideline recommendations. *BMC Womens Health*. 2010 Jan 31;10:5.

[59] Diabetes Canada Clinical Practice Guidelines Expert Committee, Feig DS, Berger H, Donovan L, Godbout A, Kader T, et al. Diabetes and Pregnancy. *Can J Diabetes*. 2018 Apr;42 Suppl 1:S255-82.

[60] Ivey TL, Hughes D, Dajani NK, Magann EF. Antenatal management of at-risk pregnancies from a distance. *Aust N Z J Obstet Gynaecol*. 2015 Feb;55(1):87-89.

[61] Mitric C, Desilets J, Brown RN. Recent advances in the antepartum management of diabetes. *F1000Res*. 2019 May 8;8(622):622.

[62] Mohamed A, Hamza K, Babker A. Physiological changes in some hematological and coagulation profile among Sudanese healthy pregnant women [Internet]. Vol. 5, *International Journal of Medical Science and Public Health*. 2016. p. 525. Available from: <http://dx.doi.org/10.5455/ijmsph.2016.30092015149>

[63] Ramsay M. Normal hematological changes during pregnancy and the puerperium [Internet]. *The Obstetric Hematology Manual*. p. 3-12. Available from: <http://dx.doi.org/10.1017/cbo9780511676451.002>

[64] Bernstein IM, Ziegler W, Badger GJ. Plasma Volume Expansion in Early Pregnancy [Internet]. Vol. 97, *Obstetrics & Gynecology*. 2001. p. 669-72. Available from: <http://dx.doi.org/10.1097/00006250-200105000-00005>

[65] Taylor DJ, Lind T. Red cell mass during and after normal pregnancy. *Br J Obstet Gynaecol*. 1979 May;86(5):364-370.

[66] Micronutrient Initiative, Gallego EB, International Development Research Centre (Canada). Severe Anemia in Pregnancy: Report of a Workshop Held at the Institute of Child and Mother Health in Dhaka, Bangladesh. L'Initiative micronutriments = Micronutrient Initiative; 2000. 31 p.

[67] P S, Sharma P, Research Scholar, Department of Home Science, University of Rajasthan, Jaipur. Hematological profile of anemic pregnant women attending antenatal hospital [Internet]. Vol. 1, *IOSR Journal of Nursing and Health Science*. 2013. p. 11-5. Available from: <http://dx.doi.org/10.9790/1959-0141115>

[68] Weerd S de, de Weerd S, Steegers-Theunissen RPM, de Boo TM, Thomas CMG, Steegers EAP. Maternal periconceptional biochemical and hematological parameters, vitamin profiles and pregnancy outcome [Internet]. Vol. 57, *European Journal of Clinical Nutrition*. 2003. p. 1128-34. Available from: <http://dx.doi.org/10.1038/sj.ejcn.1601654>

[69] Ramakers C, Van Der WOUDE DAA, Verzijl JM, Pijnenborg JMA, Van WIJK EM. An added value for the hemoglobin content in reticulocytes (CHr) and the mean corpuscular volume (MCV) in the diagnosis of iron deficiency in postpartum anemic women [Internet]. Vol. 34, *International Journal of Laboratory Hematology*. 2012. p. 510-6. Available from: <http://dx.doi.org/10.1111/j.1751-553x.2012.01423.x>

[70] Madsen H, Ditzel J. Red cell 2,3-diphosphoglycerate and hemoglobin--oxygen affinity during

normal pregnancy. *Acta Obstet Gynecol Scand.* 1984;63(5):399-402.

[71] Akinlaja O. Hematological Changes in Pregnancy - The Preparation for Intrapartum Blood Loss [Internet]. Vol. 4, *Obstetrics & Gynecology International Journal*. 2016. Available from: <http://dx.doi.org/10.15406/ogij.2016.04.00109>

[72] Osonuga IO, Osonuga OA, Onadeko AA, Osonuga A, Osonuga AA. Hematological profile of pregnant women in southwest of Nigeria [Internet]. Vol. 1, *Asian Pacific Journal of Tropical Disease*. 2011. p. 232-4. Available from: [http://dx.doi.org/10.1016/s2222-1808\(11\)60036-4](http://dx.doi.org/10.1016/s2222-1808(11)60036-4)

[73] Edelstam G, Löwbeer C, Kral G, Gustafsson SA, Venge P. New reference values for routine blood samples and human neutrophilic lipocalin during third-trimester pregnancy. *Scand J Clin Lab Invest.* 2001;61(8):583-592.

[74] Eledo BO. Evaluation of Some Haematological Parameters Among Post-menopausal Women in Bayelsa State, Nigeria: A Case Study of Patients Attending Federal Medical Centre, Yenagoa [Internet]. Vol. 2, *American Journal of Laboratory Medicine*. 2017. p. 132. Available from: <http://dx.doi.org/10.11648/j.ajlm.20170206.14>

[75] Fay RA, Hughes AO, Farron NT. Platelets in pregnancy: hyperdestruction in pregnancy. *Obstet Gynecol.* 1983 Feb;61(2):238-240.

[76] Ahmed Y, Van Iddekinge B, Paul C, Sullivan MHF, Elder MG. Retrospective analysis of platelet numbers and volumes in normal pregnancy and in pre-eclampsia [Internet]. Vol. 43, *International Journal of Gynecology & Obstetrics*. 1993. p. 230-230. Available from: [http://dx.doi.org/10.1016/0020-7292\(93\)90342-t](http://dx.doi.org/10.1016/0020-7292(93)90342-t)

[77] Gill KK, Kelton JG. Management of idiopathic thrombocytopenic purpura in pregnancy [Internet]. Vol. 37, *Seminars in Hematology*. 2000. p. 275-89. Available from: <http://dx.doi.org/10.1053/shem.2000.8960>

[78] Webert KE, Mittal R, Sigouin C, Heddle NM, Kelton JG. A retrospective 11-year analysis of obstetric patients with idiopathic thrombocytopenic purpura. *Blood.* 2003 Dec 15;102(13):4306-4311.

[79] Vesely SK, Li X, McMinn JR, Terrell DR, George JN. Pregnancy outcomes after recovery from thrombotic thrombocytopenic purpura-hemolytic uremic syndrome [Internet]. Vol. 44, *Transfusion*. 2004. p. 1149-58. Available from: <http://dx.doi.org/10.1111/j.1537-2995.2004.03422.x>

[80] Scully M, Hunt BJ, Benjamin S, Liesner R, Rose P, Peyvandi F, et al. Guidelines on the diagnosis and management of thrombotic thrombocytopenic purpura and other thrombotic microangiopathies [Internet]. Vol. 158, *British Journal of Haematology*. 2012. p. 323-35. Available from: <http://dx.doi.org/10.1111/j.1365-2141.2012.09167.x>

[81] Pavord S, Myers B, Robinson S, Allard S, Strong J, Oppenheimer C, et al. UK guidelines on the management of iron deficiency in pregnancy. *Br J Haematol.* 2012 Mar;156(5):588-600.

[82] Bothwell TH. Iron requirements in pregnancy and strategies to meet them [Internet]. Vol. 72, *The American Journal of Clinical Nutrition*. 2000. p. 257S – 264S. Available from: <http://dx.doi.org/10.1093/ajcn/72.1.257s>

[83] Jacob A. Medical Disorders Associated with Pregnancy [Internet]. *A Comprehensive Textbook of Midwifery*. 2008. p. 335-335. Available from: http://dx.doi.org/10.5005/jp/books/10008_29

[84] Myers B. Diagnosis and management of maternal thrombocytopenia in pregnancy [Internet]. Vol. 158, British Journal of Haematology. 2012. p. 3-15. Available from: <http://dx.doi.org/10.1111/j.1365-2141.2012.09135.x>

[85] Provan D, Stasi R, Newland AC, Blanchette VS, Bolton-Maggs P, Bussel JB, et al. International consensus report on the investigation and management of primary immune thrombocytopenia [Internet]. Vol. 115, Blood. 2010. p. 168-86. Available from: <http://dx.doi.org/10.1182/blood-2009-06-225565>

[86] Izak M, Bussel JB. Management of thrombocytopenia. F1000Prime Rep [Internet]. 2014 Jun 2 [cited 2021 May 28];6(45). Available from: <https://facultyopinions.com/prime/reports/m/6/45/pdf>

[87] Greaves M, Letsky EA. Guidelines on the investigation and management of thrombocytopenia in pregnancy and neonatal alloimmune thrombocytopenia [Internet]. Vol. 104, BJOG: An International Journal of Obstetrics and Gynaecology. 1997. p. 1108-1108. Available from: <http://dx.doi.org/10.1111/j.1471-0528.1997.tb10931.x>