

Hemodynamic Changes in the Uterus and its Blood Vessels in Pregnancy*†

F. Burbank

INTRODUCTION

Postpartum hemorrhage (PPH) most commonly originates from disrupted blood vessels of the uterus, a unique circulation supplied by two arterial systems and drained by two venous plexes. At term, the uterus receives one-tenth of the output of the heart.

During pregnancy fetal tissue invades the uterus and transforms a few hundred tiny arterioles into large, trumpet-shaped arteries that supply the placenta. At delivery, these huge arteries are torn apart, spilling blood into the uterine cavity.

All women do not die from hemorrhage during delivery because the potential for blood clotting and the accumulation of fibrinolytic substances build up in the mother's circulation during pregnancy. At the onset of true labor, clotting starts in the uterine circulation to prevent blood loss. A few hours following delivery, fibrinolysis ensues to ensure that blood flow resumes to the uterus.

It is the balance between blood clotting and fibrinolysis that determines the outcome of PPH.

Vascular imaging studies are performed using film-based or digital angiography, ultrasound with or without Doppler encoding, contrast medium-enhanced computed tomography (CT), and unenhanced and contrast medium-enhanced magnetic resonance imaging (MRI). At one extreme, an ultrasound examination could be restricted to visualization of the right uterine artery, in cross-section, at the level of uterine isthmus. At the other extreme, a dynamic arteriogram recorded on multiple 14 × 14 inch cut-films or on a

14 inch image intensifier, could display all the blood vessels in the pelvis on a sequence of images beginning with arterial filling and ending with venous drainage. A contrast medium-enhanced, spiral CT can study all major arteries in the chest, abdomen and pelvis during a single injection of contrast medium within one breath-hold! Finally, imaging studies can examine blood flow during the whole cardiac cycle, providing information from systole through diastole, and they can characterize the entire vascular tree, from arteries to capillaries and veins. Vascular imaging is the foundation of modern vascular surgery, cardiac surgery, interventional cardiology and interventional radiology. Anatomic, surgical and imaging studies each have a place in developing a coherent understanding of the vasculature of the uterus.

UTERINE BLOOD VESSELS BEFORE PREGNANCY – ARTERIES OF THE UTERUS

Arteries that touch or are within the uterus – extrinsic arteries of the uterus

Uterine arteries

Blood reaches the uterus primarily from the right and left uterine arteries, secondarily from small right and left communicating arteries that connect ipsilateral ovarian and ascending uterine arteries, and to a minor extent from tiny, unnamed, randomly distributed arteries that reach the uterus through the broad ligament (Figure 1)^{1–14}. Uterine arteries are of medium

*Excerpted from selected chapters in: Burbank F. *Fibroids, Menstruation, Childbirth, and Evolution: The Fascinating Story of Uterine Blood Vessels*. Tucson, AZ: Wheatmark, 2009; ISBN: 978–1–60494–170–8.

†Editor's comments: The first edition of this Textbook was directed towards treatment modalities. In this second edition, the editors have made great effort to expand the thrust beyond traditional and non-traditional therapeutic measures. This chapter is an effort to make the complexity of the vascular supply of the uterus more easily understandable. It supplements much of the material presented in the chapters by Professor Palacios-Jaraquemada and his colleagues which clearly points out the differences between the therapeutic measures required when PPH is from the upper uterine segment (S1) as opposed to the lower uterine segment (S2). Moreover, the language selected by Dr Burbank is simple and straightforward. In other words, this chapter deals with anatomy but it is not written in anatomical jargon.

This chapter is a synopsis of material presented in the full chapter available at www.glowm.com in *The Global Library of Women's Medicine* by Dr Fred Burbank, a radiologist residing in California USA. The editors are grateful to Dr Burbank for presenting readers with a easily understandable impression of what every obstetrician/gynecologist deals with everyday but certainly cannot always be expected to have at his/her fingertips when PPH arises. L.G.K.

size. As a point of reference, the common iliac artery – which is a large artery – is approximately 13 mm in diameter¹⁵.

Ultrasound measurements of uterine artery diameters have been published and, in general, these diameters are smaller than angiographically measured diameters. Because angiographic measurements of arterial sizes have to be corrected for magnification, the process is imprecise, and ultrasound size measurements are probably more accurate. Each method visualizes only the internal lumen of an artery or vein. Average diameter of the left uterine artery in one series which examined 27 non-pregnant women was 1.6 mm¹⁶. Palmer *et al.* reported the average diameter of the right and left uterine arteries to be 1.4 mm in 12 non-pregnant women¹⁷. Taken together, these ultrasound and angiographic studies place the range of normal, non-pregnant uterine artery diameters somewhere between 1.5 and 5 mm, considerably smaller than the internal iliac artery, approximately the size range of the coronary arteries. As is shown later, the uterine arteries increase in diameter during pregnancy.

Each uterine artery arises from the ipsilateral internal iliac artery or from one of its major branches or divisions (Figure 1)^{7,18}. Variation exists in the division of the internal iliac artery into branches¹⁹.

During angiography, the uterine arteries are definitively identified by their unique shape and by their insertion into the uterus, not by their origin from the internal iliac artery origin (Figure 1). The proximal third of each uterine artery descends inferiorly, in a relatively straight line. In their mid and distal thirds, however, the uterine arteries undulate in a tortuous pattern that angiographically looks like loops. They are not loops; rather they are undulations.

The undulations in the uterine arteries are not acquired features. They are redundant arterial length which is present in both fetuses and nulliparous women²⁰. As is shown later, the uterine arteries grow rapidly in diameter in response to increasing blood flow during pregnancy, but do not appear to have the capacity to grow rapidly in length. The undulations seem to be reserve arterial length that is used when the uterus expands into the abdominal cavity during pregnancy.

A triangular space described by Beliaeva as the ‘cavity of the broad ligament’ is present at the base of the broad ligament (Figure 2)¹⁴. Within this cavity the uterine arteries join the lateral borders of the uterus at the level of the isthmus. The junction of the uterine arteries with the isthmus is almost always within 15 mm of the lateral vaginal fornices²¹. When the cervix is viewed as a clock face from the perspective of a vaginal examination, the right uterine artery joins the uterus at approximately 9:00 o’clock; the left, at approximately 3:00 o’clock. Very little variation in this clock pattern exists²¹.

Ovarian arteries

The ovarian arteries originate in the abdomen where the ovaries and their arterial supply form during the

embryologic period (see Figure 1). During intra-uterine development the ovaries migrate from the abdomen to the pelvis and drag their blood supply along with them. The ovarian arteries most commonly arise directly from the abdominal aorta but can originate as branches of the right or left renal arteries, from lumbar, adrenal, or iliac arteries, and can be duplicated^{7,19,22–24}. In the lower abdomen and pelvis the ovarian arteries undulate in a pattern similar to the tortuosity seen in the uterine arteries^{8,25}. Like the uterine arteries, tortuosity in the ovarian arteries appears to provide redundant arterial length that is

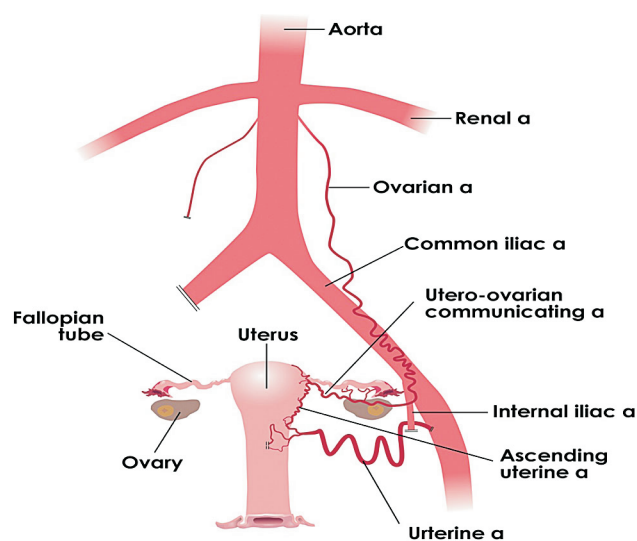


Figure 1 Diagram showing the extrinsic arteries of the left side of the uterus, including the aorta and renal and ovarian arteries arising from the abdominal aorta, the uterine artery arising from the internal iliac artery, and the utero-ovarian communicating artery. Symmetrical arteries are present on the right but are not shown

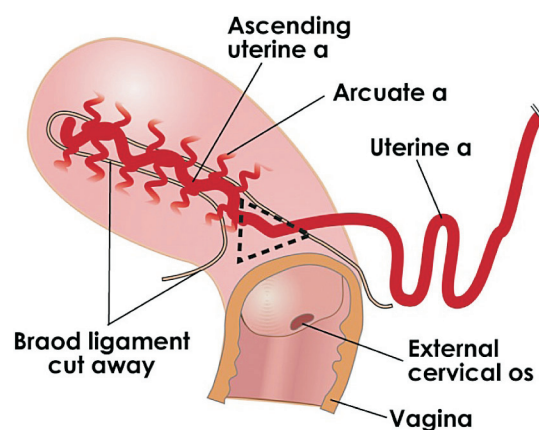


Figure 2 Drawing of the uterus as seen from the left lateral perspective with the broad ligament and vagina partially cut away to show the cavity of the broad ligament which is shown as a dashed line triangle. The left uterine artery reaches the uterus within this triangle at the 3:00 o’clock position of the cervix, approximately at the level of the internal cervical os. Note: Like coronary arteries, the ascending uterine arteries are surface vessels. The arcuate arteries are not; they cannot be seen from outside of the uterus. They traverse the uterus within the vascular layer of the myometrium

called upon during rapid enlargement of the uterus in pregnancy.

Utero-ovarian communicating arteries

Blood flow within the communicating arteries is tidal. Flow can pass from the uterine circulation to the ovarian circulation or from the ovarian circulation to the uterine depending on resistance differences between the two systems. In most women, the utero-ovarian communicating arteries are smaller in diameter than the uterine arteries or the ovarian arteries^{26,27}.

The communicating arteries are small enough to be difficult to visualize on routine angiography.

In general, each communicating artery is much smaller than its corresponding uterine artery. Consequently, each ovarian artery can only *potentially* supply the full blood flow needs of the uterus. To fully supply the uterus, the communicating arteries and their ipsilateral parent ovarian arteries must first experience increased blood flow. Once increased blood flow occurs, the ovarian and communicating arteries grow in diameter. When their diameters are equal to the diameter of the uterine arteries, they can supply the full metabolic needs of the uterus.

Broad ligament arteries

Broad ligament arteries are tiny vessels that arise from the main uterine arteries along their paths within the broad ligament¹⁴. They connect the main uterine arteries with the ascending uterine artery, and other branches, at random locations.

Arteries that touch or are within the uterus – intrinsic arteries of the uterus

Anatomy

Just prior to contact with the uterus, the uterine arteries give rise to branches that run along the right and left lateral borders of the cervix and vaginal dome. These arteries are referred to as either the ‘descending’ uterine arteries or ‘vaginal’ branches of the uterine arteries. The descending uterine arteries supply the isthmus, cervix and upper vagina^{28,29}. After joining the uterus, the uterine arteries ascend along the right and left lateral margins of the body of the uterus and are referred to as the right and left ‘ascending’ uterine arteries (Figures 1 and 2). As they ascend, the uterine arteries undulate and give rise to a dozen or more arteries that course between the outer and middle thirds of the myometrium³⁰. This zone is referred to as the ‘vascular zone’ or in older literature as the ‘stratum vasculare’. Because of their semicircular course, these arteries are referred to as ‘arcuate’ arteries (Figure 3)³¹. Arcuate arteries arise from the ascending uterine arteries in a haphazard manner with thicker branches compensating for thinner ones. Direct and continuous anastomotic connections are present between right and left arcuate arteries which connect anteriorly and posteriorly near the uterine sagittal midline forming

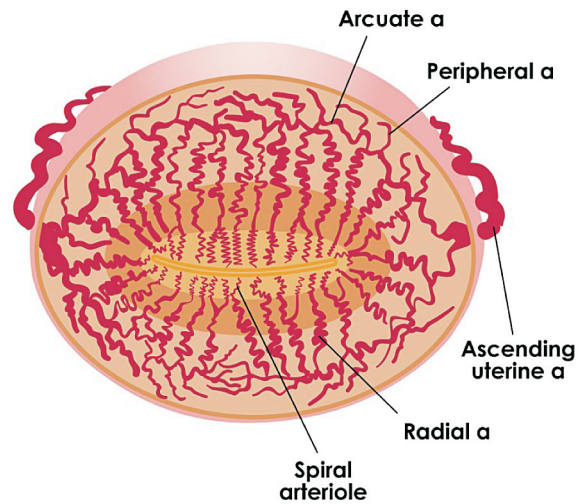


Figure 3 Drawing of transverse section through the body of the uterus showing the intrinsic arteries of the uterus: the ascending uterine, arcuate, peripheral and radial arteries and the spiral arterioles. Ascending uterine arteries are shown in perspective. This arterial pattern is repeated throughout the body of the uterus. Mucus on the surface of the endometrium is depicted as the innermost zone; the endometrium next; the junctional zone next; the myometrium next; and finally the serosa. Adapted from Sampson³¹

an arterial grid throughout the uterus³². The arcuate arteries give rise to peripheral arteries that course towards the serosal surface of the uterus and radial arteries that course toward the endometrial cavity.

Like the ovarian, uterine and ascending uterine arteries, the radial arteries undulate along their path. Again, the undulation is most likely present to provide reserve arterial length during the rapid volume growth of the uterus during pregnancy. They terminate at the endometrial–junctional zone border by giving off multiple branches that enter and supply the endometrium. Longer, tortuous branches are described as ‘spiral arterioles’. They are termed ‘spiral’ for their corkscrew or spiral appearance and are classified as arterioles since most cannot be seen by the naked eye. Shorter, straight arterioles also arise from the terminal radial arteries. These supply the basal layer of the endometrium, that portion that does not slough during menstruation. Compared with the density of arteries in the myometrium, endometrial arterioles are sparse³⁰.

Vascular embryology

All of the intrinsic arteries of the uterus are formed prior to birth.

Arteries that do not touch the uterus – collateral uterine arterial pathways

Although the uterine arteries originate from the internal iliac artery, the proximal occlusion of an internal iliac artery does not stop blood flow in the ipsilateral uterine arteries to the uterus. A network of collateral arteries supplies blood to the uterine arteries when

the internal iliac arteries are occluded. Collateral flow reaches the uterus from multiple branches of the aorta (inferior mesenteric artery, lumbar and vertebral arteries, and middle sacral), from multiple branches of the external iliac artery (deep iliac circumflex and inferior epigastric artery) and from femoral artery branches (medial femoral circumflex and lateral femoral circumflex)^{5,33–35}. When bilateral internal iliac artery occlusion was performed *proximal* to the posterior division of the internal iliac artery, reversed collateral flow from the iliolumbar and lateral sacral arteries filled the anterior divisions of the internal iliac arteries and re-established antegrade blood flow in each uterine artery. When bilateral internal iliac occlusion was performed *distal* to the posterior division, reverse flow in the middle hemorrhoidal artery reconstituted antegrade flow in each uterine artery. Under these two conditions, antegrade flow in each uterine artery persisted, but flow was not normal. Pulse pressure was dampened, resembling pressure variations in a venous system instead of an arterial system. Bilateral occlusion of the internal iliac arteries changes the character of perfusion to the uterus; it does not stop antegrade perfusion of the uterus through the uterine arteries. Following bilateral iliac artery occlusion during cesarean delivery, Chitrit *et al.* noted no change in Doppler flow velocity waveforms in the uterine arteries³⁶.

If the right and left uterine arteries are occluded, the uterus does not die. This is a unique organ response. For example, if the right and left coronary or renal arteries were occluded, the heart or kidneys, respectively, would die. The uterus does not die because the ovarian and broad ligament arteries can supply sufficient blood to the uterus to keep it alive while they increase in diameter and eventually provide the full needs of the uterus. If an ascending uterine artery were occluded, blood flow from the contralateral ascending uterine artery could supply the uterus³⁷. If the right anterior arcuate arteries were occluded, the left anterior arcuate arteries could supply the right-sided territory, and so on. As a result of these redundant extrinsic and intrinsic uterine arterial connections, the vasculature of the uterus functions like a big-city electric power grid. Short of hysterectomy, long-term power outage in the uterus is nearly impossible.

UTERINE BLOOD VESSELS DURING PREGNANCY – EMBRYO AND FETUS NOURISHMENT BY PLACENTA

The mature placenta

The discoid, hemochorial placenta is an organ in which maternal blood comes into direct contact with fetal trophoblast cells that cover placental villi. Maternal and fetal circulatory systems come into very, very close contact, separated only by a lining of trophoblasts. However, they are separate, and the separation of the maternal and fetal circulation has been known since 1786³⁸. The maternal side of the mature placental circulation is shown in Figure 4. Blood is delivered to the intravillous space by uteroplacental

arteries which spray oxygenated blood over fetal villi. Blood returns to the maternal circulation from the placenta by way of uteroplacental veins.

The mature placenta – growth and ‘migration’ later in pregnancy

Until the end of the fourth month of pregnancy, the normal placenta grows in both thickness and circumference. After this period there is no appreciable increase in placental thickness but the placenta continues to grow circumferentially until near the end of pregnancy.

The absolute position of the placenta on the surface of the uterus is fixed at the time of implantation. Over time, the placenta grows centrifugally. At the same time as the placenta grows in mass, the uterus expands in volume due to fetal and amniotic fluid growth and by myometrial growth. The growth is *pari passu* with that portion of the wall of the uterus to which the placenta is attached (Figure 5)³⁹. A placenta cannot pick up and move like a crab or a spider. It is firmly connected to the underlying myometrium and has large arterial and venous connections that cannot move. However, differential growth of the placenta and the uterine cavity does occur over time resulting in an apparent shift or ‘migration’ of placental location. Most commonly, the apparent shift is away from the internal cervical os^{40–42}.

At 18 weeks, 45.1% of placentas were posterior and 42.1% anterior. By 34 weeks, slight variations in placental location were seen with more apparent ‘migration’ for placentas attached to the posterior wall than the anterior. All of the posterior low lying placentas and all but 3.4% of the anterior low lying placentas ‘migrated’ away from the cervical os.

When the placenta does remain within or very near to the internal cervical os, placenta previa is present.

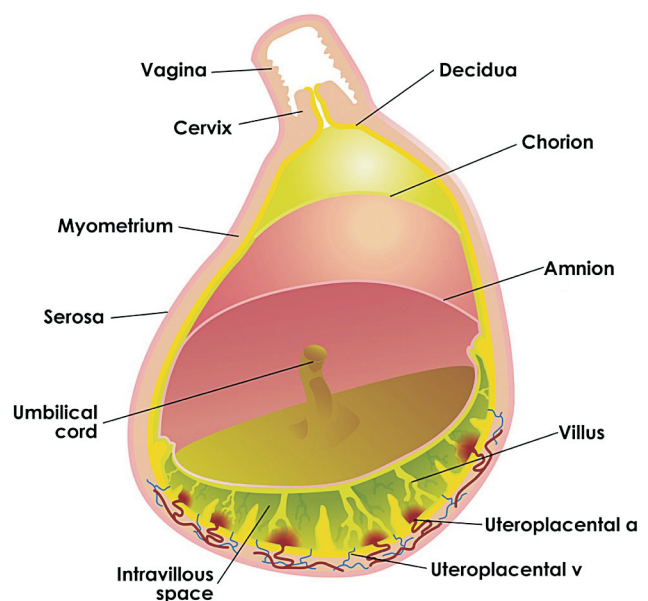


Figure 4 Illustration of the maternal circulation to and from a mature placenta. Fetal circulation is not shown

Placenta previa is variously described as ‘total’ if the internal os is covered by the placenta, ‘partial’ if the os is only partially covered, and ‘marginal’ if the edge of the placenta is at the edge of the os. Because the cervix dilates during labor, bleeding from separation of the placenta from the uterine wall can occur. The risk of placenta previa increases with maternal age and parity⁴⁴. Preterm detachment of the normally implanted placenta, commonly termed ‘abruptio placenta’, is attributed variously to abnormal myometrial arteries at the placental base, abnormal uterine contractions, or is considered idiopathic^{45–49}.

The mature placenta – term placental size in relation to uterine surface area

At term the average placenta is 185 mm in diameter, 23 mm in thickness, 497 mm³ in volume and weighs 508 g³⁹. The term placenta is in contact with approximately 20% of the surface area of the uterus (Figure 6). Average placental base surface area is 252 cm² which corresponds to a diameter of 18 cm^{39,50–53}. This area is referred to as the ‘placental footprint’ which is the area where hemostasis must occur if the mother is to survive placental separation.

UTERINE BLOOD VESSELS DURING PREGNANCY – HEMODYNAMIC CHANGES DURING PREGNANCY

Parallel blood flow circuits in the uterus

Of all the thousands of spiral arterioles in the uterus, however, only 200 spiral arterioles are transformed into uteroplacental arteries. To feed decidua and myometrium throughout the uterus, spiral and straight arterioles remain arterioles, with their high vascular resistance. As a result, two classes of arteries exist in the uterus. A small number of spiral arterioles and radial arteries are transformed into uteroplacental arteries, and a much larger number of arterioles are not transformed.

Placental and non-placental circulations are parallel sub-systems within the uterus. In parallel blood flow, some blood flow is distributed to one limb of the sub-systems and some to the other. Blood does not flow first through one sub-system and then another as in the serial sub-system.

Uterine artery resistance drops dramatically between weeks 8 and 16, decreasing little thereafter. Over an entire pregnancy, uterine artery resistance in a pregnant woman drops to approximately half the level of that in a non-pregnant woman by 24 weeks.

Uterine artery diameter increases during pregnancy

As vascular resistance in the arterioles of the uterus drops, blood flow increases, and the diameter of the uterine arteries increases during pregnancy. Average uterine artery diameter increases from 3 mm at the beginning of pregnancy to 7 mm at term.

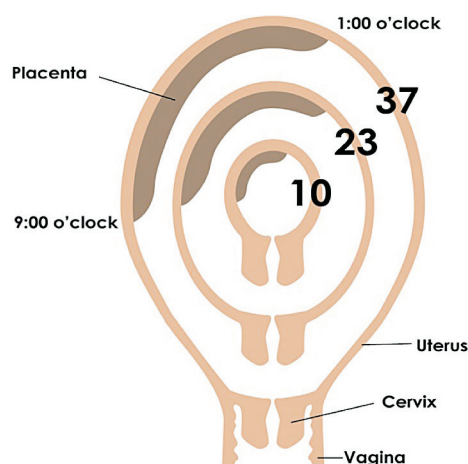


Figure 5 An illustration in the coronal plane showing placental attached to the uterus from 9:00 o'clock to 1:00 o'clock at 10 weeks, 23 weeks and 37 weeks of gestation. Adapted from a published figure in reference 43

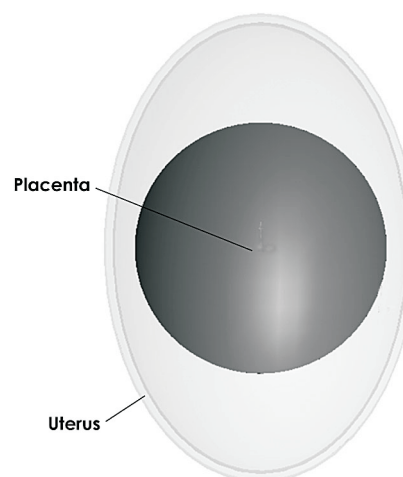


Figure 6 Illustration showing the area of contact between a term uterus and a term placenta based on a circular placenta and a prolate ellipse uterus. In this illustration the anterior wall of the uterus is cut away and the placenta is attached to the posterior wall of the uterus. The placenta is in contact with 20% of the wall of the internal surface of the uterus

Uterine artery blood flow

At term

Wide variations in uterine artery blood flow values at term pregnancy have been published. Uterine artery blood flow near term is considered to be approximately 800 ml/min⁵⁴. This places a boundary for bilateral uterine artery blood flow change during pregnancy: 100 ml/min before pregnancy to 800 ml/min near term. Blood flow during pregnancy must fall between these two extremes.

Throughout pregnancy, blood flow increases week by week. The rate of change, however, is not constant and the overall shape of the blood flow curve during pregnancy is the typical ‘S’ shaped curve seen throughout biology. Twenty weeks’ gestation, or mid-pregnancy, appears to be the inflection point of

the curve. Before 20 weeks, the increase in uterine blood flow accelerates; after that, it decelerates.

Uterine artery blood flow increases secondary to increases in both uterine artery diameter and uterine artery red blood cell velocity (Figure 7).

Ovarian vein diameter – increase during pregnancy

The ovarian and uterine veins grow sufficiently in diameter during pregnancy to accommodate the large increase in blood flow to the uterus. No published uterine vein diameter measurements exist taken during pregnancy.

If one uses the surgical estimate of 9 mm for the non-pregnant ovarian vascular pedicle diameter and compares that with 3.9 mm by CT measurement of the ovarian vein alone, then proportionately at 38.9 weeks the ovarian vein, alone, would be 18.4 mm in diameter, a growth in diameter of 372%.

Ovarian vein diameter – decrease following pregnancy (smaller but still enlarged)

After delivery, when blood flow to the uterus returns to normal, the uterine and ovarian veins decrease in diameter. However, they do not return to their nulliparous diameters.

Arteries and veins in the pregnant woman

Taken together the anatomical changes of the arteries and veins of the uterus and ovary during mid-pregnancy are shown in Figure 8. See Figure 9 for a comparison with a non-pregnant woman. The ovarian and uterine veins have grown in diameter but growth is much more pronounced in the ovarian veins which empty into the inferior vena cava below the insertions

of the renal veins. The ovarian and uterine arteries have grown, too, but the growth of the uterine arteries has outstripped growth of the ovarian arteries. The tortuosity of the uterine and ovarian arteries has been extended as they were stretched with enlargement of the uterus. Though quite rare, spontaneous rupture of enlarged uterine or ovarian veins and uterine arteries has been reported^{54,55}.

UTERINE BLOOD VESSELS DURING PREGNANCY – LABOR AND DELIVERY

Mechanics of placental separation

After delivery of a baby, the uterus continues to contract. During these postpartum contractions, the

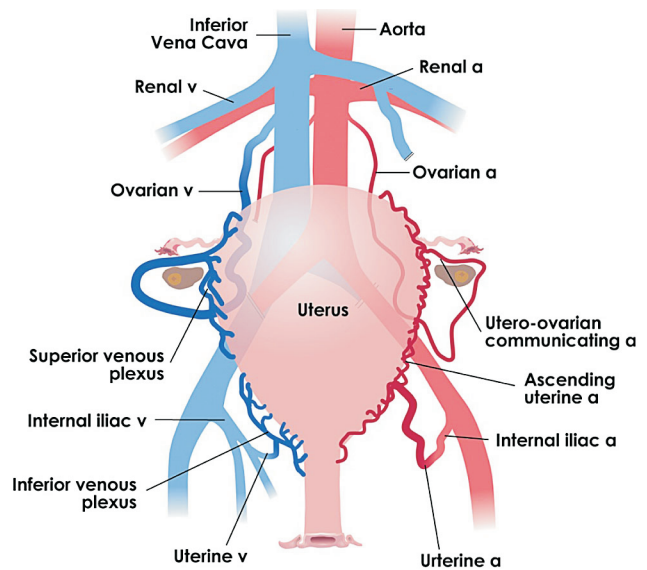


Figure 8 Composite drawing of uterine arteries and veins and their systematic origins and insertions, respectively, at 24 weeks of gestation

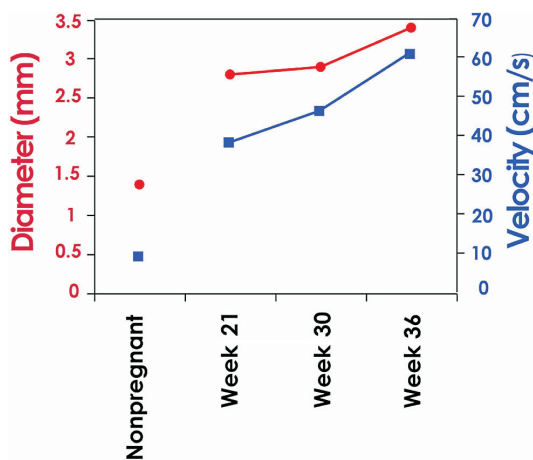


Figure 7 Graph of uterine artery diameter and red blood cell velocity in women not pregnant and at 21, 30 and 36 weeks of gestation. Uterine artery diameter is displayed as circles; velocity, as squares. Adapted from published tabular data¹⁷. The most rapid increases in diameter and red blood cell velocity occur between the time a woman gets pregnant and 21 weeks pregnancy

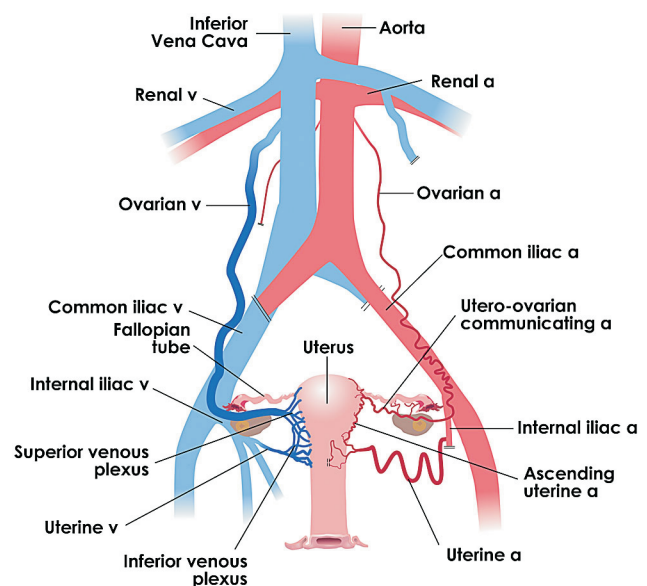


Figure 9 Composite drawing of left-sided uterine arteries and right-sided uterine veins showing their systematic origins and insertions in a non-pregnant woman

myometrium of the placental footprint becomes smaller and smaller, while the placenta, itself, being a solid structure, remains fixed in shape. The drop in the myometrial contact area of the placental footprint without change in the shape of the placental causes shear stress between the placenta and the uterus. The shear stresses at the junction between the placental base and the placental footprint tear the placenta away from the uterus, creating 200 torn and ragged uteroplacental arteries and veins!

Goto⁵⁶ defined three types of placental separation. In type I separation (53.0%), as a whole, the placenta smoothly slides off the wall of the uterus. In type II (35.5%), a portion of the placenta and fetal membranes adhere to the uterine wall preventing smooth separation. In type III (11.5%), a retroplacental hematoma forms between the uterine wall and the placenta during separation.

PRACTICE POINTS

- The uterus is a highly vascular organ with two arterial and two venous systems intertwined
- During pregnancy clotting and fibrinolytic factors in the blood build up in concentration in mother's blood. Throughout pregnancy both systems are active. However, the clotting system is a little more active than the fibrinolytic system. This imbalance during pregnancy has consequences for maternal health
- To stop hemorrhage following delivery, during true labor clotting commences within the uterine circulation
- Two hours following delivery, fibrinolysis commences in the uterus to restore blood flow.

References

1. Lipshutz B. A composite study of the hypogastric artery and its branches. *Ann Surg* 1918;67:584–608
2. Borell U, Fernstrom I. The adnexal branches of the uterine artery. An arteriographic study in human subjects. *Acta Radiol* 1953;40:561–82
3. Fernstrom I. Arteriography of the uterine artery. *Acta Radiol* 1955;S122:3–128
4. Radberg C, Wickbom I. Pelvic angiography and pneumoperitoneum in the diagnosis of gynecologic lesions. *Acta Radiol Diagn (Stockh)* 1967;6:133–44
5. Merland JJ, Chiras J. Arteriography of the pelvis. Diagnostic and therapeutic procedures. Berlin, Heidelberg, New York: Springer-Verlag, 1981
6. Pron GE, Common AA, Sniderman KW, Bell SD, Simons ME, Vanderburgh LC. Radiological embolization of uterine arteries for symptomatic fibroids: preliminary findings of a Canadian multicenter trial [Abstract]. *Minim Invasive Ther Allied Technol* 1998;7(Suppl 1):26
7. Pelage JP, De Dref O, Soyer P, et al. Arterial anatomy of the female genital tract: Variations and relevance to transcatheter embolization of the uterus [Pictorial Essay]. *AJR* 1999;172:989–94
8. Borell U. [Arteriography of uterine and adnexal vessels]. *Geburtshilfe Frauenheilkd* 1955;15:497–513
9. Nikolic B, Spies JB, Abbara S, Goodwin SC. Ovarian artery supply of uterine fibroids as a cause of treatment failure after uterine artery embolization: A case report. *J Vasc Interv Radiol* 1999;10:1167–70
10. Binkert CA, Andrews RT, Kaufman JA. Utility of non-selective abdominal aortography in demonstrating ovarian artery collaterals in patients undergoing uterine artery embolization for fibroids. *J Vasc Interv Radiol* 2001;12:841–5
11. Razavi MK, Wolanske KA, Hwang GL, Sze DY, Kee ST, Dake MD. Angiographic classification of ovarian artery-to-uterine artery anastomoses: initial observations in uterine fibroid embolization. *Radiology* 2002;224:707–12
12. Pelage JP, Walker WJ, Le Dref O. Re: utility of nonselective abdominal aortography in demonstrating ovarian artery collaterals in patients undergoing uterine artery embolization for fibroids. *J Vasc Interv Radiol* 2002;13:656
13. Worthington-Kirsch RL, Andrews RT, Siskin GP, et al. Uterine fibroid embolization: Technical aspects. *Tech Vasc Interv Radiol* 2002;5:17–34
14. Beliaeva YA. [Age-related properties of the arteries of the large ligament]. *Arkhiv Anatomii* 1965;48:98–107
15. Zamudio S, Palmer SK, Droma T, et al. Effect of altitude on uterine artery blood flow during normal pregnancy. *J Appl Physiol* 1995;79:7–14
16. Thaler I, Manor D, Itskovitz J, et al. Changes in uterine blood flow during human pregnancy. *Am J Obstet Gynecol* 1990;162:121–5
17. Palmer SK, Zamudio S, Coffin C, Parker S, Stamm E, Moore LG. Quantitative estimation of human uterine artery blood flow and pelvic blood flow redistribution in pregnancy. *Obstet Gynecol* 1992;80:1000–6
18. Gomez-Jorge J, Keyoung A, Levy EB, Spies JB. Uterine artery anatomy relevant to uterine leiomyomata embolization. *Cardiovasc Intervent Radiol* 2003;26:522–7
19. Lippert H, Pabst R. Arterial variations in man. Munich: Bergmann, 1985
20. Holmgren B. Some observations on the blood vessels of the uterus under normal conditions and in myoma. *Acta Obstet Gynecol Scand* 1938;18:192–213
21. Cooper JM, Dickner SK. A Doppler-guided transvaginal approach leading to uterine artery occlusion may be a less invasive means to control uterine perfusion [Abstract]. *J Am Assoc Gynecol Laparosc* 2002;9:S12
22. Frates RE. Selective angiography of the ovarian artery. *Radiology* 1969;92:1014–9
23. Shlansky-Goldberg R. Uterine artery embolization: Historical and anatomic considerations. *Semin Intervent Radiol* 2000;17:223–36
24. Pelage JP, Cazejust J, Pluot E, et al. Uterine fibroid vascularization and clinical relevance to uterine fibroid embolization. *Radiographics* 2005;25 (Suppl 1):S99–117
25. Borell U, Fernstrom I. The ovarian artery: an arteriographic study in human subjects. *Acta Radiol* 1954;42:253–65
26. Kozik W. [Arterial vasculature of ovaries in women of various ages in light of anatomic, radiologic and microangiographic examinations]. *Ann Acad Med Stetin* 2000;46:25–34
27. Kozik W, Czerwinski F, Pilarczyk K, Partyka C. [Arteries of the hilum and parenchymal part of the ovary in reproductive age in microangiographic studies]. *Ginekol Pol* 2002;73:1173–8
28. Ide P, Bonte J. [Arterial supply of the uterine isthmus and cervix]. *Bull Soc Roy Belg Gynec Obstet* 1964;34:365–73
29. Palacios Jaraquemada JM, Monaco RG, Barbosa NE, Ferle L, Iriarte H, Conesa HA. Lower uterine blood supply: Extra-uterine anastomotic system and its application in surgical devascularization techniques. *Acta Obstet Gynecol Scand* 2007;86:228–34
30. Farrer-Brown G, Beilby JO, Tarbit MH. The blood supply of the uterus. 1. Arterial vasculature. *J Obstet Gynaecol Br Commonw* 1970;77:673–81
31. Sampson JA. The blood supply of uterine myomata. *Surg Gynecol Obstet* 1912;14:215–34

32. Lindenbaum E, Brandes JM, Itskovitz J. Ipsi- and contralateral anastomosis of the uterine arteries. *Acta Anat* 1978;102:157-61
33. Chait A, Moltz A, Nelson JH Jr. The collateral arterial circulation in the pelvis. An angiographic study. *Am J Roentgenol Radium Ther Nucl Med* 1968;102:392-400
34. Mattingly RF, Thompson JD. *Te Linde's Operative Gynecology*, 6th edn. Philadelphia: J. B. Lippincott Company, 1985
35. Nasu K, Fujimoto H, Yamamoto S, Naitou H, Maekawa I, Yasuda S, Itou H. [Collaterals after flow alternation in pelvic arteries: precondition for pelvic reservoir therapy]. *Nippon Igaku Hoshasen Gakkai Zasshi* 1998;58:204-11
36. Chitrit Y, Guillaumin D, Caubel P, Herrero R. Absence of flow velocity waveform changes in uterine arteries after bilateral internal iliac artery ligation. *Am J Obstet Gynecol* 2000;182:727-28
37. Lindenbaum E, Brandes JM, Itskovitz J. Ipsi- and contralateral anastomosis of the uterine arteries. *Acta Anat* 1978;102:157-61
38. Hunter J. On the structure of the placenta. In: Hunter J (Editor). *Observations on certain parts of the animal economy*. London: No. 13, Castle-Street, Leichestre-Square, 1786:127-40
39. Boyd JD, Hamilton WJ. *The Human Placenta*. Cambridge, England: W. Heffer & Sons Ltd, 1970
40. King DL. Placental migration demonstrated by ultrasonography. A hypothesis of dynamic placentation. *Radiology* 1973;109:167-70
41. McClure N, Dornal JC. Early identification of placenta praevia. *Br J Obstet Gynaecol* 1990;97:959-61
42. Taipale P, Hiilesmaa V, Ylostalo P. Transvaginal ultrasonography at 18-23 weeks in predicting placenta previa at delivery. *Ultrasound Obstet Gynecol* 1998;12:422-5
43. Hamilton WJ, Boyd JD, Mossman HW. *Human embryology*. Baltimore: Williams & Wilkins Company, 1962
44. Ananth CV, Wilcox AJ, Savitz DA, Bowes WA Jr, Luther ER. Effect of maternal age and parity on the risk of uteroplacental bleeding disorders in pregnancy. *Obstet Gynecol* 1996;88:511-6
45. Alvarez H, Caldeyro R. [Contractility and premature separation of placenta]. *An Fac Med Montevideo* 1950;35:682-95
46. Minh HN, Smadja A, Orcel L. [Intimate mechanism of premature separation of the normally inserted placenta]. *J Gynecol Obstet Biol Reprod (Paris)* 1977;6:301-10
47. Amoa AB, Augerea L, Klufio CA. Antepartum haemorrhage at the Port Moresby General Hospital: a retrospective study of 130 consecutive cases. *P N G Med J* 1992;35:17-22
48. Pumarino R. [Premature separation of placenta and uterine apoplexia]. *Bol Hosp Vina Del Mar* 1951;7:30-4
49. Dommissie J, Tiltman AJ. Placental bed biopsies in placental abruption. *Br J Obstet Gynaecol* 1992;99:651-4
50. Bekova KS. [The relationship between the weight of the fetus, the weight and surface area of the placenta and the structure of the veins of the fetal surface of the placenta]. *Vopr Okhr Materin Det* 1972;17:20-2
51. Woods DL, Malan AF, de V Heese H, van Schalkwyk DJ. Placental size at birth. *S Afr Med J* 1978;54:778-9
52. Mapfurira MJ, Msamati BC, Banadda BM. Correlations between weights of newborn babies, placental parameters and gestational age. *Cent Afr J Med* 1992;38:414-20
53. Blickstein I, Ron A. Can placental surface area and neonatal weight be predicted from placental surface measurements? Preliminary observations on normal-term pregnancies. *Gynecol Obstet Invest* 1995;40:253-6
54. Maguire P. Spontaneous rupture of utero-ovarian vessels during pregnancy. *J Okla Med Ass* 1962;55:123-4
55. Steinberg LH, Goodfellow C, Rankin L. Spontaneous rupture of the uterine artery in pregnancy. *Br J Obstet Gynaecol* 1993;100:184
56. Goto M. [A survey of placental separation by real-time B-mode scanning]. *Nippon Sanka Fujinka Gakkai Zasshi* 1984;36:1171-9