Exploring the functionality of the adult’s venous compartment is of interest to the field of obstetrics

W. Gyselaers, MD PhD

Dept. Obstetrics & Gynaecology, Ziekenhuis Oost Limburg, Genk Belgium
Dept. of Physiology, Hasselt University, Diepenbeek Belgium

Correspondence at: wilfried.gyselaers@zol.be

Key words: Maternal veins, gestational physiology, venous hemodynamics, preeclampsia, gestational cardiovascular adaptation.
Fig. 1. — Schematic presentation of the normal physiologic control of flow-pressure-volume relations in the human systemic circulation (Gelman, 2008). The red elements on the right hand side in every panel represent the arterial system with a circulating volume (upper) and an arteriolar volume (lower). The arterioles drain into the capillary system, represented by the shaded rectangle at the bottom of each panel. This in turn is connected to the venular system (lower blue) and the large veins (upper blue). The circulation is indicated by arrows and is maintained by the heart (yellow circle with rod).

Next to a circulating volume, the venous system also contains a capacitance reservoir (blue box on the left hand side in each panel). A reduction of cardiac output (panel a) evokes a reflexory constriction of the venous compartment (panel b), which actively mobilises blood from the venous to the arterial compartment. In further control of cardiac blood perfusion, there is a dynamic process maintaining a balance between blood volume and vascular tone of veins and arteries (panels c and d). However with increased reduction of cardiac output (panel e), constriction of the arteriolar system reduces influx of blood into the capillary and venous system (panel f). Venous blood then is mobilised passively from the venous capacity reservoir into the circulation due to the pumping activity of the heart (panel f).
Acute or chronic heart failure can induce cirrhosis with liver dysfunction (Naschitz et al., 2000) and/or renal insufficiency (Ronco et al., 2008; Tang et al., 2010). Venous congestion in decompensated heart failure has been recognised as the most important hemodynamic factor contributing to renal dysfunction. (Tang et al., 2010; Mullens et al., 2009; Wencker, 2007).

Similar to the arterial compartment, the venous compartment is subject to gestational adaptive changes. During uneventful pregnancy, venous distensibility is increased, which serves the increased capacitance function of the venous system necessary to accommodate the increase in plasma volume. (Sakai et al., 1994) Venous capacitance returns to nonpregnant values in the first three months postpartum. (Skudder et al., 1990) Venous compliance also increases by 30% and the diameter of the inferior vena cava increases up to 70% above nonpregnant values. (Krabbendam et al., 2007) A dilatation of the left atrium occurs already in early gestation, (Duvekot et al., 1998) whereas a rise of Atrial Natriuretic Peptide (ANP) is observed in the second half of pregnancy. (Krabbendam et al., 2007) This rise of ANP originates from extension of the atrium due to expansion of the plasma volume, and prevents overfilling of the cardiovascular system. Despite an overall increase of venous capacitance, a decrease of compliance of splanchnic veins has been reported in pregnant rats as compared to nonpregnant controls. (Hohmann et al., 1992) Considering the splanchnic vascular bed as the most important blood reservoir of the body, the combination of both increased capacitance and reduced compliance in the splanchnic veins allows for punctual control of cardiac output. Under these conditions, subtle changes of splanchnic arteriolar or venular tone have a prominent impact on mobilisation of stored blood volumes into the circulation. As such, gestation induced changes of the venous compartment seem to upgrade its physiologic properties towards regulation of cardiac output, in which it becomes more powerful than in nonpregnant conditions.

Preeclampsia is known as a maternal cardiovascular maladaptation syndrome with diminished plasma volume expansion, (Ganzevoort et al., 2004) decreased cardiac output, (Rang et al., 2008) and reduced dilation of the left atrium already present in the first trimester (Krabbendam et al., 2007; Duvekot et al., 1998). Reduced plasma volume expansion occurs before increase of progesterone or reduction of aldosterone in women, destined to develop preeclampsia (Salas et al., 2006). The adaptation of the venous compartment, as explained above, is also blunted in preeclampsia: venous distensibility, (Sakai et al., 1994) capacitance, (Goodlin, 1986) and compliance are reduced (Krabbendam et al., 2007). Nearly half of women with a history of preeclampsia show postpartum persistence of subnormal plasma volume, (Spaanderman et al., 2000) which is associated with low venous capacitance (Aardenburg et al., 2005) and impaired venous drainage of the conjunctival microcirculation (Houben et al., 2007). For these women in nonpregnant condition, the autonomic response to volume expansion (Krabbendam et al., 2009) and cardiovascular adaptation to exercise (Aardenburg et al., 2005) is blunted. These women are also at higher risk for recurrence of preeclampsia in subsequent pregnancy, (Aardenburg et al., 2003) and they show a condition of relative overfill already present at the very early beginning of pregnancy, leading to atrial stretch and overshooting of ANP-release (Krabbendam et al., 2007). In pregnant women who subsequently develop early-onset preeclampsia, first trimester cardiac output is lower than normal, whereas this is higher in women destined to develop late-onset preeclampsia (Valensise et al., 2008). Until now, this preeclampsia – related dysregulation of cardiac output hasn’t been linked to the function of the venous compartment.

The information presented above can be summarised as follows:

- the venous compartment is one of the most important systems in the human body towards control and regulation of cardiac output
- gestation induced changes of the venous compartment upgrade the physiologic properties of the venous system towards regulation of cardiac output
- in pregnant women who subsequently develop early-onset preeclampsia, first trimester cardiac output is lower than normal, whereas this is higher than normal in women destined to develop late-onset preeclampsia
- arterial hypertension and liver and/or renal dysfunction can be secondary to abnormal venous hemodynamics

Today it is not yet fully understood what the exact role is for the venous compartment in physiologic conditions with changing patterns of cardiac output, such as normal pregnancy. It is even less clear to what extend the venous compartment is involved in the preclinical and clinical stages of preeclampsia. The information summarised above reveals a new and tempting hypothesis: the clinical stage of preeclampsia, a condition generally considered as one of the most serious gestational complications of which background mechanisms are not yet fully understood, might be a systemic response to a prece-
ing failure of the venous system to regulate cardiac output appropriately. In order to accept or refute this hypothesis, data from more studies and observations are needed. This is why the exploration of the adult’s venous compartment, both in non-pregnant and pregnant conditions, is of interest to all obstetricians, cardiologists, sonographers and scientists involved in research on gestation-induced maternal cardiovascular adaptation mechanisms and/or background mechanisms behind preeclampsia.

Glossary

Distensibility: The general definition of the ability of a vessel to distend in response to volume and/or pressure changes

Compliance: The relation between distensibility and transmural pressure (inside minus outside pressure). It is quantified as the change in volume (∆V) divided by the change in pressure (∆P).

Capacitance: The relationship between the intravascular blood volume and the pressure distending the vascular walls.

References

Aardenburg R, Spaanderman ME, Courtar DA et al. A subnormal plasma volume in formerly preeclamptic women is associated with a low venous capacitance. J Soc Gynecol Investig. 2005;12(2):107-11.

Aardenburg R, Spaanderman ME, Ekhart TH et al. Low plasma volume following pregnancy complicated by pre-eclampsia predisposes for hypertensive disease in a next pregnancy. BJOG. 2003;110(11):1001-6.

Aardenburg R, Spaanderman ME, van Eijndhoven HW et al. Formerly preeclamptic women with a subnormal plasma volume are unable to maintain a rise in stroke volume during moderate exercise. J Soc Gynecol Investig. 2005;12(8):599-603.

Ahmed K, Sampath R, Khan MS. Current trends in the diagnosis and management of renal nutcracker syndrome: a review. Eur J Vasc Endovasc Surg. 2006;31(4):410-6.

Bateman GA, Giles W, England SL. Renal venous Doppler sonography in preeclampsia. J Ultrasound Med. 2004;23(12):1607-11.

Berne R, Levy M. Control of cardiac output: coupling of heart and blood vessels. In: Berne R, Levy M, editors. Cardiovascular physiology. London: The C.V. Mosby Company, 2001:199-226.

Berne R, Levy M. Special circulations. In: Berne R, Levy M, editors. Cardiovascular physiology. London: The C.V. Mosby Company, 2001:241-70.

Boulaep EL. Regulation of arterial pressure and cardiac output. In: Boron WF, Boulaep EL, editors. Medical physiology. Philadelphia: Elsevier Inc., 2005:534-57.

Corradi A, Arendshorst WJ. Rat renal hemodynamics during venous compression: roles of nerves and prostaglandins. Am J Physiol. 1985;248(6 Pt 2):F810-F820.

Dilley JR, Corradi A, Arendshorst WJ. Glomerular ultrafiltration dynamics during increased renal venous pressure. Am J Physiol. 1983;244(6):F650-F658.

Doty JM, Saggi BH, Blocher CR et al. Effects of increased renal parenchymal pressure on renal function. J Trauma. 2000;48(5):874-7.

Doty JM, Saggi BH, Sugarman HJ et al. Effect of increased renal venous pressure on renal function. J Trauma. 1999;47(6):1000-3.

Duvekot JJ, Peeters L. Very early changes in cardiovascular physiology. In: Chamberlain G, Pipkin F, editors. Clinical physiology in obstetrics. Oxford: Blackwell Science Ltd, 1998:3-32.

Ganzvoort W, Rep A, Bousel GI et al. Plasma volume and blood pressure regulation in hypertensive pregnancy. J Hypertens. 2004;22(7):1235-42.

Gelman S. Venous function and central venous pressure: a physiological story. Anesthesiology. 2008;108(4):735-8.

Goodlin RC. Venous reactivity and pregnancy abnormalities. Acta Obstet Gynecol Scand. 1986;65(4):345-8.

Gyselaers W, Molenaerghs G, Mesens Y et al. Maternal Hepatic Vein Doppler Velocimetry During Uncomplicated Pregnancy and Pre-Eclampsia. Ultrasound Med Biol. 2009.

Gyselaers W, Molenaerghs G, Van Mieghem W et al. Doppler measurement of Renal Interlobar Vein Impedance Index in uncomplicated and pre-eclamptic pregnancies. Hypertens Pregnancy. 2009;28(1):23-33.

Hohmann M, McLaughlin MK, Kunzel W. [Direct assessment of mesenteric vein compliance in the rat during pregnancy]. Z Geburtshilfe Perinatol. 1992;196(1):33-40.

Horton JD, San Miguel FL, Membrino F et al. Budd-Chiari syndrome: illustrated review of current management. Liver Int. 2008;28(4):455-66.

Houben AJ, de Leeuw PW, Peeters LL. Configuration of the microcirculation in pre-eclampsia: possible role of the venular system. J Hypertens. 2007;25(8):1665-70.

Itoh S, Yoshida K, Nakamura Y et al. Aggravation of the nutcracker syndrome during pregnancy. Obstet Gynecol. 1997;90(4 Pt 2):661-3.

Juncqueira L, Carneiro J. The circulatory system. In: Juncqueira L, Carneiro J, editors. Basic histology: text and atlas. New York: McGraw-Hill Professional, 2005:205-22.

Karabulut N, Baki YA, Karabulut A. Renal vein Doppler ultrasound of maternal kidneys in normal second and third trimester pregnancy. Br J Radiol. 2003;76(907):444-7.

Krabbendam I, Courtar DA, Janssen BJ et al. Blunted autonomic response to volume expansion in formerly preeclamptic women with low plasma volume. Reprod Sci. 2009;16(1):105-12.

Krabbendam I, Spaanderman ME. Venous adjustments in healthy and hypertensive pregnancy. Expert Rev Obstet Gynecol. 2007;2(5):671-9.

Lotgering FK. Wallenburg HC. Hemodynamic effects of caudal and uterine venous occlusion in pregnant sheep. Am J Obstet Gynecol. 1986;155(6):1164-70.

Mullens W, Abrahams Z, Lewis RJ. Very early changes in cardiovascular physiology in the rat. Am J Physiol. 1985;248(6 Pt 2):F810-F820.

Naschitz JE, Slobodin G, Lewis RJ et al. Heart diseases affecting the liver and liver diseases affecting the heart. Am Heart J. 2004;140(1):111-20.

Pang CC. Autonomic control of the venous system in health and disease: effects of drugs. Pharmacol Ther. 2001;90(2-3):179-230.

Pang CC. Measurement of body venous tone. J Pharmacol Toxicol Methods. 2000;44(2):341-60.

Rang S, van Montfrans GA, Wolf H. Serial hemodynamic measurement in normal pregnancy, preeclampsia, and intrauterine growth restriction. Am J Obstet Gynecol. 2008;198(5):519.

Ronco C, Haapio M, House AA et al. Cardiorenal syndrome. J Am Coll Cardiol. 2008;52(19):1527-39.

Roobottom CA, Hunter JD, Weston MJ et al. Hepatic venous Doppler waveforms: changes in pregnancy. J Clin Ultrasound. 1995;23(8):477-82.

Sakai K, Imazumzi T, Maeda H et al. Venous distensibility during pregnancy. Comparisons between normal pregnancy and preeclampsia. Hypertension. 1994;24(4):461-6.

Salas SP, Marshall G, Gutierrez BL et al. Time course of maternal plasma volume and hormonal changes in women with
preeclampsia or fetal growth restriction. Hypertension. 2006; 47(2):203-8.

Skudder PA, Jr., Farrington DT, Weld E et al. Venous dysfunction of late pregnancy persists after delivery. J Cardiovasc Surg (Torino). 1990;31(6):748-52.

Spaanderman ME, Ekhart TH, van Eyck J et al. Latent hemodynamic abnormalities in symptom-free women with a history of preeclampsia. Am J Obstet Gynecol. 2000; 182(1 Pt 1):101-7.

Tang WH, Mullens W. Cardiorenal syndrome in decompensated heart failure. Heart. 2010;96(4):255-60.

Valensise H, Vasapollo B, Gagliardi G et al. Early and late preeclampsia: two different maternal hemodynamic states in the latent phase of the disease. Hypertension. 2008;52:873-80.

Wencker D. Acute cardio-renal syndrome: progression from congestive heart failure to congestive kidney failure. Curr Heart Fail Rep. 2007;4(3):134-8.

Zigman A, Yazbeck S, Emil S et al. Renal vein thrombosis: a 10-year review. J Pediatr Surg. 2000;35(11):1540-2.