Pregnancy is a high-flow, low-resistance state of cardiovascular homeostasis associated with remarkable hemodynamic changes. In the human and most other mammalian species, cardiac output increases during early pregnancy, reaching a peak of 30%-50% above nonpregnant values at 20–24 weeks’ gestation. It remains unchanged or falls slightly thereafter [1, 2]. This increment in maternal cardiac output is accounted for by the dramatic increase in uterine blood flow needed to satisfy the metabolic demands of the conceptus and by the significant increase in blood flow to certain nonreproductive organs that are involved in the physiologic adjustments to pregnancy (e.g., kidneys, skin, gastrointestinal tract) [3]. There is also a 20% increase in maternal heart rate, a slight fall in mean arterial pressure, and a significant decrease in systemic vascular resistance – hence the need for the 40% expansion in maternal blood volume during normal pregnancy [4].

In addition to these alterations, there is the development of attenuated pressor responses to several vasoactive agents during normal pregnancy. These agents include angiotensin II, a-agonists, and arginine vasopressin [5–7].

The hemodynamic changes described above are mandatory for the continuous growth and development of the uteroplacental and fetoplacental circulations. Interference with the normal growth and development of the uteroplacental and fetoplacental circulations may result in disruption of the oxygen and nutrient supply to the fetus, leading to reprogramming of fetal development. It ultimately may result in intrauterine growth retardation, one of the leading causes of perinatal mortality and morbidity. Thus assessment of uteroplacental and fetal hemodynamics is of primary clinical importance. Until recently, fetal heart rate monitoring and the biophysical profile have been useful routine surveillance techniques for detecting the fetus that is either already asphyxiated or soon likely to become so. Despite three decades of experience in this field, controversies still exist regarding the effectiveness of these tests for reducing perinatal mortality and morbidity.

The use of Doppler ultrasound technology for investigating human fetal and uteroplacental hemodynamics offers a novel approach to the identification of a wide variety of disorders related to pregnancy. Knowledge of the normal physiology of maternal and fetal hemodynamics is a prerequisite for the development of pathophysiologic hypotheses that can lead to the establishment of clinical principles for investigating the fetomaternal circulatory status. Most of the present knowledge of fetal and maternal cardiovascular physiology is derived from animal experiments, particularly in the pregnant sheep; there is a paucity of comparable information relating to human pregnancy. Although the general course of the fetal and maternal circulations are similar in various mammalian species, it is important to appreciate that significant variations are known to exist among species. This chapter briefly reviews the basic hemodynamic concepts of the fetal and maternal circulations relevant to the potential clinical application of Doppler ultrasound technology for the assessment of fetal well-being.

**Uterine Circulation**

**Functional Anatomy of the Uteroplacental Circulation**

The main uterine artery branches off the internal iliac artery. At the level of the internal cervical os it bifurcates into the descending (cervical) and ascending (corporal) branches. At the uterine tubal junction the ascending branch turns toward the ovary and anastomoses with the ovarian artery to form an arterial arcade that perfuses the internal genital organs. The tortuous ascending uterine artery gives off approximately eight branches – the arcuate arteries which extend inward for about one-third the thickness of the myometrium and envelope the anterior and posterior walls of the uterus [8, 9]. The origin of these branches is asymmetric; some are large and thick and supply a large area of the uterus, whereas others are thin and supply smaller areas of the uterine wall. These arteries have a tortuous course and anastomose with the corresponding arteries from the other side closer to the midline [9, 10]. From this net-
work arise the radial arteries, which are directed toward the uterine mucosa. The spiral arteries undergo cyclical changes that are synchronous with the ovarian cycle. During normal pregnancy, trophoblastic cells enter the lumen of the spiral arteries, partially replacing the endothelium and progressing down the inner wall of the arteries up to the level of the endometrium. At the end of the third month of pregnancy the invading trophoblast begins to destroy the elastic lamina, and at 16–22 weeks' gestation it replaces the smooth muscle elements of the intramyometrial portion of the spiral arteries and then degenerates [11, 12]. This transformation eventually leads to formation of a low-resistance vascular system in which relatively large maternal arteries pump blood directly into the intervillus spaces (Fig. 9.1). Failure or impairment of the trophoblastic cells to invade the spiral arteries has been associated with pregnancy-induced hypertension and intrauterine growth restriction [14, 15].

Anatomic and radiographic studies and uterine perfusion have demonstrated the richness of the arterial anastomosis of the human uterine circulation [8–10, 16, 17]. These anastomoses include ipsilateral connections between uterine and ovarian arteries, ipsilateral anastomosis of vessels of differing diameter of the uterine branches (arcuate and radial arteries), contralateral anastomosis between the right and left uterine arteries and their branches, and extraterine connections between the uterine circulation and the systemic circulation (e.g., inferior mesenteric, middle sacral, and inferior epigastric arteries). This vast ipsilateral and contralateral anastomotic network ensures ample uteroplacental perfusion and may be activated to provide alternative routes of blood supply to the placenta. Data from human pregnancy indicate that anastomotic connections increase in size and are of functional significance after occlusion of major vessels [16, 17]. In the rhesus monkey the contribution of the ovarian artery to uterine blood flow in the nonpregnant state and during early pregnancy is negligible. During late pregnancy it contributes about half of the flow to the upper third of the uterus [18]. Studies in sheep have shown that the uterine arteries contribute approximately 80% of the total uterine flow [19, 20], and that functional arterial anastomoses are present between the right and left sides of the uterine vasculature. Short-term changes in flow on one side are accompanied by compensatory changes in the flow rate on the contralateral side [21]. The venous drainage of the placenta usually follows its arterial supply, but it has also been demonstrated that in some instances the drainage of placental blood shifts rapidly between the two uteroovarian veins [22]. Thus the uteroplacental circulation is a dynamic system in which the magnitude of blood flow through a single vessel may vary considerably over a short time, making single vessel measurements of blood flow sometimes difficult to interpret.

**Alters in Uterine Blood Flow**

Only limited information is available regarding the changes in uteroplacental blood flow throughout human pregnancy. Total uterine blood flow is estimated to increase from approximately 50 ml/min during early pregnancy to 500 ml/min near term. However, these figures were derived from studies that employed a diffusion-equilibrium technique [23, 24] or radioisotope-dilution methods [25–29], the accuracy of which is questionable [30].

Our most complete knowledge of uterine vascular changes have been obtained in farm animals (sheep, pigs), which have been shown to develop some hemo-

---

**Fig. 9.1.** Fully developed changes in the uteroplacental arteries of normal pregnancy. The hatched portions of the wall of spiral arteries indicate the extent of the physiological changes. IVS intervillus space. (Reprinted from [13] with permission)