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Uterovaginal Blood Supply: the S1 and S2 Segmental Concepts and their Clinical Relevance

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ORIGIN OF THE CONCEPT

Recent careful and precise anatomic studies in cadavers demonstrate that the female reproductive system has two separate and completely distinguishable blood supplies. These investigations confirm earlier observations[1] that the blood supplies are anastomotic in nature[2,3]. The distinctive natures of the two areas and their differing blood supplies are shown in the sagittal view of the pelvis as depicted in Figure 1. In practical terms, the two areas can be distinguished by drawing a line perpendicular to the posterior wall of the bladder.

As defined in the writings of Palacios-Jaraquemada, who first used this terminology in 2005, the S1 segment comprises the body of the uterus[2]. In this construct, the S2 segment corresponds to the lower uterine segment, cervix, upper part of the vagina and the respective parametria.

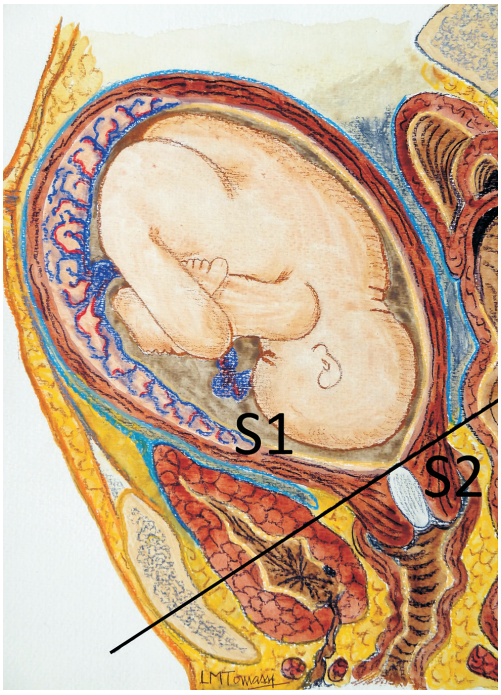

Figure 1 Sagittal line demarcating the S1 and S2 areas of blood supply to the uterus and upper vagina at term.

The S1 segment is supplied by ascending branches of the uterine artery and, to a lesser extent, by the descending branches of the ovarian artery. Rarely, the round ligament artery contributes to the collateral blood supply of the uterus. In contrast, the S2 segment is supplied by branches of the uterine, cervical, upper vesical, vaginal and pudendal arteries. *All of these blood vessels are located subperitoneally*.

A thorough understanding of the differences in the blood supply to both uterine segments (S1 and S2) underlies the proper selection of a therapeutic intervention, be it surgical or radiological, for the treatment of postpartum hemorrhage (PPH). It is possible that the recent increases in maternal mortality in several countries are related to the changes in the rates of PPH, with more severe bleeding related the increased incidence of adherent placentas.

ANATOMIC EVIDENCE

Figure 2 shows a fetal angiographic preparation which facilitates an understanding of the rich arterial anastomotic system between the left uterine artery and the corresponding left lower and middle vaginal arteries[1]. These arteries also anastomose with the descending branches of the uterine artery on the ipsilateral side. In practical terms, this means that the whole of one half of the lower part of the uterus and the upper part of the vagina receives blood from an interconnected system. In this figure, the uterine artery and the middle left vaginal artery are branches of the anterior division of the internal iliac artery, whereas the lower left vaginal artery is a branch of the posterior division of the internal iliac artery. (Note: it could also arise from the internal pudendal artery, which in turn is a branch of the posterior division of internal iliac.)

It is crucial to understand that the system illustrated in this fetal angiogram is fully maintained in the adult and is the system responsible for continuation of uterine blood supply when the uterine arteries are embolized.

The cross anastomoses between the right and the left uterine arteries over the uterine surface shown in

Figure 3 are not evident to the naked eye in either the non-pregnant or the normal pregnant uterus. However, *in the presence of abnormal placentation, they become grossly enlarged and engorged, and are visible on the uterine surface, especially in the lower segment*. This is because the lower segment of pregnant uterus is stretched and thinned.

Figure 4 is a rare adult angiographic preparation showing the features previously described in Figures 2 and 3 which derive from a fetus. First, the anatomy present in the fetus is present and persists through adult life. Second, the bilateral anastomotic flow pathways between the vaginal and uterine arteries remain visible. Third, the side-to-side anastomoses between both uterine arteries are present. This latter point has exceedingly practical implications, because *if one is going to perform stepwise devascularization in cases of major PPH, it must be bilateral (as is the case in bilateral uterine artery embolization) or the procedure will be less than effective as a result of the cross anastomoses*. In contrast, vertical uterine compression sutures (B-Lynch, Hayman) act over both systems.

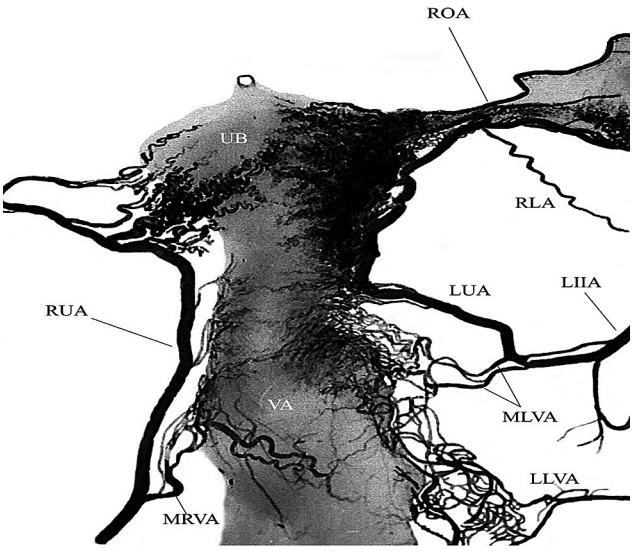

Figure 2 Fetal cadaveric angiographic preparation demonstrating the rich arterial anastomotic system between the left uterine artery and the left lower and middle vaginal arteries. LIIA, left internal iliac artery; LLVA, lower left vaginal artery; LUA, left uterine artery; MLVA, middle left vaginal artery; MRVA, middle right vaginal artery; RLA, round ligament artery; ROA, right ovarian artery; RUA, right uterine artery; UB, uterine body; VA, vagina. From Palacios-Jaraquemada *et al.*, 2007[3], with permission

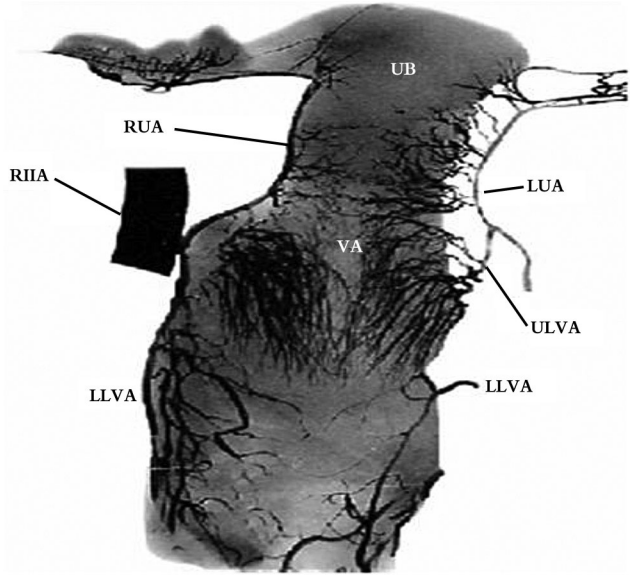

Figure 3 Another view of the preparation shown in Figure 2 but demonstrating cross anastomoses between the right and the left uterine arteries over the uterine surface. LLVA, left lower vaginal artery; LUA, left uterine artery; RIIA, right internal iliac artery; RUA, right uterine artery; UB, uterine body; VA, vagina. From Palacios-Jaraquemada *et al.*, 2007[3], with permission

REASONS FOR FAILURE OF SOME INTERVENTIONAL RADIOLOGICAL PROCEDURES

Figure 4 demonstrates the anatomic basis for the failures of some uterine embolization procedures. It also provides an explanation of why non-target embolizations occur, especially in the bladder where they may cause necrosis. In the majority of instances, the interventional radiologists cannot explain these occurrences, because the embolization material has been injected into the uterine artery and the damage is in the area supplied by the vesical artery.

Under normal circumstances, the connection between the uterine artery and the vesical artery is microscopic; however, in the presence of abnormal placentation and also because of the effect of vascular growth factors, these vessels enlarge and represent neovascularization as they lack a tunica media which in turn allows them to become high flow low resistance reservoirs.

When patients undergo interventional radiological procedures in situations involving abnormal placentation, it is important to be aware of the potential for abnormal connections between uterine and vesical arteries. If the embolization material is injected under pressure and if the particle size is small (usually less than 700 μm), then it is possible to have non-target embolization to the bladder which occasionally becomes necrotic (see Chapter 49 for further reading).

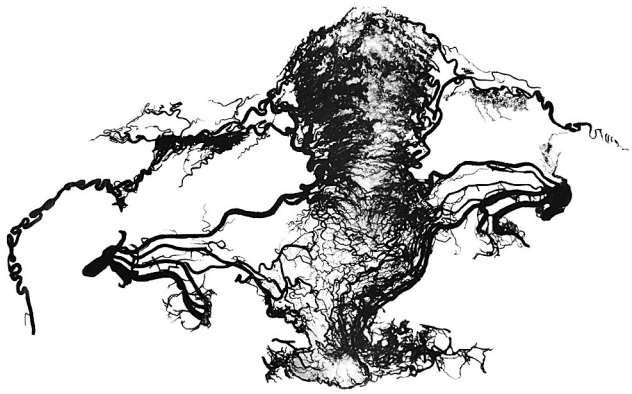

Figure 4 Adult angiographic preparation which shows the features previously described in Figures 2 and 3. From Rohen *et al.*, 2003[5], with permission

The radiograph shown in Figure 5 illustrates the dense anastomotic system present between the vaginal and uterine arteries. In this patient, the catheter was placed at the level of the internal pudendal artery. The contrast material was injected and passed as high as the uterus via intercommunicating anastomoses.

This radiograph can also explain two common clinical scenarios. In the first, where a patient suffers severe PPH following normal vaginal delivery, the interventional radiologist performs embolization of uterine artery and a second embolization attempt is made at the anterior division of internal iliac arteries. Both may fail to produce significant reduction in the intensity of bleeding. This failure can be explained by the presence of rich anastomoses between the internal pudendal anastomotic branches (S2 segment) and the uterine artery (S1) and also by the coexisting uncorrected clotting abnormalities.

In the second clinical scenario, a traumatic instrumental delivery results in deep vaginal tears or causes a retroperitoneal hematoma. When the bleeding cannot be controlled by routine measures, the clinician often elects to perform hysterectomy or uterine artery embolization, either one of which has its main effect on the S1 segment. Both can fail, however, because the origin of the bleeding is from the vessels that arise in the S2 segment shown in Figure 5 (internal pudendal artery).

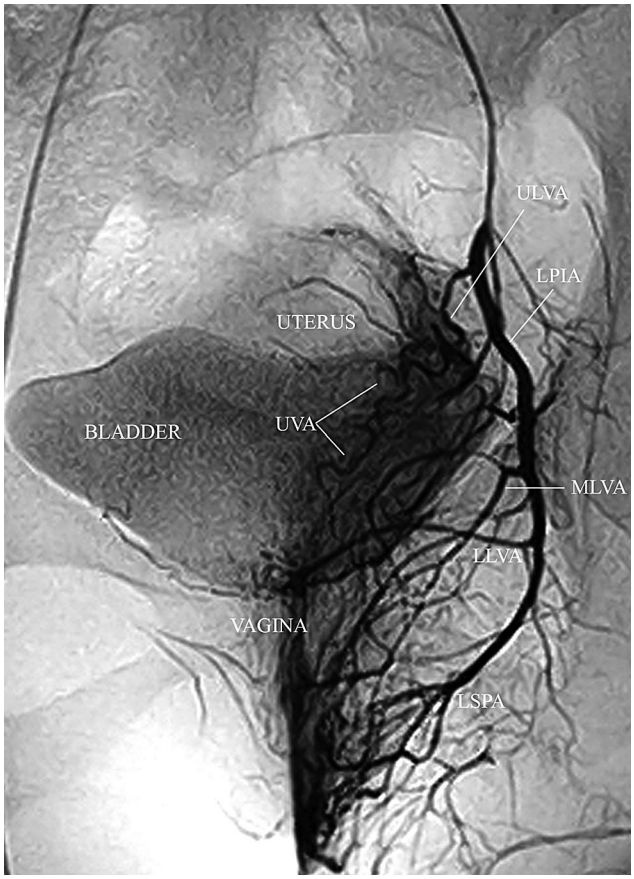

Figure 5 Angiographic illustration of the dense anastomotic system between the vaginal and uterine arteries. From Palacios-Jaraquemada *et al.*, 2007[3], with permission

COMMON PERCEPTIONS AND MISPERCEPTIONS

Students of history are well aware that if one does not learn from the mistakes of the past, one is bound to repeat them. The same is true in anatomy, where 18th century dissections provide important clues to the 21st century dilemmas. Figure 6, to the best of the senior author's (J.P.J.) knowledge, is the first illustration depicting the relationship between the relative diameters of the vaginal and uterine arteries. The common perception is that the diameter of the uterine artery is larger than that of the vaginal artery.

As shown in Figure 6, however, this is not always the case, and the French anatomist's observation[4] has been duplicated by later authors using specialized dissection techniques[3]. In a series of 39 cadaver dissections, the first author demonstrated that the diameter of the inferior vaginal artery was 33% thicker than that of the uterine artery (Table 1).

This important difference between the major blood supplies of the S2 segment and the S1 segment should not be

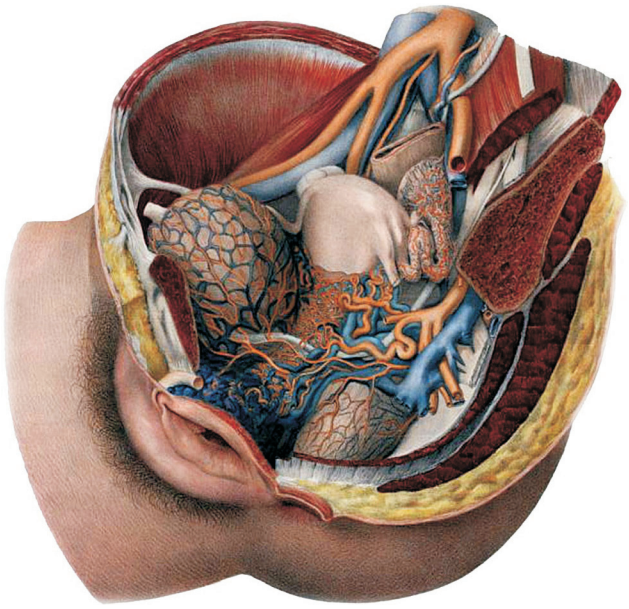

Figure 6 Eighteenth century illustration of the relative diameters of the uterine and vaginal arteries, respectively. From Bourgery, 1833[4]

Table 1 Lower uterine vascular supply. From Palacios-Jaraquemada *et al.*, 2007[3], with permission

Upper pedicle	Uterine artery	100% from IIA
Middle pedicle	Cervical artery	67% from UA 23% from Vas 10% from LVeA
Lower pedicle	UVA: 18% from UA MVA: 11% from IIA LVA: 71% from PIA	 75% as descending branch 25% as ascending branch
Sectional diameters	UA: 1.81 mm	MaVA: 1.88 mm

IIA, iliac internal artery; UA, uterine artery; Vas, vaginal arteries; LVeA, lower vesical artery; UVA, upper vaginal artery; MVA, middle vaginal artery; LVA, lower vaginal artery; PIA, pudendal internal artery; MaVA, main vaginal artery

underestimated. When the obstetrician is trying surgically to correct a PPH involving a traumatic vaginal tear, hysterectomy will not solve the problem. Although it is commonly believed that patients die from uncontrolled uterine arterial tears and do not die from vaginal arterial tears, this misconception is secondary to knowledge gained from reading standard textbooks which fail to explain the differences in the blood supplies of the S1 and S2 segments. Figures 7 and 8 illustrate these anatomic points with cadaver specimens.

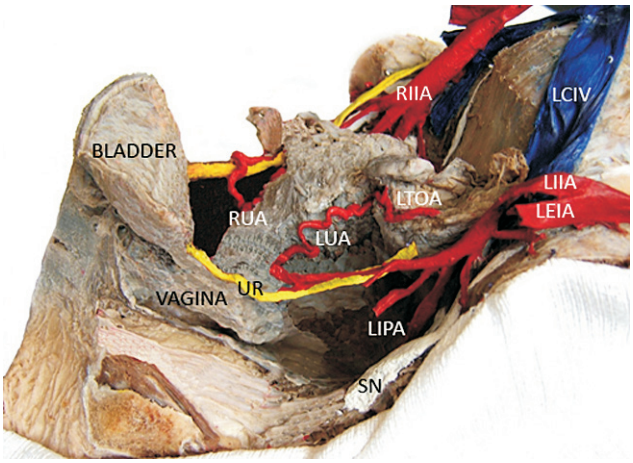

Figure 7 Normal non-pregnant uterine blood supply as commonly appreciated by the general physician. It consists of the uterine artery arising from the anterior division of the internal iliac artery and the ovarian artery which arise directly from the aorta. This is the S1 segmental blood supply area. LIPA, left internal pudendal; LSPA, left superficial perineal; LLVA, lower left vaginal; and LLVeA, lower left vesical artery; LCIA, left common iliac artery; LEIA, left external iliac artery; LIIA, left internal iliac artery; LIPA, left internal pudendal artery; LLVA, lower left vaginal artery; LLVeA, lower left vesical artery; LSPA, left superficial perineal artery; LTOA, left tubo-ovarian artery; LUA, left uterine artery; RIIA, right internal iliac artery; ROA, right ovarian artery; RUA, right uterine artery; SN, schiatic nerve; ULVeA, upper left vesical artery; UR, ureter; URVeA, upper right vesical artery

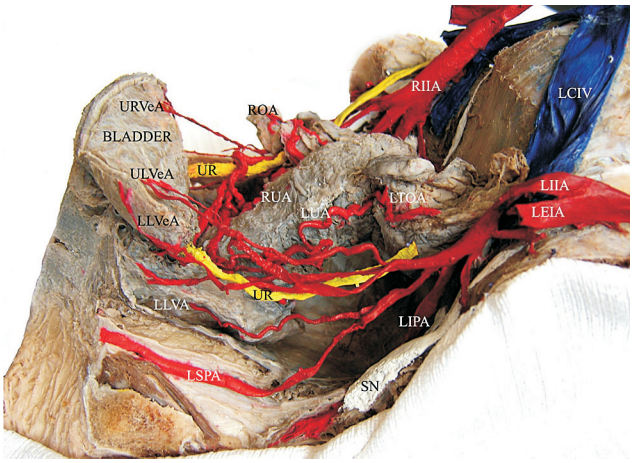

Figure 8 High density and close proximity of the numerous vessels of the S2 segment which comprise branches that mainly arise from the internal pudendal artery. See Figure 7 for abbreviations. From Palacios-Jaraquemada *et al.*, 2007[3], with permission

CLINICAL APPLICATION

Figures 9 and 10 represent initial views of two types of topography associated with abnormal placentation. In Figure 9, the abnormal placentation is located in the S1 segment. The importance of Figure 9 is that the clinician should ensure that there is no abnormal vasculature in the area where the hysterotomy is made to deliver the baby. If PPH supervenes, therapy with subtotal hysterectomy is an appropriate intervention.

In contrast, in Figure 10, the abnormal placentation occupies the S2 segment. The first challenge for the clinician is safely to deliver the baby and safeguard the health of the mother. This can be accomplished by delivering the baby through a uterine fundal incision without any attempt to touch, detach or deliver the placenta. In other words, the placenta will be left *in situ*, the umbilical cord cut short, and the fundus closed. Immediate hysterectomy in this kind of circumstance will precipitate catastrophic hemorrhage putting mother's life at risk.

When the placenta is left *in situ* the following three options may be considered. First is to observe closely the patient in a high dependency environment with the risk of sepsis that could complicate systemic inflammatory response syndrome (SIRS), or hemorrhage and coagulation abnormalities (disseminated

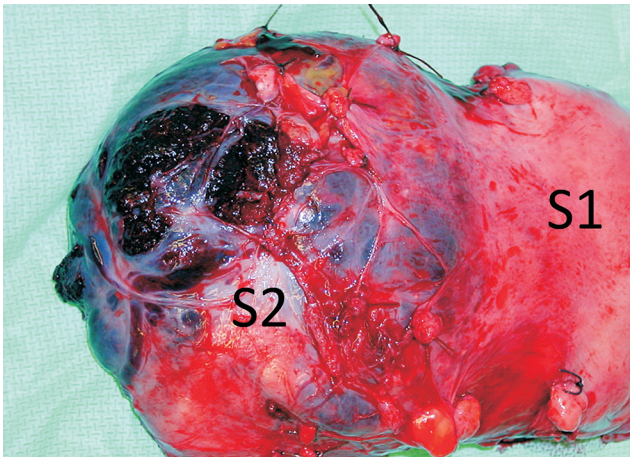

Figure 9 Abnormal placentation in S1 segment

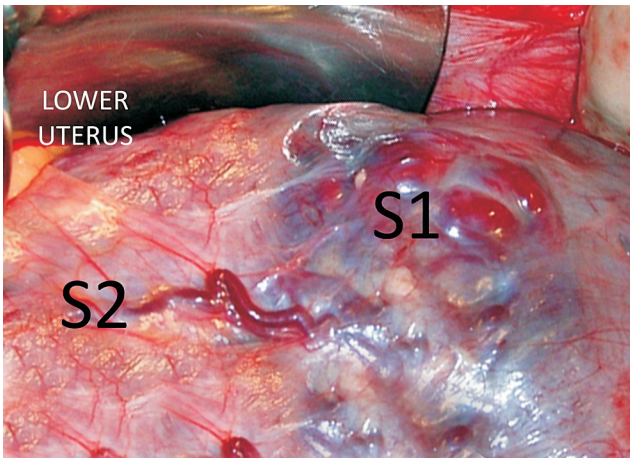

Figure 10 Abnormal placentation in S2 segment

intravascular coagulopathy). Second is to transfer the patient to a tertiary center where hysterectomy can be performed with the full team. Third, embolization may be considered if the patient experiences intermittent hemorrhage with the placenta *in situ* or an adjuvant treatment for a planned hysterectomy.

DISSECTION COURSES AND SELF LEARNING WITH VIDEOS

The challenge for the general obstetrician is to realize that a thorough understanding of the differences between the S1 and S2 segments will be of great value in planning safe, appropriate intervention for PPH. The authors propose that the general obstetrician who regularly performs instrumental deliveries should make every effort to become familiar with the vasculature of the S2 segment and safe access to this area. To gain access to the upper part of the vagina, one should have thorough knowledge of safe dissection of ureters (Video 1) (videos can be viewed via *The Global Library of Women's Medicine* at www.glowm.com). Access to the middle and lower part of the vagina and also to the roof of ischiorectal fossa and internal fascicle of levator ani muscle (pubo rectalis part) is shown in Video 2. This space is accessed by blunt dissection of the retropubic space (space of Retzius, which in the past was used for Burch colposuspension).

One means by which suggested anatomic knowledge and skill can be gained and maintained is by attending fresh cadaver dissection courses (hyperlink: St George's through RCOG at http://www.bgcs.org.uk/events/2012/03/gynaecological-oncology-cadaveric-dissection-course.html). Another way is to access surgical dissection videos on a regular basis. The authors also propose that physicians and their co-workers discuss the case in detail when any surgical intervention has failed.

CONCLUSION

The chapter closes with Video 3 which demonstrates all of the anatomic points discussed above. A major point of the video is to show that after bilateral uterine arterial embolization, the uterine blood flow is compensated by the blood which comes from the inferior vaginal arteries through the anastomotic channels described above.

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